PROGRAM BOOK

of the
Joint Section on
Pediatric Neurological Surgery
of
The American Association of
Neurological Surgeons
and
Congress of Neurological Surgeons

26th ANNUAL MEETING

The Westin Canal Place Hotel
New Orleans, Louisiana

December 2–5, 1997

Jointly Sponsored by
The American Association of Neurological Surgeons
Joint Section on Pediatric Neurological Surgery of The American Association of Neurological Surgeons and Congress of Neurological Surgeons

26th Annual Meeting

The Westin Canal Place Hotel New Orleans, Louisiana

December 2–5, 1997

Jointly Sponsored by The American Association of Neurological Surgeons

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

The American Association of Neurological Surgeons designated this educational activity for a maximum of 20 hours (with attendance at Breakfast Seminars) in category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours that he/she actually spent in the educational event.
Disclosure Information

The Joint 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 will be designated by an “▲”.

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

Paolo Raimondi Lecturers .............................................. 4
Shulman Award Recipients ............................................. 4
Hydrocephalus Association Award Recipients ....................... 5
Pediatric Section Chairmen ............................................ 5
Annual Winter Meeting Sites .......................................... 5
Exhibitor Listing ......................................................... 6

Officers of the Joint Section
on Pediatric Neurological Surgery .................................... 7

Program of the Pediatric Section ..................................... 8

Poster Session ............................................................ 19

Scientific Program Abstracts .......................................... 23

Scientific Program Posters .............................................. 97

1997 Membership Roster ............................................... 125
PAOLO RAIMONDI LECTURERS

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

KENNETH SHULMAN AWARD RECIPIENTS

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 Chemotrophic 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 Hematoma 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.
HYDROCEPHALUS ASSOCIATION AWARD RECIPIENTS

1989 Eric Altschuler: Management of Persistent Ventriculomegaly Due To Altered Brain Compliance
1991 Nesher G. Asner: Venous Sinus Occlusion and Ventriculomegaly in Cranietoxinized 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. Webby-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 Hydrocephalus

PEDIATRIC SECTION CHAIRMEN

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 Nilsen 1993-95 Arthur Martin
1978-79 Luis Schut 1995-97 Harold L. Rekate
1979-81 Fred J. Epstein 1997-99 Marion L. Walker
1981-83 Joan L. Venes

ANNUAL WINTER MEETING SITES

1972 Cincinnati 1986 Pittsburgh
1973 Columbus 1987 Chicago
1974 Los Angeles 1988 Scottsdale
1976 Toronto 1990 San Diego & Pebble Beach
1977 Cleveland 1991 Boston
1979 New York 1993 San Antonio
1980 New York 1994 St. Louis
1981 Dallas 1995 Pasadena
1982 San Francisco 1996 Charleston
1983 Toronto 1997 New Orleans
1984 Salt Lake City 1998 Indianapolis
1985 Houston
EXHIBITOR LISTING

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

**Acra-Cut, Inc.**
989 Main Street
Acton, MA 01720
(508)263-2210

**Booth 11**

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

**The Anspach Companies**
4500 Riverside Drive
Palm Beach Gardens, FL 33410
(561)627-1080

**Booth 9**

**Leica Inc.**
110 Commerce Drive
Allendale, NJ 07401
(800)526-0355

**BrainLAB USA, Inc.**
600 Glen Court
Moorestown, NJ 08057
(609)439-1615

**Booth 4**

**Medtronic PS Medical**
125 Cremona
Goleta, CA 93117
(805)968-1546

**Carl Zeiss, Inc.**
One Zeiss Drive
Thornwood, NY 10594
(914)681-7792

**Booth 5**

**NeuroCare Group**
8401 102nd Street, Suite 200
Pleasant Prairie, WI 53158
(414)947-4900

**Codman/Johnson & Johnson Professional, Inc.**
325 Paramount Drive
Raynham, MA 02767
(508)880-8393

**Booth 3**

**Radionics**
22 Terry Avenue
Burlington, MA 01803
(617)272-1233

**Colorado Biomedical Inc.**
6851 Highway 73
Evergreen, CO 80439
(303)674-5447

**Booth 15**

**Surgical Laser Technologies**
147 Keystone Drive
Montgomeryville, PA 18936
(215)619-3201

**Cook Incorporated**
925 South Curry Pike
Bloomington, IN 47403
(812)339-2235

**Booth 13**

**Synthes**
1690 Russell Road
Paoli, PA 19301
(610)647-9700

**Elekta Instruments, Inc.**
8 Executive Park West
Atlanta, GA 30329
(404)315-1225

**Booth 7**

**Walter Lorenz Surgical, Inc.**
1520 Tradeport Drive
Jacksonville, FL 32218
(800)874-7711

**Booth 2**
OFFICERS OF THE JOINT SECTION ON PEDIATRIC NEUROLOGICAL SURGERY

Chairman: Marion L. Walker
Secretary-Treasurer: Thomas G. Lucassen
Executive Council: Rick Abbott
                 Mark S. Dias
                 Andrew D. Parent
                 Joseph H. Piatt
Membership Committee: S. Terence Myles, Chairman
Rules and Regulations: Alan R. Cohen
Nominating Committee: Harold Rekate, Chairman
Ad Hoc Committees
Committee for Resident Travel Fellowship: R. Michael Scott
Committee for Publication: Art Marlin
Committee for CPT Codes: Joseph H. Piatt
Committee for Child Advocacy: Hector James
Committee for Guidelines: Harold Rekate
PROGRAM OF THE JOINT SECTION ON PEDIATRIC NEUROLOGICAL SURGERY

26TH ANNUAL MEETING

Jointly Sponsored by The American Association of Neurological Surgeons

PROGRAM SCHEDULE

TUESDAY, DECEMBER 2

12:00 NOON–5:00 PM  Nurses Seminar–Imperial Room
1:30 PM–5:30 PM  Executive Committee Meeting of the
                  Joint Pediatric Section–River Room
4:30 PM–8:00 PM  Registration–Foyer III
6:00 PM–8:00 PM  Welcoming Reception–Crescent Ballroom

WEDNESDAY, DECEMBER 3

7:00 AM–8:00 AM  Breakfast Seminar–Ballroom II
                  Posterior Plagiocephaly: When to Treat and Not to Treat
                  Faculty: Harold Rekate, James T. Goodrich
7:00 AM–8:30 AM  Continental Breakfast with Exhibits and Poster
                  Viewing–Ballroom I
7:00 AM–4:00 PM  Registration–Foyer III
7:00 AM–4:00 PM  Exhibit and Poster Viewing–Chairman’s Room/
                  Executive Room, Ballroom I, Foyer I & II
8:00 AM–10:00 AM  Spouse Hospitality–Imperial Room
8:15 AM–8:30 AM  Opening Remarks
8:30 AM–10:00 AM  General Scientific Session I–Ballroom II
                  Moderator: Alan R. Cohen

8:30 AM–8:45 AM

1. Multiple Suture Synostosis and Increased Intracranial Pressure
   Following Repair of Single Suture, Non-Syndromal Craniosynostosis
   Roger Hudgins, MD, Steven Cohen, MD, Fernando Burstein, MD,
   William Boydston, MD, Carita Gilreath, RN (Atlanta, GA)

8:45 AM–9:00 AM

2. The “Crab Procedure” for the Late Repair of Scaphocephaly*
   Ken S. Sato, MD, John E. Kalesbeck, MD, Thomas Raley,
   Thomas G. Luerssen, MD, Robert Havlik, MD (Indianapolis, IN)

*Considered for Shulman Award.
3. **Extended Vertex Craniectomy: Results in 63 Patients with Sagittal Synostosis**
   Jeffrey S. Weinberg, MD, Diana L. Freed, BS, Howard L. Weiner, MD,
   Jeffrey Wisoff, MD *(New York, NY)*

4. **Biodegradable Fixation Techniques for Pediatric Craniofacial Surgery**
   Shekar N. Kurpad, MD, PhD, Jeffrey A. Goldstein, MD,
   Alan R. Cohen, MD *(Cleveland, OH)*

5. **Bio-Resorbable Plates and Screws in Pediatric Neurological and Craniofacial Surgery**
   Alan S. Waitze, MD, Joseph Petronio, MD, Ajay V. Kumar, MD,
   David A. Staffenberg, MD, Christine Narad, RN, PN *(Atlanta, GA)*

6. **Incidence of Chiari I Malformation in Children with Craniosynostosis**
   Sanjay K. Gupta, MD *(Ann Arbor, MI)*, Jennifer C. Hirsch, MD,
   Karin M. Muraszko, MD, Steven R. Buchman MD *(Ann Arbor, MI)*

10:00 AM–10:30 AM  **Coffee Break with Exhibit and Poster Viewing**
                     – Ballroom I

10:30 AM–12:00 NOON  **General Scientific Session II–Ballroom II**
                      Moderator: Walker L. Robinson

10:30 AM–10:45 AM  **Obstetrical Brachial Plexus Injury: Operative and Non-Operative Treatment in Seventy-Four Patients**
                    Eric W. Sherburn, MD, Stuart S. Kaplan, MD, Michael J. Noetzcel, MD,
                    T.S. Park, MD *(St. Louis, MO)*

10:45 AM–11:00 AM  **External Hydrocephalus: A Pitfall in the Evaluation of Suspected Child Abuse**
                    Joseph Piatt MD *(Portland, OR)*

11:00 AM–11:15 AM  **Corpus Callosum Damage and Interhemispheric Transfer of Information Following Closed Head Injury in Children**
                    Harvey S. Levin, PhD, Debra A. Benavidez, PhD, Jack M. Fletcher, PhD *(Houston, TX)*, Dianne B. Mendelsohn, MD, Derek A. Bruce, MD *(Dallas, TX)*

*Considered for Shulman Award.*
11:15 AM–11:30 AM
10. The Infant Face Scale: A Novel Coma Scale for Children Less Than 2 Years Old
    Ann-Christine Duhaime, MD, Robert R. Clancy, MD, Peter P. Sun, MD, Susan R. Durham, MD, Matthew Philips, MD (Philadelphia, PA)

11:30 AM–11:45 AM
11. MRI for Evaluation of Cervical Spine Injury in Young Children
    Peter P. Sun, MD, Susan R. Durham, MD, Anne-Christine Duhaime, MD (Philadelphia, PA)

11:45 AM–12:00 NOON
12. Direct Brain PO2, PCO2, pH Monitoring in Pediatric Patients with Severe Head Injury
    John D. Ward, MD, W.R. Bullock, MD, Steve Darren, E. Doppenberg, MD, JoAnn Tillet, RN (Richmond, VA)

12:00 NOON–1:00 PM  Lunch–Crescent Ballroom

1:00 PM–2:00 PM  Paolo Raimondi Lecture–Ballroom II
Modern Prospective on Craniosynostosis
M. Michael Cohen, Jr., D.M.D., Ph.D.
Professor of Oral Pathology, Faculty of Dentistry
Professor of Pediatrics, Faculty of Medicine
Dalhousie University
Halifax, Nova Scotia, Canada

2:00 PM–3:00 PM  General Scientific Session III–Ballroom II
Moderator: Allen S. Joseph

2:00 PM–2:15 PM
    Yuruzu Tashiro, MD, PhD, James M. Drake, FRCSC (Toronto, Ontario, Canada)

2:15 PM–2:30 PM
14. The Living Shunt: Tissue Engineering in the Treatment of Hydrocephalus*
    Joseph R. Madsen, MD, Joseph Vacanti, MD, George A. Taylor, MD, II-Wood Lee, MD (Boston, MA)

2:30 PM–2:45 PM
15. Does Age or Other Factors Influence the Incidence of Shunt Infection?
    Steven E. Davis, BA, J. Gordon McComb, MD, Lena Mastri-Lavine, MS, Michael L. Levy, MD (Los Angeles, CA)

*Considered for Shulman Award.

3:00 PM–3:15 PM
   Coffee Break with Exhibit and Poster Viewing
   –Ballroom I

3:15 PM–5:15 PM
   General Scientific Session IV–Ballroom II
   Moderators: Michael D. Heafner and John P. Laurent

3:15 PM–3:30 PM
17. The Delta Valve: How Does Its Clinical Performance Compare with Two Other Pressure Differential Valves Without Anti-Siphon Control?
   Steven E. Davis, BA, J. Gordon McComb, MD, Lena Masri-Lavine, MS, Michael L. Levy, MD (Los Angeles, CA)

3:30 PM–3:45 PM
18. Percutaneous Endoscopic Shunt Recanalization: A Clinical Study of 10 Cases
   Jogi V. Pattisapu, MD, Kay R. Taylor, RN, BSN, Eric Trumble, MD, Tina Kovach, RN, Denise Howard (Orlando, FL)

3:45 PM–4:00 PM
19. Endoscopic Third Ventriculostomy: Patterns of Failure*
   Shenandoah Robinson, MD, Alan R. Cohen, MD (Cleveland, OH)

4:00 PM–4:15 PM
20. Third Ventriculostomy: An Outcome Analysis
   Douglas L. Brockmeyer, MD, Keyvan Abtin, MD, Lyn Carey, MD, Marion L. Walker, MD (Salt Lake City, UT)

4:15 PM–4:30 PM
21. Evaluation of Patency of Endoscopic Third Ventriculostomy Using MRI*
   Haroon F. Choudhri, MD, Menahem M. Maya, MD, Jeffrey Weinberg, MD, David Feinberg, MD, Jeffrey Wisoff, MD (New York, NY)

4:30 PM–4:45 PM
22. Telemetric Intraventricular Pressure Measurements Following Third Ventriculo-Cisternostomy
   David M. Frim, MD, PhD (Chicago, IL), Liliana C. Gournerova, MD (Boston, MA)

4:45 PM–5:00 PM
23. Endocrinologic Sequellae Following Third Ventriculostomy for Hydrocephalus*
   Christopher G. Gaposchkin, MD, PhD, Alan R. Jacobs, MD, Mark M. Souweidane, MD (New York, NY)

*Considered for Shulman Award.
5:00 PM–5:15 PM
24. Endoscopic Third Ventriculostomy for the Treatment of Acute Hydrocephalus*
   David P. Gruber, MD, Jeffrey J. Larson, MD, Adam I. Lewis, MD, Jamal M. Taha, MD, Kerry R. Crone, MD (Cincinnati, OH)

5:15 PM–6:15 PM
   Business Meeting–Ballroom II

THURSDAY, DECEMBER 4

7:00 AM–8:00 AM
Breakfast Seminar–Ballroom II
AVM: Non-Surgical Treatment, Interventional Radiography & Radiation Therapy
John J. Connors III, Roland B. Hawkins

7:00 AM–8:30 AM
Continental Breakfast with Exhibits and Poster Viewing–Ballroom I

7:00 AM–12:00 NOON
Exhibit and Poster Viewing–Chairman's Room/Executive Room, Ballroom I, Foyer I & II

7:00 AM–12:30 PM
Registration–Foyer III

8:00 AM–10:00 AM
Spouse Hospitality–Imperial Room

8:00 AM–10:00 AM
General Scientific Session V–Ballroom II
Moderator: Timothy B. Mapstone

8:00 AM–8:15 AM
25. Predicting Disease Progression in Childhood Cerebellar Astrocytomas
   Daniel M. Lieberman, MD, Mitchell S. Berger, MD (San Francisco, CA),
   Daniel W. Smoots, BS, J. Russell Geyer, MD (Seattle, WA)

8:15 AM–8:30 AM
   Nejat Akalan, Assoc. Pro., Faruk Zorlu, Assoc. Pro., Tezer Kutlug, Professor (Ankara, Turkey)

8:30 AM–8:45 AM
27. Cerebellar Astrocytomas During the Age of Modern Imaging*
   Jeffrey W. Campbell, MD, Ian F. Pollack, MD, Ronald Hamilton, MD,
   Barbara Shultz (Pittsburgh, PA)

8:45 AM–9:00 AM
28. The Craniofacial Team Approach to Management of Unilateral Optic Nerve Astrocytomas
   Leslie N. Sutton, MD, Scott Bartlett, MD (Philadelphia, PA)

*Considered for Shulman Award.
9:00 AM–9:15 AM
29. Neurologic Manifestations of Cowden Disease: Further Characterization*
   Shenandoah Robinson, MD, Alan R. Cohen, MD (Cleveland, OH)

9:15 AM–9:30 AM
30. Hyperfractionated Stereotactic Radiosurgery in the Management of Primary Brain Tumors
   Lucia J. Zamorano, MD*, Arturo Saenz, MD, Razvan Buciuc, MD, James Fontanesi, MD, Fernando G. Diaz, MD (Detroit, MI)

9:30 AM–9:45 AM
31. Permanent Iodine-125 Implants in the Treatment of Pediatric Brain Tumors
   Lucia J. Zamorano, MD*, Arturo Saenz, MD, Razvan Buciuc, MD, Laurie Gaspar, MD, Fernando G. Diaz, MD, PhD (Detroit, MI)

9:45 AM–10:00 AM
32. Up-Front Chemotherapy as the Primary Treatment Modality for Hypervascular Tumors*
   Ashfaq Razzaq, MD, Alan R. Cohen, MD (Cleveland, OH)

10:00 AM–10:15 AM    Coffee Break with Exhibit and Poster Viewing
                      –Ballroom I
10:15 AM–12:15 PM    General Scientific Session VI–Ballroom II
                      Moderator: Andrew D. Parent

10:15 AM–10:30 AM
33. Okadaic Acid Induces Apoptosis of Medulloblastoma Cells
   David H. Harter, MD, Lawrence Chin, MD (Baltimore, MD)

10:30 AM–10:45 AM
34. Sodium Phenylbutyrate for Medulloblastoma*
   Jeffrey W. Campbell, MD, Ian F. Pollack, MD, John Freund (Pittsburgh, PA)

10:45 AM–11:00 AM
35. Converging Development Pathways in Medulloblastoma
   Corey Raffel, MD, PhD, Russell H. Zurawel, BA, Cory A. Brown, BA, Robert B. Jenkins, MD, C. David James, PhD (Rochester, MN)

11:00 AM–11:15 AM
36. Endoscopic Transsphenoidal Approach to Pediatric Pituitary Tumors
   Charles Teo, MD, Charles Bower, MD, Ronald L. Young III, MD, Frederick Boop, MD (Little Rock, AR)

*Considered for Skulman Award.
*Disclosure Information
11:15 AM–11:30 AM
37. The Orbitozygomatic Transcavernous Approach for Giant Supracellular Lesions
   Michael L. Levy, MD, J. Diaz Day, MD, Takanori Fukushima, MD,
   Steven L. Giannotta, MD, J. Gordon McComb, MD (Los Angeles, CA)

11:30 AM–11:45 AM
38. Limitations of Image-Guided Techniques in the Surgical Management of Pediatric Brain Tumors*
   Roger Frankel, MD, Joseph Petronio, MD (Atlanta, GA)

11:45 AM–12:00 NOON
39. Results of Pediatric Neurosurgery Manpower Survey
   Anne Marie Flannery, MD (Augusta, GA)

12:00 NOON–12:15 PM
40. Been Surfing Lately? A Critical Appraisal of the Internet as a Pediatric N/S Information Resource*
   Todd A. Maugans, MD (Los Angeles, CA)

12:15 PM–6:30 PM
   Free Afternoon

12:30 PM–2:00 PM
   American Society of Pediatric Neurosurgeons
      Executive Committee Meeting—Imperial Room

2:00 PM–3:30 PM
   NEUROSURGERY://ON-CALL® Editorial Board
      Meeting—River Room 1

6:30 PM
   Meet in Westin Canal Place Hotel Lobby for walk to Cajun Queen Riverboat

7:00 PM–10:00 PM
   Reception/Banquet aboard the Cajun Queen Riverboat

Friday, December 5

7:00 AM–1:00 PM
   Registration—Foyer III

7:00 AM–8:30 AM
   Continental Breakfast with Exhibits and Poster Viewing—Ballroom I

7:00 AM–8:00 AM
   Breakfast Seminar—Ballroom II
   Cranial Base Surgery in Children
   Derek A. Bruce

7:00 AM–3:30 PM
   Exhibit and Poster Viewing—Chairman's Room/
      Executive Room, Ballroom I, Foyer I & II

8:00 AM–10:00 AM
   Spouse Hospitality—Imperial Room

8:00 AM–10:15 AM
   General Scientific Session VII—Ballroom II
   Moderator: Alexa I. Canady

*Considered for Shulman Award.
8:15 AM–8:30 AM
41. The Embryogenesis of Congenital Vertebral Dislocation: Early Embryonic Buckling?
Mark S. Dias, MD, Paul A. Grabb, MD, Michael Landy, MD (Buffalo, NY)

8:30 AM–8:45 AM
42. Dynamic MR-Imaging of the Craniovertebral Junction in Achondroplasia
John S. Myseros, MD, Donald H. Reigel, MD, William Rofthus, MD, Leach Burke, MD, Reed E. Pyeritz, MD, PhD (Pittsburgh, PA)

8:45 AM–9:00 AM
43. Early Manual Reduction for the Treatment of Atlanto-Axial Rotatory Fixation in Children
Nobu Morota, MD, Hitoshi Nakagawa, MD, Junichi Mizuno, MD (Aichi, Japan)

9:00 AM–9:15 AM
44. Search for Major Candidate Genes for Human Neural Tube Defects
Marcy C. Speer, PhD, Elizabeth Melvin, MA, Gordon Worley, MD, Herbert E. Fuchs, MD, PhD, Timothy M. George, MD (Durham, NC)

9:15 AM–9:30 AM
45. Simple Repair of Open Neural Tube Defects*
Carla A. Toms, Teresa Harpold, MD, J. Gordon McComb, MD, Michael L. Levy, MD (Los Angeles, CA)

9:30 AM–9:45 AM
46. The Timing of In Utero Myelomeningocele Repair: Quantitative Analysis*
Michael J. Drewek, MD, Joseph P. Bruner, MD, William O. Whetsell, MD, Noel B. Tulipan, MD (Nashville, TN)

9:45 AM–10:00 AM
47. Relationship between Anorectal Anomalies and Tethered Spinal Cord*
Tanvir F. Choudhri, MD, Indro Chakrobarti, BS, Neil A. Feldstein, MD, John Shullinger, MD, E. Sander Connolly, MD (New York, NY)

10:00 AM–10:15 AM
48. Split Cord Malformation in Myelomeningocele Patients
Bermans J. Iskandar, MD (Madison, WI), Colleen McLaughlin, RN, W. Jerry Oakes, MD (Birmingham, AL)

10:15 AM–10:30 AM Coffee Break with Exhibit and Poster Viewing –Ballroom I

10:30 AM–12:00 NOON General Scientific Session VIII–Ballroom II
Moderator: A. Leland Albright

*Considered for Shulman Award.
10:30 AM–10:45 AM
49. Developmental Biomechanics of the Pediatric Human Cervical Spine
Srirangam Kumaresan, MS, Narayan Yoganandan, PhD, Frank Pintar, PhD,
Wade Mueller, MD *(Milwaukee, WI)*

10:45 AM–11:00 AM
50. Pediatric Intramedullary Spinal Cord Tumors: Is Surgery Alone Enough?
Keith Y. Goh, FRCS, Linda Velasquez, PhD, Fred J. Epstein, MD
*(New York, NY)*

11:00 AM–11:15 AM
George I. Jallo, MD, Diana Freed, BS, Gary Breslow, MD,
Peter Alexander, MD, Fred J. Epstein, MD *(New York, NY)*

11:15 AM–11:30 AM
52. Impact of Selective Dorsal Rhizotomies on Urodynamic Function in
Cerebral Palsy
Jean-Pierre Farmer, MD *(Montréal, Québec, Canada)*, Olivier Vernet, MD
*(Lausanne, Switzerland)*, Anne-Marie Houle, MD, José Luis Montes, MD
*(Montréal, Québec, Canada)*.

11:30 AM–11:45 AM
53. Quantitation of Spasticity in Rhizotomy Patients with Combined EMG-
Joint Motion Analysis
Mark G. Luciano, MD, PhD, Julee E. Perry, MS, Brian Davis, PhD,
Alan Gurd, MD, Jennifer J. Ahl, BSN, CNRN *(Cleveland, OH)*

11:45 AM–12:00 NOON
54. Separation of Craniopagus Twins*
Keyvgan Abtin, MD, Marion L. Walker, MD *(Salt Lake City, UT)*

12:00 NOON–1:00 PM Lunch–Crescent Ballroom
1:00 PM–3:00 PM General Scientific Session IX–Ballroom II
Moderator: John W. Walsh

1:00 PM–1:15 PM
55. Ventral Compression of the Cervicomedullary Junction in Children and
Young Adults with Chiari I Malformation
Paul A. Grabb, MD, W. Jerry Oakes, MD, Timothy B. Mapstone, MD
*(Birmingham, AL)*

*Considered for Shulman Award.
1:15 PM–1:30 PM
56. Toward a Simpler Surgical Management of the Chiari I Malformation
   Mark D. Krieger, MD, Michael L. Levy, MD, J. Gordon McComb, MD
   (Los Angeles, CA)

1:30 PM–1:45 PM
57. The Chiari Zero Malformation
   Bermans J. Iskandar, MD (Madison, WI), Gary L. Hedlund, DO,
   Paul A. Grabb, MD, W. Jerry Oakes, MD (Birmingham, AL)

1:45 PM–2:00 PM
58. Chiari II Malformation: Lesions Found within the Fourth Ventricle
   Joseph Piatt, MD, Anthony D. Agostino, MD (Portland, OR)

2:00 PM–2:15 PM
59. Treatment of Intracranial Aneurysms in the Pediatric Population*
   Howard A. Riina, MD, Alejandro Berenstein, MD (New York, NY),
   Eugene S. Flamm, MD, Leslie Sutton, MD (Philadelphia, PA)

2:15 PM–2:30 PM
60. Cerebral Aneurysms in Children: A Series of 39 Patients*
   Larry T. Khoo, MD, Michael L. Levy, MD, J. Gordon McComb, MD,
   Steven L. Giannotta, MD (Los Angeles, CA)

2:30 PM–2:45 PM
61. Pediatric Arteriovenous Malformations: A Multi-Modality Approach
   Kevin M. Cockroft, MD, Mary L. Marcellus, RN, Michael P. Marks, MD,
   Stephen L. Huhn, MD, Gary K. Steinberg, MD, PhD (Stanford, CA)

2:45 PM–3:00 PM
62. Use of Intraoperative Angiography in Pediatric Vascular Abnormalities
   Sanjay Ghosh, MD, Michael L. Levy, MD, Stephen L. Giannotta, MD,
   J. Gordon McComb, MD (Los Angeles, CA)

3:00 PM–3:15 PM
   Coffee Break with Exhibit and Poster Viewing
   –Ballroom I

3:15 PM–5:15 PM
   General Scientific Session X–Ballroom II
   Moderators: Frederick A. Boop and Glenn Morrison

3:15 PM–3:30 AM
63. fMRI Assessment of the Relationship Between Eloquent Cortical
    Function to Cerebral Lesions
   Elaine T. Kirakopoulos, MD, James T. Rutka, MD, PhD,
   James M. Drake, MD, PhD, Harold J. Hoffman, MD,
   Robin P. Humphreys, MD, David J. Mikulis, MD (Toronto, Ontario, Canada)

*Considered for Shulman Award.
3:30 PM–3:45 PM
64. Limbic Resection Through Parahippocampal Gyrus for Treatment of Mesial Temporal Lobe Epilepsy in Children: Preliminary Outcome
   T.S. Park, MD, Blaise FD Bourgeois, MD, Edwin W. Dodson, MD,
   Susa Arnold, MD, Bruce A. Kaufman, MD (St. Louis, MO)

3:45 PM–4:00 PM
65. Temporal Lobectomy for Epilepsy in Children
   Paul M. Kaney, MD (Detroit, MI)

4:00 PM–4:15 PM
66. Corpus Callosotomy in the Treatment of Intractable Childhood Epilepsy
   Nejat Akalan, Assoc. Pro., Guzide Turanli, Assoc. Pro., Yavuz Renda, Professor,
   Vural Bertan, Professor (Ankara, Turkey)

4:15 PM–4:30 PM
67. Pediatric Hemispherectomies: Is a Shunt Needed?
   Omar Jimenez, MD, Glen Morrison, MD, Prasana Jayakar, MD,
   Michael Duchowny, MD, Maria Penate, RN (Miami, FL)

4:30 PM–4:45 PM
68. Ventriculoscopic Procedures Using Frameless Stereotactic Guidance
   Stephanie L. Einhaus, MD, Michael S. Muhlbauer, MD,
   E. Christopher Troup, MD, Robert A. Sanford, MD (Memphis, TN)

4:45 PM–5:00 PM
69. Concurrent 3-Dimensional Endoscopy: A Preliminary Laboratory Experience
   Joseph Chen, MD, Michael L. Levy, MD, J. Gordon McComb, MD
   (Los Angeles, CA)

5:00 PM–5:15 PM
70. Children with Central Diabetes Insipidus Secondary to Nonspecific Inflammatory Disorders of the Pituitary and Hypothalamus
   Donald P. Atkins, MD, Robert A. Sanford, MD, Lela Ostby, RN
   (Memphis, TN)

5:15 PM Closing Comments
1. **Chemical Analysis of Fluid from Intracranial Arachnoid Cysts**
   Todd A. Maugans, MD, J. Gordon McComb, MD, Michael L. Levy, MD
   *(Los Angeles, CA)*

2. **Nonsurgical Treatment of Post-meningitis Subdural Empyema**
   Márcia C da Silva, MD, MSc, Christovão C. Xavier, MD, Andrea L. Oliveira, MD,
   Juliana E. Gurgel, MD, Valeria M. Leal, MD *(Belo Horizonte, Brazil)*

3. **Evidence That Oxidative Stress Contributes to the Pathophysiology of Hydrocephalus in a Hydrocephalic Rat Model**
   Debra Socci, PhD *(Orlando, FL)*, H. C. Jones, PhD *(Gainesville, FL)*,
   Jogi V. Patrisapu, MD, FAAP *(Orlando, FL)*, K. Blugstad, G. W. Arendash, PhD
   *(Tampa, FL)*

4. **Association of the Chiari II Malformation with Parieto-occipital Encephaloceles: Further Support for the Unified Theory of Chiari II Malformations**
   Michael D. Partington, MD, Ken R. Winston, MD *(Denver, CO)*

5. **The Microglial Response to Progressive Hydrocephalus in a Model of Inherited Aqueductal Stenosis**
   James P. McAllister II, PhD *(Cleveland, OH)*, Richard M. Kriebel, PhD,
   Francesco T. Mangano, BA *(Philadelphia, PA)*, Mark G. Luciano, MD, PhD
   *(Cleveland, OH)*

6. **Induction of Neuronal Nitric Oxide Synthase During the Late Stage of Congenital Hydrocephalus in the HTX Rat**
   Stephen L. Kinsman, MD, Donald Eslin, BA *(Baltimore, MD)*,
   Hazel C. Jones, PhD *(Gainesville, FL)*, Mary S. Lange, MA *(Baltimore, MD)*,
   James P. McAllister II, PhD *(Cleveland, OH)*

7. **Extended Midline Incision for Nasal Dermoid Cyst Excision: Nasal Tip to Intracranial Extradural Extension**
   Michael W. Born, MD, Joel W. Winer, MD *(York, PA)*

8. **Cavernous Sinus Dermoid Cysts**
   Paul M. Kanev, MD *(Detroit, MI)*

9. **The Role of Radical Surgery for Intracranial Melanotic Neuroectodermal Tumor of Infancy**
   Shervin Rahimdashti, BS, Mark L. Cohen, MD, Alan R. Cohen, MD
   *(Cleveland, OH)*

*Considered for Shulman Award.
*Disclosure Information
10. Primary Hypothyroidism Mimicking Pituitary Macroadenoma*
Princemill U. Ehirim, MD, Alan R. Cohen, MD (Cleveland, OH)

Matthieu Vinchon, Annie Laquerriere, PhD, Jean-Pierre Vannier, PhD, Jean-Marc Kuhn, PhD, Pierre Freger, PhD (Rouen Cedex, France)

12. Revision of Ventricular Catheter Obstruction with Bugbie Wire: Retrospective Review at Two-Year Follow-up
Stanley H. Kim, MD, Christopher Madden, MD, Henry Bartkowski, MD, Edward Kosnik, MD, Pramit S. Malhotra (Columbus, OH)

13. Volumetric Study of the Ventricular System in a Canine Model of Obstructive Hydrocephalus*
Peter Chovan, MD, Charles P. Steiner, MS, James P. McAllister II, PhD, Martha J. Johnson, PhD, Mark G. Luciano, MD, PhD, Igor Ayzman, BA, Arcangela S. Wood, BA, Jean A. Tkach, PhD, Joseph F. Hahn, MD (Cleveland, OH)

14. Children with Hydrocephalus and Congenital Heart Disease: What Is Their Outlook?
Todd Crawford, MD, William C. Olivero, MD, William C. Hanigan, MD (Peoria, IL)

15. Atypical Presentations of Low-Pressure Shunt Malfunction
Lori A. McBride, MD, Michael D. Partington, MD, K.R. Winston, MD (Denver, CO)

16. Unusual Presentation of the Complete Currarino Triad with Unique Teratoma Histopathology*
Christopher A. Gegg, MD, Micam W. Tullous, MD, Kathleen Kagan-Hallet, MD (San Antonio, TX)

17. Post-Operative Use of Chalasia Wedge After Craniotomy and Morcellation of Skull for Craniosynostosis: A Technical Note
Stanley H. Kim, MD, Christopher Madden, MD, Henry Bartkowski, MD, Edward Kosnik, MD, Pramit S. Malhotra, MD (Columbus, OH)

18. Spasticity and Strength at the Ankle in Children with Cerebral Palsy
Jack R. Engsberg, PhD, Kenneth S. Olree, MS, Sandy A. Ross, MHS, T. S. Park, MD (St. Louis, MO)

*Considered for Shulman Award.
19. **Traumatic Brain Injury in Children Versus Adults: Differences & Outcomes**  
   Omar F. Jimenez, MD, David Petrin, RN, Doreen Berguestein, RN,  
   John Ragheb, MD *(Miami, FL)*

20. **Trends in Firearms Related Deaths in Children: A Public Health Crisis for Pediatric Neurosurgery**  
   John Ragheb, MD, Andrew Jea, Dorene Beguiristain, RN, Mimi Sutherland, RN  
   *(Miami, FL)*

21. **Stereolithographic Models of Complex Tumors**  
   Michael H. Handler, MD, FACS *(Denver, CO)*

22. **Benign Migratory Cerebellar Endotheliosis**  
   John R. Mawk, MD, Patricia T. Franklin, MSN, James Schimschock, MD  
   *(Portland, OR)*

23. **Volumetric Reduction of a Choroid Plexus Carcinoma Using Preoperative Chemotherapy**  
   Mark M. Souweidane, MD, James H. Johnson, Jr., MD, Eric Lis, MD  
   *(New York, NY)*

24. **Diffuse Subarachnoid Spread of Juvenile Pilocytic Astrocytoma (JPA)**  
   Daniel M. Lieberman, MD, Susan M. Chang, MD, Kenneth D. Aldape, MD,  
   Daniel B. Vigneron, PhD, Charles B. Wilson, MD *(San Francisco, CA)*

25. **Posterior Fossa Craniotomy: An Effective Alternative to Craniectomy**  
   Shekar N. Kurpad, MD, PhD, Alan R. Cohen, MD *(Cleveland, OH)*

*Considered for Shulman Award.*
SCIENTIFIC ABSTRACTS*

*Considered for Shulman Award.
Multiple Suture Synostosis and Increased Intracranial Pressure Following Repair of Single Suture, Non-Syndromal Craniosynostosis

Roger Hudgins, MD, Steven Cohen, MD, Fernando Burstein, MD, William Boydston, MD, Carita Gilreath, RN (Atlanta, GA)

Objective: Increased intracranial pressure, frequently associated with closure of multiple cranial sutures, has been reported to occur in 36% of cases following correction of syndromal craniosynostosis. Although much more uncommon, multiple suture closure may occur following repair of single suture, non-syndromal craniosynostosis and we present two such children.

Results: Two children with non-syndromal craniosynostosis, one metopic and one left coronal, underwent fronto-orbital advancement at age 3 months. At ages 19 months and 5 years, respectively, they both re-presented with headaches, decrease in head circumference percentile, and acceptable cosmetic outcome. Both had CT evidence of multiple closed cranial sutures and increased intracranial pressure (ICP) determined by monitoring. Both improved following a cranial expansion procedure. Etiologic possibilities are discussed.

Conclusion: Delayed closure of multiple sutures and resultant increased ICP may occur following correction of non-syndromal, single suture craniosynostosis. This may be more likely when the initial suture involved is continuous with the facial sutures. Children should be followed for many years following craniosynostosis repair with cranial, neurologic and possibly fundoscopic examinations as well as head circumference measurements to detect delayed closure of cranial sutures.
2  The “Crab Procedure” for the Late Repair of Scaphocephaly*

Ken S. Sato, MD, John E. Kalesbeck, MD, Thomas Raley, Thomas G. Luerssen, MD, Robert Havlik, MD (Indianapolis, IN)

Objectives: A procedure has been developed specifically for the late correction of scaphocephaly. This procedure utilizes interlocking struts to achieve biparietal expansion and preserves the bone over the sagittal sinus. Using this procedure, excellent cosmetic results with minimal morbidity, including blood loss, can be achieved.

Methods: From 1991 through 1997, we have performed 11 Crab procedures on 11 children. All children were over the age of 6 months.

Results: Of the 11 cases, the average age was 18 months at time of operation. The average length of the procedure was 4 hours and 28 minutes with an average hospital stay of 4.2 days. The average blood loss was 347 cc with an average patient weight of 9.5 kg. All of the procedures were completed in one stage. Further procedures were required in 3 cases: one child required removal of protruding screws and plates and one child experienced a post-op wound dehiscence requiring scar revision. There was one further complication of temporary laryngeal dysfunction most likely resulting from endotracheal tube injury while in the upright position.

Conclusions: For those children with scaphocephaly who are not corrected within the first 6 months of life, the “Crab Procedure” allows for excellent results with minimal morbidity.
3 Extended Vertex Craniectomy: Results in 63 Patients with Sagittal Synostosis*

Jeffrey S. Weinberg, MD, Diana L. Freed, BS, Howard L. Weiner, MD, Jeffrey Wisoff, MD (New York, NY)

Objective: We present a retrospective review of 63 patients who underwent an extended vertex craniectomy for sagittal synostosis between 1991 and 1996 by a single surgeon. Follow-up data was obtained from office records and interviews with parents.

Technique: Following infiltration with lidocaine and epinephrine, a routine skin incision is made with a Shaw scalpel. Scalp flaps are retracted laterally in a subgaleal fashion. A 7-centimeter-wide bone flap is elevated from the posterior limits of the anterior fontanelle to the lambdoid suture. Using rongeurs, 6- to 8-centimeter lateral osteotomies are performed bilaterally posterior to the coronal and anterior to the lambdoid sutures. The occipital boss is removed, and the temporal squamosa is fractured outward in a greenstick fashion. Hemostasis is maintained with gelfoam and bone wax. The wound is closed with subgaleal sutures. Steristrips and a bulky dressing are applied.

Results: Forty-seven males and 17 females (mean age 3.6 months) underwent this procedure. Average blood loss was 43 cc. 41% of patients required a blood transfusion. Mean operative time was 51 minutes, and average hospital stay was 1.7 days postoperatively. 94% of parents felt the outcome was excellent. Two patients required reoperation for deformational plagiocephaly.

Conclusion: Extended vertex craniectomy can be performed in less than one hour, with minimal blood loss, brief hospital stay, and excellent cosmetic outcome. We recommend this as the procedure of choice in treating infants less than six months of age with sagittal synostosis.
Biodegradable Fixation Techniques for Pediatric Craniofacial Surgery*

Shekar N. Kurpad, MD, PhD, Jeffrey A. Goldstein, MD, Alan R. Cohen, MD (Cleveland, OH)

We describe our experience with the use of a polymeric biodegradable system for the correction of pediatric craniofacial malformations. These fixation methods, initially described by Eppley in 1996 in the repair of craniofacial trauma for the stabilization of facial fractures, presented, in principle, several advantages over more conventional metallic fixation systems with their risks of allergic reactions, infection, visibility under thin skin, and extrusion or passive intracranial migration.

Our series consists of 17 patients that underwent craniofacial surgery, 14 for craniosynostosis, 2 for brain tumor, and 1 for encephalocele. One of the 14 craniosynostosis patients underwent a two stage procedure, with initial posterior, followed by anterior cranial vault reconstruction. The mean age of the patients was 3 years. All patients with craniosynostosis required a bifrontal craniotomy through a coronal incision. Concomitant cranial vault and/or orbital reconstruction was then performed. The encephalocele defect was repaired with an onlay bone graft, affixed with resorbable fixation to the nasal dorsum. Plates (cut from a prefabricated mesh) and screws, obtained from a Lactosorb 1.5mm set (Lorenz/Biomet, Warsaw, IN), were used with well-established surgical fixation techniques. Cranial bone was used as the source of bone graft when required. Pre- and postoperative clinical, radiologic (3-dimensional CT scans), and pictorial examinations were performed on all patients by both the neurosurgeon and craniofacial surgeon. At 1 year follow-up, no evidence of infection, extrusion, erythema, instability of bony fragments or relapse has been noted. There has been progressive reduction in the profile of the plates and screws on palpation. The excellent short-term results in this series supports the use of resorbable fixation systems in the correction of congenital craniofacial deformities.
5 Bio-Resorbable Plates and Screws in Pediatric Neurological and Craniofacial Surgery*

Alan S. Wairze, MD, Joseph Petronio, MD, Ajay V. Kumar, MD, David A. Staffenberg, MD, Christine Narad, RN, PN (Atlanta, GA)

Objectives: Metal plates have long been used in cranial surgery in both adult and pediatric patients, providing rigid fixation of fractures and osteotomies. However, retained hardware and rigid fixation are not without complications in pediatric patients, including restriction of craniofacial growth and transdural migration of hardware. In this report we review our preliminary experience with bio-resorbable microplates in pediatric neurological and craniofacial surgery.

Methods: Since April 1996, we have performed 51 neurosurgical or craniofacial procedures on 50 children using bio-resorbable craniofacial microplates and screws. Patients ranged in age from 6.0 months to 18.5 years (median, 4.0 years) at the time of operation. Surgical indications for acute traumatic injuries (n=11) included elevation of depressed skull fractures (n=7), reconstruction or stabilization of orbital fractures (n=4), and evacuation of extradural hematomas (n=2). Elective indications have included craniotomy closure for brain tumor or arachnoid cyst (n=12), frontal bone advancement and/or cranial vault remodeling for craniosynostosis (n=17), and cranioplasty or cranial reconstruction (n=9).

Results: Follow-up has ranged from one week to 13.1 months (median, 3.9 months). The rigidity of the fixation was felt by the operating surgeon to be good to excellent in all but two cases. No major complications have been reported although transient headaches were described in 4 patients, and a postoperative cellulitis of the scalp flap occurred in one patient.

Conclusion: Since the problems associated with the use of titanium or other metal plates are particularly relevant to surgery in infants and young children, the use of bio-resorbable plates for rigid fixation of fractures or reconstructive osteotomies should be strongly considered in this group.
Incidence of Chiari I Malformation in Children with Craniosynostosis

Sanjay K. Gupta, MD (Ann Arbor, MI), Jennifer C. Hirsch, MD, Karin M. Muraszko, MD, Steven R. Buchman, MD (Ann Arbor, MI)

Skull abnormalities stand in close relation to associated intracranial abnormalities. An abnormality associated with craniosynostosis is the Chiari I malformation. It has been proposed that Chiari I malformation is secondary to axial skeletal defects. The malformation is associated with weakness, pain, sensory loss, and ataxia. Improvement after posterior fossa decompression in patients is closely allied to early detection. We present eight patients with concordant craniosynostosis and Chiari malformation. The incidence is 6% among 128 patients who presented from 1989-1996 to the University of Michigan Pediatric Craniofacial clinic. This incidence is significantly greater than the estimated 0.08% prevalence of Chiari in the general population. This represents 2.88 standard deviations above the mean with (P<.01). Our series represented a variety of syndromic and non-syndromic synostoses. Specifically, our series included two sagittal synostoses, one Goldenhar's association, one Kleebarttschadel, one FG, two Crouzon's syndrome and one multiple synostoses best categorized as coronal and sagittal synostoses. 75% were categorized as having late diagnosis of their craniosynostosis. We defined late diagnosis as greater than six months in those patients with sagittal synostosis and greater than one year in those patients with other skull abnormalities. Complete neurological evaluation should be performed on all children presenting with a craniosynostosis. A high index of suspicion should be maintained in such children for the diagnosis of Chiari I and should be evaluated with MRI scanning. Our data supports that this management strategy leads to early detection of Chiari and with earlier operative intervention may lead to improved outcome.
Obstetrical Brachial Plexus Injury: Operative and Non-Operative Treatment in Seventy-Four Patients

Eric W. Sherburn, MD, Stuart S. Kaplan, MD, Michael J. Noetzel, MD, T.S. Park, MD (St. Louis, MO)

Introduction: Obstetrical palsies occur on 0.19-2.5/1000 live births. The majority of patients will recover spontaneously; however, predicting which patients will recover and when surgery is indicated is controversial. To understand these issues and evaluate the results of surgical treatment, we reviewed the initial experience of a multidisciplinary brachial plexus clinic.

Methods: From 1991-1996, 103 patients with obstetrical palsies were referred to our clinic. 54 patients treated conservatively and 20 consecutive surgical patients had adequate follow-up for review. Muscle strength in the biceps, triceps, and deltoid (BTD) were measured. The rate of spontaneous recovery and results of surgical treatment were retrospectively reviewed.

Results: Fifty-four children were followed until complete recovery or until a cessation of recovery for 2 years. Complete recovery was seen in 35 (64.8%) while 4 (7.4%) had minimal weakness. Antigravity strength in BTD was seen by 3 months of age in the former and 5.5 months of age in the latter. 6 (11.1%) patients had moderate weakness and 9 (16.7%) had permanent severe weakness. These children had <3/5 strength at 6 months of age. Of 20 surgical cases, 2 patients were lost to follow-up, and 2 underwent exploration without repair. 15 (93%) patients improved postoperatively. 14 (75%) attained antigravity strength in the biceps and triceps, and 8 (50%) achieved this in the deltoid muscle.

Conclusions: Strength testing at 6 months is an accurate predictor of neurologic outcome. Brachial plexus reconstruction is safe and effective for those who fail conservative therapy.
External Hydrocephalus: A Pitfall in the Evaluation of Suspected Child Abuse

Joseph Piatt, MD (Portland, OR)

Objective and Importance: The combination of acute subdural hematoma and retinal hemorrhage in infancy is widely viewed as pathognomonic for inflicted injury. Less widely recognized by pediatricians, social workers, and police investigators is the propensity of patients with craniocerebral disproportion to suffer acute subdural hemorrhage after trivial head trauma. External hydrocephalus is a common cause of craniocerebral disproportion in infancy, and acute subdural hemorrhage after innocent head injuries in infants with external hydrocephalus can lead to unwarranted accusations of child abuse, particularly if retinal hemorrhages are present.

Clinical Presentation: Two infants are presented who suffered acute neurological depression after what their caretakers described as minor trauma. Imaging studies in both patients demonstrated diffuse enlargement of the subarachnoid spaces with modest acute subdural hematomas. Funduscopic examination by consultant ophthalmologists disclosed retinal hemorrhages in both cases, documented by fundus drawings and fundus photography.

Intervention: There was no neurosurgical intervention in either case, and both patients recovered their respective neurological and developmental baselines. Because the neurosurgical consultant had to concede that the caretakers’ descriptions of the injuries might be true, both infants were eventually returned to their homes. Neither infant has suffered recurrent injury after 18 and 9 months follow-up.

Conclusions: Physicians who offer treatment to and testimony about infants with head injuries must be cognizant of the susceptibility of patients with external hydrocephalus to subdural and retinal hemorrhage after minor trauma.
Corpus Callosum Damage and Interhemispheric Transfer of Information Following Closed Head Injury in Children

Harvey S. Levin, PhD, Debra A. Benavidez, PhD, Jack M. Fletcher, PhD (Houston, TX), Dianne B. Mendelsohn, MD, Derek A. Bruce, MD (Dallas, TX)

Neuropathological studies of fatal closed head injury (CHI) in adults have documented a high frequency of corpus callosal (CC) lesions, and magnetic resonance imaging (MRI) has disclosed CC atrophy following severe CHI in adults. We used MRI to evaluate the relationship of CC atrophy and/or lesions to functional hemispheric disconnection following CHI in the pediatric age range. Fifty-one patients ages 7 to 16 years were divided into three groups: mild CHI, moderate to severe CHI with extracallosal (EC) lesions, and moderate to severe CHI with CC atrophy and/or lesions.

Interhemispheric transfer of information was assessed using verbal and nonverbal dichotic listening and other tests of interhemispheric transfer of motor, tactile, and visual information which generated ratio scores reflecting the relative efficiency of left vs. right hemisphere information processing. The CC group demonstrated a greater right ear advantage (i.e., left hemisphere superiority) on a verbal dichotic listening test (consonant vowel syllables) than the mild CHI group (p<.002). No significant group differences were found for the other tests of interhemispheric transfer. CC area measures were based on digitized MR midsagittal images (obtained at least 3 months postinjury) using a semiautomated program that divided the anterior-posterior length into seven segments. To correct for individual variation in brain size, a ratio of CC to total brain area was analyzed. Area measures of the posterior regions of the CC were negatively correlated with verbal dichotic listening laterality indices.

We conclude that the presence of CC atrophy and/or lesions is associated with functional hemispheric disconnection following severe CHI in children.
10 The Infant Face Scale: A Novel Coma Scale for Children Less Than 2 Years Old

Ann-Christine Duhaime, MD, Robert R. Clancy, MD, Peter P Sun, MD, Susan R Durham, MD, Matthew Philips, MD (Philadelphia, PA)

Objectives: While a variety of coma scales have been developed to modify the Glasgow Coma Scale (GCS) for use in infants and young children, none has completely filled the need for a practical and reliable bedside assessment tool. We have developed a novel scale which differs from others in the following ways: 1) it relies on objective measures; 2) it assesses cortical function; 3) it relies on age-appropriate motor development; and 4) it can be applied to intubated patients. Along with other modifications, the Infant Face Scale (IFS) replaces the verbal component of the GCS with a score based on the response of the infant’s face to a standard noxious stimulus. This report will demonstrate the use of the IFS and present interreliability data.

Methods: Twenty-five hospitalized infants under two years of age have been assessed simultaneously by two independent observers who scored the patients using both the GCS and IFS. Interrater reliability was assessed using intraclass correlation coefficients and generalized measures of agreement.

Result: The IFS demonstrated improved interrater reliability compared to the GCS in this age range.

Conclusions: The IFS has several advantages in coma assessment in infants; ongoing studies are analyzing its predictive ability with respect to outcome indices at discharge.
Objective: MRI is of particular value in children with spinal cord injury where there is frequently no body abnormalities. However, its usefulness in detecting occult cervical spine injuries is unclear and traumatic soft tissue abnormalities of the cervical spine has not been well documented in children. This retrospective review was performed to examine the usefulness of MRI in identifying cervical spine injuries in pediatric trauma patients.

Methods: Between 8/93 and 7/97, cervical spine MRI was obtained in 75 children <10 years to rule out a cervical injury per protocol with any of the following: 1) young age or obtundation with a likely mechanism of injury 2) abnormal or equivocal plain film/CT 3) signs or symptoms referable to the cervical spine.

Results: MRI revealed abnormalities in 20 children (27%); predominately ligamentous and soft tissue signal changes of the upper cervical spine. There were no compressive lesions. Eleven children with MRI abnormalities had normal plain films/CT scans. These children were treated with collar immobilization and followed with serial dynamic cervical films. None of these patients required surgery for instability and none developed delayed myelopathy (mean 5.5 month follow-up). Nine children also had abnormalities on plain film/CT scans. Of these, 2 children required cervical fusion.

Conclusion: As a screening tool for cervical spine injury in young children, MRI is most useful in detecting upper cervical ligamentous and soft tissue abnormalities. The natural history and optimal treatment of these soft tissue injuries to the cervical spine without bony dislocations remains to be defined.
Direct Brain PO2, PCO2, and pH Monitoring in Pediatric Patients with Severe Head Injury

John D. Ward, MD, W.R. Bullock, MD, Steve Darren, E. Doppenberg, MD, JoAnn Tillett, RN (Richmond, VA)

It has always been recognized that there is a need to adequately monitor the brain in children who have had a severe insult, such as a severe head injury. Until recently, this consisted of intracranial pressure (ICP) monitoring with perhaps some estimation of cerebral blood flow (CBF). More recently, there have been reports of measurements of direct brain PO2, PCO2, pH, and temperature in adult head injury patients. To date, there is little literature about the use of this technique in pediatric patients. We report direct brain PO2, PCO2, and pH monitoring in a series of five (5) pediatric patients who were severe head injury. The results of this monitoring and a discussion will be presented.
13 Reversibility of Functional Injury of Neurotransmitter Systems with Shunting in Hydrocephalic Rats: Implications for Intellectual Impairment

Yuruzu Tashiro, MD, PhD, James M. Drake, FRCSC
(Toronto, Ontario, Canada)

Introduction: Intellectual impairment has been related to alteration of neurotransmitter innervation including the cholinergic, dopaminergic and noradrenergic systems. Recent studies of the pathogenesis of hydrocephalus have demonstrated neuronal functional injury. We evaluated the effect of hydrocephalus and shunting on these neurotransmitter systems using immunohistochemistry.

Materials and Methods: Hydrocephalus was induced in 20 Wistar rats by intracisternal injection of 0.05 ml of 25% kaolin solution. Four controls received the same volume of saline. V-P shunts were inserted at 2 and 4 weeks after kaolin injection (W), and these animals were killed at 8W (shunted group). Other hydrocephalic animals were killed at 2, 4 and 8W without shunting (non-shunted group). The calibre of frontal cortex was compared before and after shunting. The morphology and number of immunoreactive (IR) neurons to choline acetyltransferase (ChAT) in cholinergic nuclei (Chl-6), tyrosine hydroxylase (TH) in ventral tegmental area and dopamine B-hydroxylase (DBH) in locus ceruleus, were compared between shunted and non-shunted groups.

Results: Shunting restored the cortical calibre thickness. The size and number of ChAT-IR neurons were progressively reduced in the process of hydrocephalus in Ch 1, 2, 3 and 4 (p< .05). The TH-IR neurons were also reduced in number with remarkable loss of projection fibers in the terminal areas. Early (2W), but not late (4W), shunting restored ChAT and TH immunoreactivity to control levels (p< .05). The DBH-IR neurons were severely compressed by dilated fourth ventricle losing immunoreactivity in the terminal areas. Only early shunting also restored DBH immunoreactivity in this system.

Conclusions: A progressive functional injury occurs in the cholinergic, dopaminergic and noradrenergic systems in the process of hydrocephalus. This may contribute to the intellectual impairment and may be prevented only by early shunting.
14 The Living Shunt: Tissue Engineering in the Treatment of Hydrocephalus+

Joseph R. Madsen, MD, Joseph Vacanti, MD, George A. Taylor, MD, II-Wood Lee, MD (Boston, MA)

Objectives: Many of the problems with traditional ventriculoperitoneal shunts arise from undesirable interactions between host tissues and permanent plastic inserts. Susceptibility to infection by usually nonpathogenic bacteria and adhesions between choroid plexus and plastic are results of such undesirable interactions. Tissue engineering, the use of living autologous cells seeded into biodegradable polymers, allows creation of custom-designed, living implantable devices.

Methods: As a first approach to the use of tissue engineering in the treatment of hydrocephalus, we have made CSF shunt from chondrocyte-seeded polyglycolic acid (PGA) tubes coated with polylactic acid-polyglycolic acid copolymer, implanted initially with thin silastic stents removed 4 weeks after shunt insertion.

Results: When implanted in rats made hydrocephalic by cisternal injection of kaolin, shunts seeded with sufficient chondrocytes can remain patent to intraventricularly injected radioopaque contrast up to 4 weeks after stent removal, at which time the animals were sacrificed. Use of bovine xenograft cells in nude (athymic) rats resulted in more efficient seeding with chondrocytes, stiffer tube walls, and better patency. One of 7 animals implanted in the first series had excellent patency and good relief of hydrocephalus; the remaining 6 had partial obstruction of the shunt and at least mild to moderate hydrocephalus at sacrifice.

Conclusions: We conclude that polymer type, cell type, and cell density will require considerable optimization, but a working tissue engineered shunt is feasible and may one day address the problems of interactions of living tissue and inert polymer.
Does Age or Other Factors Influence the Incidence of Shunt Infection?

Steven E. Davis, BA, J. Gordon McComb, MD, Lena Mastri-Lavine, MS, Michael L. Levy, MD (Los Angeles, CA)

Some studies indicate that infants, especially those less than one month of age, have a higher incidence of shunt infection. To look at age as well as other factors that might relate to the rate of shunt infection we reviewed the records of all patients undergoing a ventriculoperitoneal shunt insertion or revision at our institution from January 1, 1985, to December 31, 1994.

There were a total of 2325 shunting procedures performed on 1193 patients with a male:female ratio of 678:515. The overall infection rate was 3.2% (74 infections). Broken down by age the infection rates were as follows: <1 month, 9/223 (4.0%); 1-6 months, 16/449 (3.6%); 6-12 months, 13/297 (4.4%); 12-18 months, 3/122 (2.5%); 18-24 months, 7/116 (6.0%); and 24+ months, 26/1118 (2.3%) with no statistically significant difference between age groups. If one selectively examines the premature neonates who developed hydrocephalus secondary to intraventricular hemorrhage from the figures given above, one finds that 2/44 (4.5%) neonates became infected, which was not significant. The infecting organism distribution was divided roughly in thirds, with relatively equal representation from staph coagulase negative and staph aureus. The remaining third was comprised of a wide variety of organisms. This represents a relative increase at our institution in the incidence of staph aureus and a relative decrease in staph coagulase negative infection. The infection rate was the same irrespective of whether the procedure was to insert or revise the shunt. The etiology of the hydrocephalus was not a factor, nor was the presence of a neural tube defect. If the patient had another operative procedure in addition to a shunt insertion or revision there was no increased rate of infection. The presence of fluid accumulation along the shunt tract or at another neurological operative site was associated with a significant increase in incidence of infection 15/168 (8.9%) compared to those with no fluid accumulation (p<.0001).
Maternal Shunt Dependency: Implications for Obstetrical Care, Neurosurgical Management and Pregnancy Outcomes


As more women with cerebrospinal fluid shunts are surviving to child-bearing age, neurosurgeons, obstetricians and other health care professionals will require information about these patients. The goal of this on-going study is to gather comprehensive data from shunted women during and immediately following pregnancy, and to make this information available to the interdisciplinary team of clinicians from whom patients will seek pre-conception counseling as well as clinical care during the course of pregnancy.

Respondents were asked to complete a questionnaire including information on maternal background and medical history including delivery, shunt performance, birth defects, and any unusual complications.

Currently, this study comprises the largest single database of this population, including 77 pregnancies and 37 mothers, ranging in age from 18 to 41. Thirty mothers reported VP shunt configurations, 6 reported VA shunt configurations, 1 reported a ventriculopleural shunt configuration, 1 reported an LP configuration. Fifteen women have given birth twice, 2 have given birth three times, and 1 has given birth four times. Thirteen women reported seizure histories, 10 of whom were prescribed seizure medication. Two women with seizure histories chose not to take anti-convulsants during pregnancy. One of these mothers reported numerous seizures during pregnancy, while the other reported no seizure activity during pregnancy. Among the women who were prescribed anti-convulsant therapy during pregnancy, 2 of 4 gave birth to children with congenital defects. Additionally, 3 of the women with seizure histories gave birth to children without apparent congenital defects, but who were later diagnosed with ADD PDD and Autism. Seventeen miscarriages, 2 tubal pregnancies, 3 elective abortions, 55 live term births, and 1 preterm live birth occurred. Of the 17 miscarriages, 2 mothers with hydrocephalus secondary to spina bifida accounted for 7 of these (41%). Among the 5 shunt malfunctions during pregnancy, two were associated with one mother who had 9 revisions in the year preceding pregnancy. Three of these required revision during pregnancy, and 2 revisions were delayed until after delivery. No acute device failures were reported during delivery. Four malfunctions were reported following delivery. A total of 48 pregnancies (86.0% of the live births) were associated with no shunt revision. No shunt infections were associated with shunt surgery during pregnancy or delivery by c-section. Four babies presented with birth defects including one affected by both hydronephrosis and neurofibromatosis, another with spina bifida and hydrocephalus, and one each with Vater’s syndrome and Trisomy-13. Among the more unusual complications of female shunt dependency, were the reports of two women with VP shunts which disconnected and wrapped around their repro
ductive organs. One of these women went on to have three successful pregnancies; the other required a hysterectomy due to the damage to her reproductive system.

While these data indicate that shunt-dependent mothers can deliver safely and successfully, this study suggests that health care providers should be educated about the special concerns of these patients. Among the clinical issues are the following: 1) late term symptomatology, 2) the incidence of shunt malfunctions during the course of or immediately following pregnancy; 3) shunt dependency and its implications for reproductive health; 4) special considerations with respect to delivery, and 5) the relative risk of birth defects.
The Delta Valve: How Does Its Clinical Performance Compare with Two Other Pressure Differential Valves Without Anti-Siphon Control?

Steven E. Davis, BA, J. Gordon McComb, MD, Lena Masri-Lavine, MS, Michael L. Levy, MD (Los Angeles, CA)

The Delta valve, a pressure differential valve with an antisiphon device, by preventing the development of a negative pressure proximal to the valve is purported to prevent the overdrainage of cerebrospinal fluid (CSF) and thereby reduce the incidence of subdural fluid collections as well as postural symptoms to the patient. In addition, it might reduce the number of obstructions as there would be no negative pressure sucking tissue and debris into the shunt system. In order to assess whether the Delta valve's use reduced the number of shunt related problems as compared with two other pressure differential valves without an antisiphon component, we reviewed the charts of 1193 patients undergoing 2325 ventriculoperitoneal shunt insertions or revisions from January 1, 1985, to December 31, 1994, at our institution.

The Delta valve and two pressure differential valves without antisiphon function were exclusively inserted during the following time periods: Holter-Hausner (H-H); January 1, 1985, to August, 1987, Heyer-Schulte (H-S); August, 1987, to June, 1991, and Delta June, 1991, through the end of the study period. Kaplan-Meier survival curves documented that H-H 56/80 (70%), H-S 64/93 (69%) and Delta 89/113 (79%) valves were functioning at 1 year follow-up and that H-H 55/80 (69%), H-S 61/93 (66%), and Delta 88/113 (78%) valves were functioning at two years. The symptomatic subdural fluid collection rate was: H-H 2/80 (2.5%), H-S 1/93 (1.1%), and Delta 2/113 (1.8%). The combined breakage/obstruction rate was: H-H 4/80 (5.0%), H-S 3/93 (3.3%), and Delta 2/113 (1.8%). No Delta valves malfunctioned secondary to fibrous capsule affecting the antisiphon device.

In conclusion, it appears that the Delta valve's performance was similar to the H-H and H-S valves, two valves without an antisiphon device. There was no significant difference in the occurrence of symptomatic subdural fluid collections based upon valve type. There was no significant difference in the combined valve breakage/obstruction rates based upon valve type. There was a trend toward a decrease in the number of revisions required when using the Delta valve though this was not statistically valid.
Percutaneous Endoscopic Shunt Recanalization: A Clinical Study of 10 Cases

Jogi V. Pattiapu, MD, Kay R. Taylor, RN, BSN, Eric Trumble, MD, Tina Kovach, RN, Denise Howard (Orlando, FL)

Proximal ventricular catheter obstruction by choroid plexus is a frequent occurrence in children with shunted hydrocephalus. In some cases, this obstruction of flow is due to membranous occlusion of the ventricular catheter by a small amount of debris/tissue within the catheter lumen. It is postulated that since the cerebral aqueduct is only 0.8 mm in diameter, only a few of the many holes at the catheter tip need be patent for adequate CSF drainage via the shunt system.

Our previous work has demonstrated excellent visualization and intraluminal dissection with laser and electrocautery using in vitro and in vivo models. We developed a "shunt dissector" that allowed us to perform intraluminal dissection and successful recanalization of the catheters. In four patients, we visualized the catheter lumen during routine shunt taps and evaluated the degree of patency, which revealed partial obstruction while maintaining adequate CSF flow.

Ten cases (8 patients) of percutaneous endoscopic shunt recanalization were performed under IRB study protocol. The mean age was 23.5 months and the etiology of hydrocephalus was spina bifida in 4 children, IVH of prematurity in 3 children, and congenital obstructive hydrocephalus in 1 child. All the children had symptoms of shunt malfunction (increasing head circumference, headaches, lethargy, etc.). CT/MRI scans identified ventricular dilatation. Proximal shunt malfunction was confirmed by ventricular taps with poor CSF flow.

The procedure was performed in the operating room under aseptic conditions. The Rickham reservoir was entered with a 16 gauge angiocath and endoscopic intraluminal dissection using electrocautery was carried out until free CSF flow was established. Distal flow was evaluated with ventricular catheter obstruction and flushing of the valve mechanism. CSF analyses did not identify increased protein levels and the cultures were negative.

Symptoms were resolved in all but one child who required a standard open proximal ventricular catheter revision within 24 hours after this procedure. Another child required an open procedure due to the inability to adequately visualize the catheter lumen, and was found to have a shunt disconnection (ventricular catheter from the reservoir). At 15-week mean follow-up (3-28 weeks at time of abstract), the symptoms resolved, and CT scans identified improvement in the eight successful cases.

The percutaneous endoscopic shunt recanalization procedure can be in certain selected cases of proximal shunt malfunction (ventricular catheter obstruction). It is unclear if permanent shunt patency can be maintained using this technique, although in some cases multiple procedures may be required. However, this minimally invasive therapeutic shunt tap may prove very useful in certain patients with proximal shunt malfunctions, thereby minimizing surgical risks.
Endoscopic Third Ventriculostomy: Patterns of Failure*

Shenandoah Robinson, MD, Alan R. Cohen, MD (Cleveland, OH)

Endoscopic third ventriculostomy (ETV) has become popular as a treatment for selected patients with non-communicating hydrocephalus. The appeal of this procedure is that it can be carried out with minimal invasiveness and, when successful, eliminates the need for ventricular shunting. With increasing experience, the selection criteria for ETV continue to be defined. Early enthusiasm has been tempered by reports of complications and failures. We critically analyzed our experience with ETV to better define the optimal candidates for this procedure.

During the past 7 years, we performed ETV on 34 hydrocephalic patients, with an overall success rate of 56%. We defined success as control of symptoms and signs of hydrocephalus without the need for ventricular shunting. Of the 15 patients who failed ETV, 7 failures occurred early (<1 month) and 3 failures occurred late (>1 year). Four of the failures occurred in infants, 5 were in children, and 6 were in adults. ETV was performed in a total of 5 infants; it was successful only in 1. Fourteen of the 15 patients who ultimately failed ETV had a history compatible with an associated communicating hydrocephalus (prior hemorrhage, infection, or disseminated intraventricular tumor). If patients with possible associated communicating hydrocephalus are eliminated from this series, then the procedure was successful in 19/20 cases (95%). Two patients developed complications: both had weight gain, presumably hypothalamic in origin, and 1 also had diabetes insipidus. No patient had hemorrhage, infection, or new neurologic deficit.

ETV is a powerful procedure that allows many patients with symptomatic hydrocephalus to avoid ventricular shunting. It is important to understand the patterns of failure of this procedure in order to improve patient selection. The optimal candidate for ETV has non-infantile non-communicating hydrocephalus, without factors suggesting associated communicating hydrocephalus.
Endoscopic third ventriculostomy has gained widespread acceptance as a way to manage hydrocephalus in selected patients. To determine which patient groups have the highest chance of successful third ventriculostomy, we performed a retrospective case review. From June 1992 to December 1996, 97 patients underwent attempted third ventriculostomy at our institution. There were 59 males and 38 females with a mean age of 8.1 years (range 1 day to 29.5 years). The patients diagnoses included: myelomeningocele = 24, aqueductal stenosis = 19, posterior fossa tumor = 10, tectal plate tumor = 9, slit ventricle syndrome = 8, congenital hydrocephalus = 7, post-hemorrhagic hydrocephalus = 7 and other = 13. Twenty-six of 97 (27%) were abandoned due to either unfavorable anatomy, inability to perform a cisternostomy, or hemorrhage. The rate of successful third ventriculostomy in 71 patients, with either complete shunt avoidance or removal, varied widely by diagnosis and patient age. It varied from 0% to 75%, with an overall success rate of 52%. Follow-up data was available in 88 patients for a mean of 14.6 months. Complications included one transient herniation syndrome, one basilar artery perforation, two cases of ventriculitis, one transient decrease in level of consciousness, and one transient hemiparesis. The implications of these findings, as well as future directions in third ventriculostomy, will be discussed.
21 Evaluation of Patency of Endoscopic Third Ventriculostomy Using MRI

Haroon F. Choudhri, MD, Menahem M. Maya, MD, Jeffrey Weinberg, MD, David Feinberg, MD, Jeffrey Wisoff, MD (New York, NY)

Introduction: Endoscopic third ventriculostomy is becoming the procedure of choice for patients with non-communicating hydrocephalus due to a reduced likelihood of infection and a closer approximation of physiologic CSF pathways. Postoperatively, ventricular size may be unchanged or only slightly diminished leading to difficulty in evaluating the success of the procedure. Doppler and MRI flow void have been used to demonstrate CSF egress from the third ventricle. We describe our experience with MR flow studies.

Methods: Six patients were treated with seven procedures utilizing a 2.3 mm Neuronavigation endoscope. The aperture was made with a Bugby wire and dilated with a 2 F Fogarty catheter. All patients were evaluated with an MR flow study with a 1.5 T system Magnetom Vision (Siemens Medical Systems, Iselin, NJ) with 25 mT/m maximum gradient strength. Sagittal and axial high-resolution T2-weighted sequences without flow compensation, and axial EKG-triggered flow-sensitive phase contrast sequences were obtained through the floor of the third ventricle.

Results: CSF flow through the ventriculostomy was demonstrated in all patients immediately postop, and a qualitative and quantitative increase was seen in 2 patients on their delayed MR flow studies. One patient with marked clinical improvement lasting two years and flow demonstrated with MR, subsequently had a return of her symptoms and absent flow. Fenestration of a membranous film covering the previous ventriculostomy yielded resolution of her symptoms and a concomitant increase in flow on MR.

Conclusions: Endoscopic third ventriculostomy is being used with increasing frequency and MRI flow study represents an effective noninvasive technique in the evaluation of patency and correlates with the clinical course of the six patients in our series.
22 Telemetric Intraventricular Pressure Measurements Following Third Ventriculo-Cisternostomy

David M. Frim, MD, PhD (Chicago, IL), Liliana C. Goumnerova, MD (Boston, MA)

Objectives: Endoscopic third ventriculo-cisternostomy (3VC) has only recently become the procedure of choice for treatment of hydrocephalus due to aqueductal stenosis. Despite the many patients that have undergone this procedure, the postoperative CSF pressure dynamics are largely unknown. We sought to describe the postural changes in intraventricular pressure (IVP) seen after 3VC hoping to develop some insight into evaluation of the success of the procedure.

Methods: Seven patients undergoing endoscopic 3VC for hydrocephalus due to aqueductal stenosis were implanted with ventricular catheters connected to a pressure telemonitoring device (TeleSensor; Radionics). These patients were monitored for postural IVP dynamics postoperatively as a measure of the success of the fenestration.

Results: Five of the patients exhibited a stereotypic time course in postural IVP that consisted of: 1) an elevation of early postoperative supine IVP (to as high as 30 cm of water in one patient) coupled with a steep drop in IVP with elevation of the head; 2) an intermediate period of low to normal supine IVP with a shallower drop in IVP with head elevation; and 3) late reconstitution of postural IVPs indistinguishable from unshunted patients. These patients had eventual resolution of their preoperative symptoms. Two of the patients demonstrated early postoperative elevations in supine IVP that did not drop to near 0 with elevation of the head. Symptoms consistent with hydrocephalus never abated and these two patients required ventriculoperitoneal shunting. MR imaging three months postoperatively revealed some decrease in ventricular size and evidence of flow through the third ventriculo-cisternostomy in the four patients with normal IVPs.

Conclusions: We conclude that after an initial period of adjustment, postural IVP after 3VC returns to an unremarkable baseline despite the novel CSF pathway into the prepontine cistern. This may represent maturation of the breach through the third ventricular floor or brain recovery from a preoperative period of high pressure. In addition, we find the telemonitor to be a useful adjunct to the management of the ventriculo-cisternostomy patient.
Endocrinologic Sequellae Following Third Ventriculostomy for Hydrocephalus*

Christopher G. Gaposchkin, MD, PhD, Alan R. Jacobs, MD, Mark M. Souweidane, MD (New York, NY)

Objective: Third ventriculostomy is becoming accepted as the treatment of choice for specific types of hydrocephalus. These generally include non-communicating hydrocephalus secondary to idiopathic aqueductal stenosis or neoplasm. In successful situations, third ventriculostomy provides the benefit of CSF diversion without the life-long burden of shunt hardware. However, disadvantages other than treatment failure are poorly characterized. Because the procedure involves fenestration of the hypothalamus with potential damage to the hypothalamic-pituitary axis, our aim was to evaluate third ventriculostomy patients for postoperative endocrine dysfunction.

Methods: Complete data on 4 third ventriculostomy patients is currently available. Their ages ranged from 4 to 18. Etiology of hydrocephalus was aqueductal stenosis (n=2), pontine tumor (n=1), and pineal tumor (n=1). The patient with pontine glioma was the only one treated with corticosteroids. In all cases, pre- and postoperative endocrine evaluations were obtained.

Results: Tests of adrenal and thyroid function (cortisol, TSH, fT4, fT3) were obtained in all patients. In three patients, gonadotropin (FSH, LH) levels were recorded. All patients had normal preoperative endocrine studies, although the glioma patient had suppressed adrenal function. Following third ventriculostomy, endocrine studies were repeated at 2-5 months; no change was observed in any of the endocrine profiles of these patients.

Conclusion: Preliminary findings suggest that fenestration of the hypothalamus during third ventriculostomy does not produce a measurable effect on endocrine function.
Endoscopic Third Ventriculostomy for the Treatment of Acute Hydrocephalus

David P. Gruber, MD, Jeffrey J. Larson, MD, Adam I. Lewis, MD, Jamal M. Taha, MD, Kerry R. Crone, MD (Cincinnati, OH)

Objectives: Acute obstructive hydrocephalus resulting from neoplastic compression has traditionally been managed by external ventricular drainage or ventriculoperitoneal shunting. However, these strategies can increase the risk of infection. Endoscopic third ventriculostomy can provide an effective alternative without requiring a shunt or without the risks associated with an external ventricular drain.

Methods: Since July 1993, six patients (ages 5 to 16) presented with acute neurologic deterioration, apnea, and bradycardia. CT and MRI scans revealed hydrocephalus caused by a brain tumor. In each case, the symptoms proved refractory to corticosteroids and diuretics. All six children underwent endoscopic third ventriculostomy.

Results: Following the procedure, all patients showed resolution of symptoms associated with hydrocephalus. Immediate postoperative CT and MRI scans demonstrated resolving transependymal flow and decreased ventriculomegaly. Each child underwent surgery for a posterior fossa tumor 3 to 7 days after the ventriculocisternotomy. MRI studies performed at follow-up (mean 12 months) showed no persistent hydrocephalus. No complications occurred from this procedure.

Conclusions: We conclude that endoscopic third ventriculostomy should be considered for patients with obstructive hydrocephalus who meet the following criteria: 1) acute neurologic deterioration, 2) older than 2 years of age, 3) third ventricle at least 5 mm wide, 4) no prior radiation treatment, and 5) patent retroclival cistern. In this study, endoscopic third ventriculostomy provided an effective alternative to ventriculoperitoneal shunting or external ventricular drainage.
Predicting Disease Progression in Childhood Cerebellar Astrocytoma

Daniel M. Lieberman, MD, Mitchell S. Berger, MD (San Francisco, CA), Daniel W. Smoots, BS, J. Russell Geyer, MD (Seattle, WA)

To help identify clinical predictors of recurrence by cerebellar astrocytomas in children we performed univariate and multivariate Cox analysis using data extracted retrospectively from the charts of 78 children with cerebellar astrocytoma who were treated between 1966 and 1993. Tumors were pilocytic (N = 62), fibrillary (N = 13), or mixed oligoastrocytoma (N = 3). Of the 74 patients without neurofibromatosis, 48 had postoperative contrast-enhanced computerized tomography or magnetic resonance scans for review; among these 17 children had residual disease after surgery. The presence of tumor on postoperative imaging studies (relative risk = 10.1, p value < 0.001), volume of residual tumor (1.5, < 0.001), surgeon's report of residual tumor (7.3, < 0.001), percent of tumor resected (0.9, 0.002) and radiotherapy (3.3, 0.008) were significant prognostic factors influencing tumor recurrence in univariate analysis. When compared to pilocytic tumors, fibrillary tumors (relative risk = 2.7, p = 0.041) had increased risk of progression, while the histologic diagnosis of oligoastrocytoma had no prognostic significance. The patient's sex, age, tumor location, and morphology were not predictive of disease progression in univariate analysis. Comparing the four measures of the extent of surgical resection using multivariate Cox analysis the volume of residual tumor was most closely linked with disease progression. When analyzed simultaneously with volume of residual tumor on imaging studies, only fibrillary histology showed an increased predictive value (relative risk = 18.0, p = 0.011). In conclusion, for benign pediatric cerebellar astrocytomas, the risk of postoperative disease progression increases significantly in the presence of larger postoperative tumor volumes and fibrillary histology.
Childhood Ependymomas: Prognostic Factors in Long-term Outcome

Nejat Akalan, Assoc. Pro., Faruk Zorlu, Assoc. Pro., Tezer Kutluk, Professor (Ankara, Turkey)

Objective: Numerous studies report conflicting results as to the prognosis and appropriate treatment for children with intracranial ependymomas. The aim of this study is to define the factors which lead to the differences in outcome in these patients.

Method: A retrospective analysis was performed with identified 64 children who presented with intracranial ependymoma at our institution between the years 1980 and 1996. Factors which may affect the overall survival rate were analyzed statistically. Factors examined included age, localization, extent of surgical resection, histopathology and adjuvant therapy.

Results: In 16 patients the tumors were of supratentorial origin and infratentorial in 48. The patients age ranged from 18 months to 16 years. The operative mortality was zero percent for supratentorial and 8.3% for infratentorial tumors. Total resection was documented in 2 of 16 supratentorial and 21 of the 48 infratentorial tumors. Overall, 38 patients received postoperative radiotherapy and an additional 23 received adjuvant chemotherapy, as well. Sixteen patients underwent reoperation (3 more than once) for tumor recurrence. Five- and 10-year overall survival rates were 56.4 and 38.0% for supratentorial ependymomas and 42.4 and 28.1% respectively for infratentorial tumors. The analysis of the results showed that an age >3 years (P=3D0.016), localization (P=3D0.018) and resection extent (P=3D0.009) were associated with better overall survival.

Conclusion: The results obtained indicate that surgery still remains the most important determinant associated with prolonged survival in childhood ependymoma. The outcome in children under 3 years of age with incomplete resections are no better than other malignant intracranial tumors.
Eighty-five patients diagnosed with cerebellar astrocytomas between 1975-1993 were identified from the Children's Hospital of Pittsburgh Tumor Registry. This time span allowed for modern imaging with computed tomography or magnetic resonance imaging in all patients as well as a minimum of three years follow-up. Tumor pathology was independently reviewed by a neuropathologist blinded to patient outcome using the WHO classification system. Patient characteristics and outcome were determined by retrospective chart review and degree of operative debulking assessed from operative reports and postoperative imaging. Nine patients were excluded either because of lack of surgical specimen to review (n=6) or because the tumor contained a significant non-astrocytic component (n=3). An additional 4 patients were excluded for either predominately brainstem location of tumor (n=2) or because they suffered from neurofibromatosis (n=2). Of the remaining 72 patients, there were 53 juvenile pilocytic astrocytomas (JPA), 12 low-grade astrocytomas (LGA), and 7 high-grade astrocytomas (HGA). The median age of patients with LGA (54 mo) was significantly lower (p=0.0001) than those with JPA (84 mo) or HGA (100 mo). Median progression-free survival (PFS) and total survival (TS) in patients with HGA were 6 and 8 months, respectively. While median PFS and TS were not reached in patients with JPA or LGA, patients with JPA had significantly better PFS (p=0.001) and TS (p=.005) than those with LGA. Life table analysis estimates that at five years after diagnosis, 80% of patients with JPA versus 50% of those with LGA will not progress, with TS of 100% versus 73%. Patients who underwent gross total resection (GTR) of either a LGA or JPA had similar PFS with 100% TS, while patients with subtotal resection had significantly worse prognosis with a LGA compared to a JPA, with 5-year PFS of 17% versus 45% (p=0.01) and 5-year TS of 25% versus 100% (p=0.003). Postoperative radiation significantly decreased the incidence of recurrence after subtotal resection of a JPA (p=0.002).

HGA of the cerebellum carry a uniformly poor prognosis in our experience, with no patient surviving two years after diagnosis. Conversely, complete resection of a JPA or LGA results in a very favorable prognosis, with on 3/44 patients experiencing any progression. Tumor pathology did not affect prognosis after GTR. Conversely, prognosis of patients with subtotal resection of tumor was significantly worse with LGA than with JPA. Postoperative radiation significantly decreased the incidence of progression in incompletely resected JPA, while having no effect on survival. The treatment of choice for JPA and LGA is complete surgical resection. Incompletely resected LGA are a fatal disease in many patients and should be treated aggressively.
Optic nerve astrocytomas present with visual loss and proptosis. Some have maintained that these are hamartomas which require no treatment, but some patients present with disfiguring proptosis suggesting that these are true neoplasms. Our strategy has been to consider curative surgical excision if there is disfiguring proptosis, the eye is blind, and MRI shows no involvement of the opposite nerve or the chiasm. Neurofibromatosis does not influence the decision. The optic nerve is inspected intradurally, and sectioned as far from the optic chiasm as possible to avoid a field cut in the unaffected eye. The optic canal is unroofed, the ophthalmic artery divided, and the intracannicular portion of the nerve removed. The orbital rim and roof are removed as a block, providing exposure of the orbit. The orbital portion of the tumor is excised, with care to protect extraocular muscle and levator function. The globe is left in situ. The operation was performed on 8 children ages 2-9 years, with follow-up of 1-8 years. None have required further treatment of their tumors, although 4 had tumor in the chiasmatic margin. One had residual tumor by MRI. The major complications have been CSF leaks. Cosmetic results were good, although 2 have required enucleation, and 3 have had proptosis corrections. Optic nerve gliomas causing proptosis may be resected with minimal morbidity, and the presence of residual tumor in the optic nerve stump does not appear to be an adverse prognostic factor. Arguments for and against radical surgery will be presented.
Neurologic Manifestations of Cowden Disease: Further Characterization

Shenandoah Robinson, MD, Alan R. Cohen, MD (Cleveland, OH)

Cowden disease, or multiple hamartoma syndrome, is a rare autosomal dominant disorder characterized by mucocutaneous lesions including facial papules, gingival papillomas and acral keratoses. Other hamartomas are often present, and there is a high incidence of associated breast and thyroid carcinoma; thus, recognition of the dermatologic manifestations of Cowden disease may lead to the early detection of malignancy.

Recently, some patients with Cowden disease have also been reported to have a peculiar cerebellar hamartoma called L Hermitte-Duclos disease, or dysplastic cerebellar gangliocytoma. Given the extreme rarity of both disorders, it seems unlikely that their association in individual patients is coincidental. The finding of an association between these two uncommon entities suggests that they may represent a single new phakomatosis.

We report 3 patients with both Cowden disease and L Hermitte-Duclos disease. All 3 patients developed signs of increased intracranial pressure and required resection of the cerebellar hamartoma. Patients manifested a variety of other hamartomas as well as benign and malignant neoplasms. Review of the literature suggests that the association between L Hermitte-Duclos disease and Cowden disease has probably been under-reported.

Inclusion of L Hermitte-Duclos disease in the Cowden spectrum suggests that Cowden disease is a true phakomatosis, with hamartomas arising from cutaneous and neural ectoderm. Patients with the Cowden phakomatosis may present in childhood with either dermatologic or neurologic manifestations. It is important to recognize that L Hermitte-Duclos disease may not occur simply as an isolated hamartoma but, instead, as a part of the larger spectrum of Cowden disease. Recognition of this association has direct clinical bearing: diligent long-term follow-up of individuals with L Hermitte-Duclos disease and the Cowden spectrum may lead to the early diagnosis of cancer.
30 Hyperfractionated Stereotactic Radiosurgery in the
Management of Primary Brain Tumors

Lucia J. Zamorano, MD*, Arturo Saenz, MD, Razvan Buciuic, MD,
James Fontanesi, MD, Fernando G. Diaz, MD (Detroit, MI)

Introduction: In the present study, we present our experience with
hyperfractionated stereotactic radiosurgery in the management of primary brain
tumors, in terms of patient selection, management and results.

Material and Methods: From December 1994 to January 1997, 20 patients
with primary brain tumors underwent hyperfractionated stereotactic radiosurgery
with a LINAC based system. Prior to the radiosurgical procedure all patients under-
went a CT scan under stereotactic conditions to define the target volume. The
follow-up ranged between 1 and 21 (mean 9) months after the radiosurgical proce-
dure, consisting of clinical and radiological assessment. There were 12 men and 8
women. Age ranged from 3 to 44 (mean 16) years. The types of tumors treated were:
malignant glioma: 8 (40%) patients (glioblastoma multiforme: 3, anaplastic
astrocytoma: 3, anaplastic ependymoma: 2); benign glioma: 3 (15%) patients
(low grade astrocytoma: 3); brainstem glioma: 3 (15%); medulloblastoma: 2 (10%);
germinoma: 2 (10%); primary neuroectodermal tumor: 1 (5%); teratoma: 1 (5%).
In 17 patients the diagnosis was confirmed histologically and in 3 (brainstem glioma)
it was made on a clinical and radiological basis. Interactive image-guided surgical
resection was performed in 14 patients, and stereotactic biopsy in 3. The location
was: 11 (55%) supratentorial (8 lobar and 3 pineal) and 9 (45%) infratentorial (5
brainstem and 2 cerebellar). All patients underwent external beam radiation therapy
(36-56 Gy) prior to the radiosurgical procedure. Hyperfractionated radiosurgery
was delivered with a LINAC based system (9-16 Gy).

Results: In terms of survival, 18 (90%) patients are alive at a median follow-up
of nine months. Of these, 12 (66%) patients had no evidence of disease, 5 (27%)
had stable disease and 1 (7%) is asymptomatic with disease. In terms of complica-
tions, 3 (15%) patients developed radionecrosis demonstrated by stereotactic biopsy
within the first 10 months after radiosurgery.

Conclusions: Our results support the usefulness of this therapeutic approach in
the multimodality management of patients with primary brain tumors. It offers
adequate local control of the lesions, with minimal risk of associated complications,
increasing the length and quality of life in these patients, although further
follow-up is required.

*Disclosure Information
31 Permanent Iodine-125 Implants in the Treatment of Pediatric Brain Tumors

Lucia J. Zamorano, MD\textsuperscript{a}, Arturo Saenz, MD, Razvan Buciu\textsuperscript{c}, MD, Laurie Gaspar, MD, Fernando G. Díaz, MD, PhD (Detroit, MI)

Introduction: We present our experience in the treatment of pediatric brain tumors using permanent Iodine-125 implants in the multimodality management of these patients, in terms of patient selection, management and results.

Materials and Methods: From June 1988 to December 1996, 20 patients underwent permanent Iodine-125 implants. Age ranged from 2 to 18 (mean 10) years. There were 12 males and 8 females. The location was supratentorial in 16 (80%) patients (12 lobar and 4 thalamic), and infratentorial in 4 (20%) patients (1 cerebellar, 3 in the brainstem). Prior to brachytherapy 15 patients underwent surgical resective procedures; in 11, stereotactic biopsy was performed. The histological diagnosis was: anaplastic astrocytoma in 9 (45%) patients; low-grade astrocytoma, 7 (35%); glioblastoma multiforme, 1 (5%); anaplastic ependymoma, 1 (5%); choroid plexus carcinoma, 1 (5%); and primary neuroectodermal tumor, 1 (5%). Implants were performed in 7 primary concurrent tumors, in 6 patients with primary tumors without external beam radiation therapy, in 5 primary tumors after external beam radiation therapy and in 5 recurrent tumors. All patients underwent CT scan (1988-1994) or CT scan and MRI (1994-1996) under stereotactic conditions. Tumor volume was defined by these image modalities, and ranged from 0.7 to 33.4 (mean 9.5%) cc. With the assistance of a three-dimensional multiplanar treatment planning system a protocol was designed to deliver 5cGy (range 4-7) per hour to the tumor volume. One to four catheters were required to obtain optimal dose distribution. The total activity ranged from 1.3 to 57.8 mCi. Catheter placement was performed stereotactically. Follow-up ranged from 2 to 88 (median 43) months consisting of clinical and radiological assessment every 3 months during the first year, and every 6 months thereafter.

Results: At the median follow-up, 13 (65%) are alive. Of these, 3 (23%) have recurrent disease and in 10 (77%) there is no evidence of progression (a more detailed multivariate statistical analysis will be presented). In terms of complications, 1 (5%) patient developed skin necrosis and 1 (5%) radionecrosis after the implants.

Conclusions: The results obtained support this therapeutic approach in pediatric patients with brain tumors (especially benign and malignant gliomas). The treatment modality with this subgroup of patients can benefit in terms of length and quality of survival, with a minimal associated risk of complications.

\textsuperscript{a}Disclosure Information
Up-Front Chemotherapy as the Primary Treatment Modality for Hypervascular Tumors

Ashfaq Razzaq, MD, Alan R. Cohen, MD (Cleveland, OH)

Whenever feasible, radical resective surgery is considered to be the primary treatment for malignant brain tumors, with chemotherapy and radiation often administered in an adjuvant setting. However, some large hypervascular tumors, particularly those occurring in young children, pose an exceptional challenge for the neurosurgeon, and radical removal is not always safe. To limit blood loss during resection of hypervascular tumors, we used up-front chemotherapy as the primary treatment modality in two children. This produced a dramatic reduction in tumor vascularity and greatly facilitated definitive surgical removal.

Two children with hypervascular supratentorial neoplasms received up-front chemotherapy as primary treatment following an initial limited surgical intervention. One was a 2 1/2-year-old boy presenting with obtundation and increased intracranial pressure from a giant hypervascular tumor that was wrapped around the carotid, middle, and anterior cerebral arteries. Resection was attempted, but only biopsy was possible due to the extreme vascularity of the lesion. Pathology was primitive neuroectodermal tumor. The patient was treated with primary induction chemotherapy, and the tumor responded dramatically with reduction in size. Residual tumor was easily removed at a second-look operation at which time it was fibrous and avascular. Two years after diagnosis the patient is neurologically normal with no observable disease. The other patient was an 8-month-old infant presenting moribund with an enormous hypervascular choroid plexus carcinoma. Only limited resection could be conducted because of extreme vascularity of the tumor. Multi-agent chemotherapy was administered, and at second-look surgery the tumor appeared fibrous and avascular. Two and 1/2 years after diagnosis the patient has no observable disease and is without deficit.

Up-front chemotherapy can be useful in the treatment of highly vascular malignant brain tumors of childhood. In selected cases, chemotherapy alters the tumors consistency and can cause a dramatic reduction in vascularity, permitting definitive surgery to be conducted with minimal blood loss.
Okadaic Acid Induces Apoptosis of Medulloblastoma Cells

David H. Harter, MD, Lawrence Chin, MD (Baltimore, MD)

Programmed cell death or apoptosis is a complex, regulated genetically controlled physiologic phenomena involved in immunologic and developmental processes. The suppression of apoptotic pathways is a recognized component in the establishment and progression of neoplastic disorders. A number of agents have been shown to induce apoptosis, including UV, ionizing radiation, oxidative stress, growth factor withdrawal and chemotherapeutic agents. It is likely that regulation of apoptosis includes a kinase/phosphatase cascade as a point of amplification and/or integration of these varied stimuli.

Daoy cells, established from a human medulloblastoma sample are shown to undergo apoptosis upon treatment with okadaic acid (OA), a membrane permeable C38 polyether fatty acid, which specifically inhibits serine/threonine protein phosphatases 1 and 2 (PP1 and PP2A). The IC$_{50}$ of OA was 20 nM, which correlates with values described in other cell lines. Treated cells were observed to undergo typical apoptotic alterations with rounding of cytoplasmic membranes, loss of adhesion and nuclear dissolution. Additional evidence of apoptosis included accumulation of a sub-G1 DNA containing population as determined by flow cytometry, and activation of endonucleases, producing oligonucleosomes as demonstrated by DNA laddering. Further study of this system may elucidate signal transduction pathways and common effectors of apoptosis applicable to many neoplastic disorders. Specifically, further findings may be directly applicable to the treatment of medulloblastoma, one of the most common pediatric tumors.
Sodium Phenylbutyrate for Medulloblastoma*

Jeffrey W. Campbell, MD, Ian F. Pollack, MD, John Freund (Pittsburgh, PA)

Twelve nude mice underwent subcutaneous implantation of 5 x 105 DAOY cells into the right flank. Four days after implantation, the mice were started on four weeks of either 400 mg/kg/dose of sodium phenylbutyrate (NaPBut) or vehicle (PBS) given orally twice a day. Tumor progression was recorded by manually palpating tumor size and using the LxWxW/2 method of estimating volume. Animals were sacrificed when the tumor size significantly impaired ambulation, at roughly 300 mm³. Four days after tumor implantation, at the tumor implantation, at the initiation of therapy, the two groups of animals had nearly identical size tumors at 75 mm³. Within one week, the six animals receiving NaPBut exhibited significantly smaller average tumor volume as compared to the six control animals (31.6 mm³ versus 95.0833 mm³, p=0.001). At the cessation of therapy after four weeks, the control animals had an average tumor volume of 233 mm³ versus 42 mm³ for the mice receiving NaPBut (p=0.0001). All six of the control animals were sacrificed for increasing tumor size between four and eight weeks, while four of the six animals receiving NaPBut had no palpable tumor at the end of eight weeks, with the other two existing small tumors. NaPBut is an effective treatment of medulloblastoma in this subcutaneous model. The efficacy of this therapy for intracranial tumor is currently under investigation. NaPBut is potentially an attractive treatment strategy for medulloblastoma because of its oral administrative and low morbidity.
Converging Developmental Pathways in Medulloblastoma

Corey Raffel, MD, PhD, Russell H. Zurawel, BA, Cory A. Brown, BA, Robert B. Jenkins, MD, C. David James, PhD (Rochester, MN)

Objectives: Medulloblastomas are associated with two inherited cancer syndromes, Gorlin's syndrome (GS) and Turcot's syndrome. GS is characterized by multiple basal cell carcinomas, skeletal abnormalities, and medulloblastoma. The gene altered in GS is PTC, a component of the sonic hedgehog (shh) pathway. Turcot's syndrome patients develop colon carcinomas and brain tumors. Patients with Turcot's who develop medulloblastomas have germ line mutations in the APC gene. APC negatively regulates the activity of B-catenin, a transcription factor. Interestingly, no mutations in APC have been reported in sporadic medulloblastomas. We hypothesize that medulloblastomas contain mutations in shh pathway or APC pathway genes.

Methods and Results: We have examined 24 sporadic medulloblastomas for LOH at the PTC locus on 9q22.3. Five (20.8%) demonstrated LOH. In three of these, a mutation in the remaining PTC allele was identified using SSCP and direct sequencing analysis. Two mutations were duplication insertions; the third was a single base deletion. Despite reports that APC is not altered in medulloblastomas, we have identified LOH in the region of APC in 5 of 34 tumors. In addition, we have examined 68 tumors for B-catenin mutations by direct sequencing of exon 3, which encodes all four serine/threonine phosphorylation sites in the protein. Phosphorylation at these sites leads to functional inactivation of the transcription activity of B-catenin. Four tumors had an activating mutation in exon 2. Interestingly, wnt-1 is upregulated by shh and wnt-1 increases B-catenin activity.

Conclusions: While the two inherited syndromes that include medulloblastoma effect different genes and pathways, the ultimate effect may be increased cell turn-over induced by B-catenin directed transcription.
Endoscopic Transsphenoidal Approach to Pediatric Pituitary Tumors

Charles Teo, MD, Charles Bower, MD, Ronald L. Young III, MD, Frederick A. Boop, MD (Little Rock, AR)

Endoscopy is rapidly becoming an essential part of every neurosurgeon's armamentarium. Its application to sinus surgery has been exemplified by our ENT colleagues. Progression of endoscopic techniques from the sinuses to the skull base was a natural step. The authors present a series of 11 pediatric cases where an endoscopic transsphenoidal approach was taken to biopsy and resect tumors in the pituitary fossa, sphenoid sinus, and upper clival region. Many pathological processes were encountered including fungal infection, primary bone lesions, pituitary adenomas, and a mucocele. There were no complications specific to this technique and positive biopsies were obtained in all cases and total tumor resection achieved where this was desired. Although, the learning curve is steep, the endoscopic technique offers better illumination and wider field of visualization. It is less invasive than traditional microsurgical techniques and does not have the associated ENT complications such as nasal fistulae and gum anesthesia. The authors will present their series of pediatric cases and show a video demonstrating the surgical technique.
The Orbitozygomatic Transcavernous Approach for Giant Suprasellar Lesions

Michael L. Levy, MD, J. Diaz Day, MD, Takanori Fukushima, MD, Steven L. Giannotta, MD, J. Gordon McComb, MD (Los Angeles, CA)

We describe the benefits and potential complications of the orbitozygomatic approach for large suprasellar lesions with posterior extension including the third ventricle. Prior to initiation of this approach, cadaver studies were performed over a 2-year period in approximately 26 specimens (52 sides). Five patients with large suprasellar lesions extending posteriorly to involve the superior aspect of the third ventricle in addition to evidence of impingement upon the basilar cisterns and basilar circulation underwent the orbitozygomatic approach. There were 2 males and 3 females in the study. Mean age was 7.2 years with a range of 3 to 20 years. Lesions included craniopharyngiomas in 2 patients, pilocytic astrocytomas in 2 patients and an atypical meningioma in 1 patient. Three patients had complete excision of their tumors. One patient with a craniopharyngioma had recurrence at 5 years. Residual tumor was noted in one patient with a pilocytic astrocytoma. Complications were only noted in 1 patient where compromise to the hypothalamic-pituitary axis was exacerbated as was a visual field cut. No infections or CSF fistulas were noted.

Advantages of the approach include enlargement of the operative corridor, easy access to the posterior aspect of tumor, easy access to the basilar cisterns, and minimizing brain retraction. Extradural removal of the anterior clinoid process facilitates mobilization of the optic nerve and allows for rapid access into the cavernous sinus. Proximal arterial control of the petrous carotid can also be achieved. In conclusion, the orbitozygomatic approach is a safe and effective approach for large lesions in the sellar and suprasellar region with posterior and superior extension. The most important is the ability to approach the superior and posterior aspects of the tumor in addition to areas of tumor volume in the posterior circulation with only a minimum of retraction and morbidity.
Limitations of Image-Guided Techniques in the Surgical Management of Pediatric Brain Tumors*

Roger Frankel, MD, Joseph Petronio, MD (Atlanta, GA)

Objectives: Frameless stereotactic and image-guided technology has been available to pediatric neurosurgeons for slightly over three years. Initial experience has shown it to be particularly well-suited to pediatric neurosurgical applications because of the preponderance of low-grade and/or well-margined lesions. In the current study we seek to define the technical limitations of this technology as it applies to the surgical management of brain tumors in children.

Materials and Methods: The present study comprises a prospective single-armed review of all image-guided procedures using the ISG/Elekta Viewing Wand® that were performed in the management of brain tumors in children at Emory University School of Medicine since January, 1995. Records were reviewed with respect to the type (fiducial or surface-fitting) and accuracy of the registration (based on root mean square values) used, the applicability and efficacy of the surgical technique as prospectively recorded in the operative reports and surgeons' notes, and the surgical time as determined from the operative records.

Results: In all, 43 procedures were performed on 38 children, ranging in age from 3 days to 18 (median, 8.2) years at the time of surgery. The surgical procedures performed included transcranial debulking or resection in 40/43, needle biopsy in 2/43, and cyst drainage in 1/43 cases. Tumors were primarily supratentorial in 25/38 patients, and infratentorial in 12/38 patients. No procedure was aborted because of patient movement or inadequate registration. Histologies included a typical distribution of pediatric CNS neoplasms. Efficacy was felt to be poor or fair in 9/43 cases. Image-guidance was felt to contribute little to the efficacy of resection in lesions located primarily in CSF spaces such as the suprasellar cistern and cerebellopontine angles, although brain shift was not felt to be significant. Primarily brainstem tumors, although there was no statistically significant difference in registration accuracy in posterior fossa tumors as compared with supratentorial lesions. The instrument was felt to be most useful in planning craniotomy flaps, selecting trajectories to subcortical lesions, and in defining tumor-brain interfaces, especially early in the procedure.

Conclusion: Based on the above findings, the authors have largely abandoned the use of image-guidance for lesions of CSF spaces such as the suprasellar cistern and cerebellopontine angle as well as for many cystic or exophytic brainstem lesions. It will continue to be used for surgical planning, including trajectory selection, and tumor/brain mapping, especially as functional MR images become integrated.
Results of Pediatric Neurosurgery Manpower Survey

Ann Marie Flannery, MD (Augusta, GA)

Manpower issues of tomorrow will be influenced by the number of physicians and specialists trained today. Little information is available to anticipate needs for pediatric neurosurgeons for the next ten years or the activity level of pediatric neurosurgeons currently in practice. The objective of this study was to determine need for pediatric neurosurgeons and current work loads.

Methods and Results: A questionnaire was distributed to pediatric neurosurgeons attending the American Association of Neurological Surgeons' Pediatric Section Meeting in 1996. Ninety-two questionnaires were returned. Nine were returned by residents. Of the remaining eight-three respondents, 73% were less than 55 years of age, 65% were in academic practice, 26% were in group private practice; the remainder were divided between solo private practice, military practice and retired. Respondents indicating that their present workload was too busy, appropriate, or not busy enough were 14%, 52%, and 34%, respectively, with geographic and practice type variations. Of the practicing pediatric neurosurgeons, 19% indicated plans to retire within the next ten years. Twenty-nine respondents, or 35%, were considering adding an associate to the practice within the next ten years. This group includes 10 of the 16 planning to retire.

Conclusion: While not all pediatric neurosurgeons were available to respond to this survey, the group attending this meeting represented a large number of those currently in pediatric neurosurgery practice. Total positions the survey predicts to be available in the next ten years is between 3.5 and 4.5 per year, not including positions to be added by practices not represented at the meeting. Planning to meet manpower needs is important for our specialty and the patients we serve.
40 Been Surfing Lately? A Critical Appraisal of the Internet as a Pediatric N/S Information Resource*

Todd A. Maugans, MD *(Los Angeles, CA)*

Objectives: Patients and their families rely upon many different resources for medical information. In recent years, the electronic superhighway has emerged as a popular means of conveying medical information. The nature of information conveyed by the Internet, its sources and accuracy (as defined by scientific measures) is highly variable, however.

Methods: This analysis attempts to define these parameters (source, information type and accuracy) for ten common pediatric neurosurgical topics: hydrocephalus, spina bifida, tethered cord, craniosynostosis, brain tumors, spinal cord tumors, head injury, spinal cord injury, meningitis and seizures. Several popular search engines were employed, using each topic as the search terms.

Results: The number of hits per topic varied from 3 to 136,720. Eleven categories of sources were identified. Eight types of informational categories were identified. The most common sources were academic neurosurgical departments, support groups and patients or family members. The most common informational types were evidence-based and personal reflections. The accuracy of information was generally high. The majority of information was user friendly, however, the volume was often overwhelming and the ease of acquisition was highly variable. In addition to dissemination of information, a clear objective of many sources was self-promotion.

Conclusions: The Internet represents an important information resource to pediatric neurosurgical patients and their families. Guidelines will be offered to assist neurosurgeons in the education of patients and their families about the use of this resource.
Congenital vertebral dislocation (CVD) is a rare congenital spinal malformation characterized by an abrupt sagittal translation (often spondylolisthesis) of a single vertebral segment (usually near the thoracolumbar junction), with an acute kyphosis at the involved segment. The more caudal vertebra is dysplastic and at first glance appears posteriorly dislocated into the vertebral canal as a posterior hemivertebra, but is actually well aligned with the more caudal vertebral column. The posterior elements are dysplastic or absent, and the spinal cord is posterior in a relatively superficial location. Congenital sensorimotor abnormalities are remarkably minor given the degree of dislocation, and many children are neurologically normal. At least one previously reported case with intradural exploration demonstrated absence of the nerve roots at the involved level. Unfortunately, the present classification of complex congenital vertebral anomalies is confusing, and CVD has been variously grouped with a number of other congenital vertebral malformations under the common terms segmental spinal dysgenesis, medical spinal aplasia, and posterior hemivertebra. Moreover, a putative embryonic mechanism has never been proposed for CVD. Based upon our experience with five personal cases and a critical review of the literature, we suggest that the present nosology of complex congenital vertebral abnormalities is confusing. We identify congenital vertebral dislocation as a distinct entity having characteristic radiographic features. We propose a novel putative embryonic mechanism—early embryonic buckling—which likely occurs before the period of axonal outgrowth during embryonic Stage 13-14 (post-ovulatory day 30) and the beginning of chondrification of the posterior elements at stage 15-16 (post-ovulatory day 39).
Dynamic MR Imaging of the Craniovertebral Junction in Achondroplasia

John S. Myskows, MD, Donald H. Reigel, MD, William Rofthus, MD, Leah Burke, MD, Reed E. Pyeritz, MD, PhD (Pittsburgh, PA)

Introduction: Seven to eight percent of infants with achondroplasia die of sudden death within the first year of life. Foramen magnum stenosis and compression at the cervico-medullary (CMC) junction may be the etiology. Ligamentous laxity, macrocranium, hydrocephalus, and occipital condylar hypoplasia may all play a role in cranovertebral junction instability. Therefore, to investigate if occipitoatlantal instability contributes to CMC, we studied 2 infants with achondroplasia in the upright position using dynamic MR imaging.

Methods and Results: Both infants had signs and symptoms of CMC. Supine MR imaging revealed modest CMC. Dynamic MR in the position of axial loading demonstrated marked CMC in the flexed and neutral positions. However, the cervico-medullary junction was spared in extension. Powers ratio measurements were consistent with occipitoatlantal instability. Following wide craniectomy in one infant, dynamic MR imaging showed no evidence of CMC in any position. The second infant was lost to follow-up.

Conclusion: Occipitoatlantal instability may be a major risk factor leading to sudden death of infants with achondroplasia. To further evaluate this question, a prospective, multi-center study of infants with upright dynamic MR imaging will be required.
43 Early Manual Reduction for the Treatment of Atlanto-Axial Rotatory Fixation in Children

Nobu Morota, MD, Hiroshi Nakagawa, MD, Junichi Mizuno, MD (Aichi, Japan)

Objective: Atlanto-axial rotatory fixation (AARF) is a well known, unique form of cervical trauma in childhood. In our institute, we have experienced 2 cases of AARF in the past 10 years. This paper emphasizes the effectiveness of manual reduction under general anesthesia with muscle relaxants in the early phase.

Clinical presentation: Case 1 is a 4-year-old girl who presented 3 weeks after a left persistent torticollis developed immediately after a head trauma. On admission, she had no neurological deficit and radiological examinations revealed AARF. Manual reduction under endotracheal general anesthesia was carried out 3 days after the admission. The head and neck were immobilized by Halo vest for a month followed by a cervical collar. Case 2 is a 10-year-old boy who developed a left spontaneous torticollis a week ago. He had no neurological deficit and diagnosed as AARF. Cervical traction using a Gardner's tong resulted in no effect and manual reduction under general anesthesia was performed 10 days after admission. Reduction was maintained by a cervicothoracic brace for 2 months followed by a cervical collar.

Conclusion: Treatment of AARF usually consists of reduction by cervical traction followed by fixation using an external orthosis. In our limited experience, we encountered difficulty in keeping pediatric patients in bed rest as well as in cervical and skull traction in this population. On the other hand, AARF was immediately reduced by manual reduction under general anesthesia without difficulty. In AARF in children, manual reduction under general anesthesia may be the procedure of choice for early ambulation rather than keeping them on the bed rest for cervical and skull traction too long.
Search for Major Candidate Genes for Human Neural Tube Defects

Marcy C. Specer, PhD, Elizabeth Melvin, MA, Gordon Worley, MD, Herbert E. Fuchs, MD, PhD, Timothy M. George, MD (Durham, NC)

Neural tube defects are among the most common of birth defects with an overall incidence in the United States of 3/10,000 live births. Although a strong environmental component to their causation has been well established via the reduced risk following preconceptual folic acid supplementation among other exogenous factors, a significant genetic component still remains to be elucidated. Experimental systems, particularly the murine model, provide candidate genes for investigation in humans. Through a national collaborative effort to identify genes predisposing to NTD development in humans, we have collected families in which one or more member has any form of NTD. We have investigated several candidate genes from the mouse system and genes involved in the folate metabolic pathway in a series of 65 American Caucasian families in which the affected patient has a lumbosacral myelomeningocele. We utilized polymorphic genetic markers within the candidate genes to search for evidence of linkage disequilibrium which would provide evidence for the candidate gene to play a major role in NTD development. These candidates included methylenetetrahydrofolate reductase (MTHFR), human T locus (BRACHYURY), PAX 3 gene, p53, and BRCA1 and all failed to show evidence for linkage disequilibrium, thereby excluding them as major genes for NTD in this population.
Simple Repair of Open Neural Tube Defects*

Carla A. Toms, Teresa Harpold, MD, J. Gordon McComb, MD, Michael L. Levy, MD *(Los Angeles, CA)*

Objectives: A myriad of techniques have been proposed to repair open neural tube defects (NTD), representing a spectrum of technical complexity. Techniques involving skin grafts, skin flaps, muscle flaps, multiple teams of surgeons, and, at times, multiple surgeries have been proposed. However, at our institution, NTDs are repaired by simply undermining the skin surrounding the defect and approximating it after reforming the neural tube and closing the dura mater. Herein the results of this technique are presented.

Materials and Methods: Available for review were 182 charts of neonates who underwent simple open NTD closure at our institution between March 1975 and January 1997 and form the basis for this study.

Technique: All of the skin, to include that which was dysplastic, was initially kept intact, undermined, and approximated in the midline with redundant skin then being excised.

Results: Of the 182 patients, 19 experienced only fluid buildup at the wound site that resolved with percutaneous aspiration followed by CSF diversion with no subsequent complications. Four patients experienced CSF leakage that, once again, resolved with CSF diversion and local wound care with no further complications. Five patients experienced minor wound dehiscence, which resolved with local care and required neither additional surgical procedure nor prolongation of hospital stay. Three patients experienced wound infections which responded to systemic antibiotics and local wound care with no prolongation of hospital stay.

Conclusions: Simple repair of open NTD is effective. More involved techniques do not appear to involve any advantage over the simple form of closure.
The Timing of In Utero Myelomeningocele Repair: Quantitative Analysis*

Michael J. Drewek, MD, Joseph P. Bruner, MD, William O. Whetsell, MD, Noel B. Tulipan, MD (Nashville, TN)

Objectives: It has been proposed that the myelodysplastic components of myelomeningocele are secondarily damaged as the result of exposure to amniotic fluid, the so called “two-hit” hypothesis. The critical time point at which the secondary insult might occur has not been clearly defined. Our experiment addresses this issue by quantitatively assessing the toxic effects of human amniotic fluid obtained from progressive stages of gestation on organotypic cultures prepared from fetal rat spinal spinal cord.

Methods: Organotypic cultures of 18 day fetal rat spinal cords were exposed to amniotic fluid of varying gestational age (15-38 weeks) for 48 hours. A lactate dehydrogenase (LDH) release assay was performed on the supernatant of each culture to quantify neuronal and glial cell injury. An average of eight cultures from each selected time point were analyzed.

Results: The LDH activity of cultures exposed to amniotic fluid from 34 and 37 weeks was significantly higher than controls (p<0.002 and p<0.025, respectively). Exposure to amniotic fluid prior to 34 weeks did not result in a statistically significant difference.

Conclusions: Amniotic fluid appears to become toxic to cultures of fetal rat spinal cord at around 34 weeks gestation. This finding suggests that closure of the myelomeningocele defect prior to this time point may help prevent further injury to the developing spinal cord.
47 Relationship Between Anorectal Anomalies and Tethered Spinal Cord

Tanvir F. Choudhri, MD, Indro Chakrobarti, BS, Neil A. Feldstein, MD, John Shullinger, MD, E. Sander Connolly, MD (New York, NY)

The association between anorectal malformations and tethered spinal cords has been reported. However, the management of patients with anorectal malformations in terms of screening for tethered spinal cords and operative management remains controversial. In particular, it has been suggested that patients with anorectal malformations should receive prophylactic untethering operations. To assess the development of symptomatic tethered spinal cords in patients with an anorectal malformation, we retrospectively reviewed the case records of 14 patients (mean age at operation 2.8 months) undergoing surgery to correct anorectal malformations between 1976 and 1986 and obtained long-term follow-up (mean follow-up interval 17.7 years). Follow-up data included any history or operation for a tethered spinal cord, and history of scoliosis, GU problems, bowel dysfunction, back pain, neurological, orthopedic or gait disorders. None of the 14 patients developed had undergone surgery for release of tethered spinal cord. A review of the neurological, urological, and orthopedic histories and their relationship to level of anorectal anomaly will be presented.
Split cord malformations (SCM) may occur in conjunction with myelomeningoceles, and are often ignored or misdiagnosed, potentially causing spinal cord tethering. In this paper, we study the incidence and clinical significance of such an association. We have retrospectively reviewed the medical records and radiographs of 20 myelomeningocele patients who had an SCM. These comprised at least 7% of our myelomeningocele patients. Five of the 20 had simultaneous repair of both lesions at birth. The other 15 were diagnosed with the SCM in a delayed fashion (mean age 4.4 years). Clinical presentations which prompted a diagnostic investigation included hypertrichosis (1), pain (2), routine radiographic follow-up (2), neurourological deterioration (10), and progressive scoliosis (5). In 17 of the 20 patients, the SCM involved the placode (hemimyelia) or was within 1 level of it. Fifteen of these were rostral. Five of the 20 patients had hypertrichosis, and 15 of the 20 patients had a bony stem (i.e., type I SCM). Several accompanying spinal dysraphic lesions also contributed to the tethering: thickened (previously inconspicuous) filum terminale (6), syringohydromyelia (5), and a neuenteric cyst with a benign teratoma (1). Arachnoiditis secondary to the prior myelomeningocele repair was found, as expected, in all 15 patients. However, in virtually all patients, there was also evidence of tethering at the level of the SCM. Long-term follow-up showed stabilization of symptoms and signs, and complications and retethering operations were uncommon. Myelomeningocele patients should be screened with clinical examinations looking for hypertrichosis, and with spinal radiographs preoperatively looking for evidence of SCM (bony stem and/or interpediculate widening not due to the myelomeningocele). Intraoperatively, the placode and the rostral spinal cord segment should be carefully inspected for an SCM and other dysraphic lesions. Our work-up and technique will be described.
Developmental Biomechanics of Pediatric Human Cervical Spine

Srirangam Kumaresan, MS, Narayan Yoganandan, PhD, Frank Pintar, PhD, Wade Mueller, MD (Milwaukee, WI)

The biomechanical consequences of surgical alterations to the adult cervical spine are widely studied. Such information is lacking for the pediatric spine. This study investigated the growth and physiological biomechanics of the one-, three- and six-year-old pediatric cervical spine structures. Anatomically accurate three-dimensional finite element models were created representing three different pediatric age group cervical spines. The one-year-old pediatric spine included the cartilages of spinous processes, the bilateral costal and neurocentral cartilages, growth plates, relatively horizontal facet joint orientations, and the absence of the uncinate processes and the uncovertebral joints. The three-year-old pediatric spine included the fusion of posterior synchondrosis, increase in the volume of the annulus fibers, and decrease in the facet joint orientations. The six-year-old pediatric cervical spine (compared to the one- and three-year structures) included the fusion of the costal and neurocentral cartilages, further increase in the volume of the annulus fibers, and decrease in the facet joint orientations. All the spine structures were exercised under physiologic compression, flexion and extension loadings. The responses were compared with the mature adult spine to study the developmental biomechanical changes of the pediatric cervical spine. All pediatric cervical spines were more flexible under all loading modes than the mature adult spine. The one-year-old spine was the most flexible followed by the three- and six-year-old cervical structures under flexion and extension modes. The three-year-old spine was the most flexible under compression followed by the six- and one-year-old cervical structures. These differing biomechanical responses provide a fundamental understanding of the behavior of the pediatric cervical spine structures under normal physiologic loads.
50 Pediatric Intramedullary Spinal Cord Tumors: Is Surgery Alone Enough?

Keith Y. Goh, FRCS, Linda Velasquez, PhD, Fred J. Epstein, MD  
(New York, NY)

Objectives: To determine the value of radical surgery in pediatric patients with intramedullary spinal cord tumors.

Methods: The case records of 44 patients (age 7 months to 16 years) with intramedullary spinal cord tumors who were primarily managed by surgical resection with no adjuvant therapy, were retrospectively reviewed.

Results: 95% (42/44) of tumors were located in the cervical or thoracic spine and were mainly low-grade (95%). Gross total resection was achieved in 73% (32/44) and subtotal or partial in 27% (12/44). Using the modified McCormick scale for functional status evaluation at a median follow-up of 45 (SEM 5.54) months, 11% (5/44) had severe weakness (scale IV-V), 66% (29/44) had mild-moderate deficits (I-III), and 16% (7/44) had bladder and/or bowel dysfunction. Scoliosis was the presenting symptom in 57% (25/44) of patients requiring bracing in 27% (12/44) and surgical fusion in 16% (7/44). The tumor recurrence rate was 18% (8/44) after a mean period of 54 months (SEM 11.34). Age, number of involved spinal cord segments and extent of surgical resection did not significantly impact on tumor recurrence rates (p = 0.28, 0.44 and 0.13, respectively). The only significant prognostic factor was histological type: higher grade tumors recurred sooner than lower grade tumors (p =0.04).

Conclusion: Radical surgery for intramedullary spinal cord tumors in children can achieve long tumor-free survival, especially in low-grade lesions, without adjuvant therapy. Because of the intramedullary nature of the tumor, there is almost always some residual neurologic deficit.
Spinal Cord Gangliogliomas: A Review of 55 Patients

George I. Jallo, MD, Diana Freed, BS, Gary Breslow, MD, Peter Alexander, MD, Fred J. Epstein, MD (New York, NY)

The current understanding of the treatment and management of patients with spinal cord gangliogliomas is derived from studies in which these tumors are located in the cerebral hemispheres. The records of 55 patients with spinal cord gangliogliomas surgically treated between January 1985 and December 1995 were retrospectively reviewed to determine the long-term survival, event-free survival and functional outcome following radical resection. There were 19 females and 36 males, ranging in age from 6 months to 18 years (mean, 5.6 years). Sixteen patients were 3 years of age and under. The tumors were found throughout the spinal axis; 10 were cervicomedullary, 6 cervical, 19 cervicothoracic, 16 thoracic, and 4 in the conus medullaris. Thirty-one patients had their initial procedure performed at an outside institution prior to referral to NYU Medical Center. Forty-three had gross total resection and 12 had radical subtotal resection. Only 8 patients underwent postoperative irradiation or chemotherapy and, therefore, the outcome was generally related to surgery alone. There were no operative deaths. The median follow-up period was 74 months. The 5-year actuarial survival rate was 88% and the 10-year survival rate 77%. The event-free survival rate at 5 years was 67%. There were 17 recurrences in this population within a median time of 27 months. Neurological function at recent follow-up evaluation was stable or improved in 72% of patients. Statistical analysis did not reveal any variable predictive for survival in patients with spinal gangliogliomas. It is concluded that radical surgery leads to long-term survival of patients with gangliogliomas, and adjuvant therapy can probably be reserved for recurrence or special cases.
Impact of Selective Dorsal Rhizotomies on Urodynamic Function in Cerebral Palsy

Jean-Pierre Farmer, MD (Montréal, Québec, Canada), Olivier Vernet, MD (Lausanne, Switzerland), Anne-Marie Houle, MD, José Luis Montes, MD (Montréal, Québec, Canada)

To evaluate bladder function in cerebral palsy, we performed preoperative urodynamic studies (UDS) on 27 patients (15 boys, 12 girls; mean age 5 years) undergoing selective dorsal rhizotomies for spasticity. None exhibited preoperative urinary symptoms. Only 5 of the 27 studies were considered normal. All others demonstrated low bladder volumes (total, 20 below and 30 below bladder capacity) and high full resting pressure compared with age-matched normal controls. To date, after a mean follow-up of 1 year, 8 patients have been reevaluated postoperatively. They all demonstrate a significantly increased total, 20 below and 30 below bladder capacity. Full resting pressure tends toward lower values, without reaching statistical significance. We conclude that cerebral palsy patients have clinically silent neurogenic bladders and the rhizotomy, just as it improves tone in the lower extremities, tends to improve bladder capacity, hopefully, to the point of reducing the risk of upper urinary tract deterioration.
Quantitation of Spasticity in Rhizotomy Patients with Combined EMG-Joint Motion Analysis

Mark G. Luciano, MD, PhD, Julee E. Perry, MS, Brian Davis, PhD, Alan Gurd, MD, Jennifer J. Ahl, BSN, CNRN (Cleveland, OH)

Spasticity that limits function and comfort in children with cerebral palsy can be reduced with selective dorsal rhizotomy. Because the degree of spasticity and its impact on movement can be difficult to assess, the selection of patients who may benefit from a rhizotomy and evaluation of outcome can be problematic. The aim of this study is to use a new approach that combines joint motion and EMG analysis to quantitated spasticity in both ambulatory and non-ambulatory patients.

Method: Four non-ambulatory children (ages 4-16) with cerebral palsy were studied before and at 6-8 weeks after a selective dorsal rhizotomy. Four normal children were also studied. Each patient was asked to lay supine and draw their legs to their chest using hip and knee flexion, followed by extension. During this movement cycle 3-D kinematic motion analysis and EMG data were combined to yield percent of time specific muscles were acting isometrically, concentrically, eccentrically, or were at rest. It was hypothesized that spasticity would result in an abnormal pattern of muscle activity in relation to joint movement such as decrease off time and increased eccentric activity. Data was compared to our routine clinical evaluation of the patient as well as to physical therapy evaluation with Ashworth and GMFM scoring.

Results: Of the vastus lateralis and biceps femoris monitored during this activity cycle, it was the latter that showed the most dramatic changes with significantly lower percent off time in spastic patients compared to control (42% vs 73%) and a return to normal levels after rhizotomy (79%). Complete normalization of increased concentric activity and isometric activity was also seen after rhizotomy. No significant abnormalities or changes were seen in percent of eccentric muscular activity. All 4 of these patients showed decrease in spasticity and clinical improvement in joint motion. Physical therapy analysis also showed a moderate but significant increase in GMFM scores after rhizotomy (47.9 ± .8 to 52.3 ± .8, P=.028). However, there was no detectable difference seen on Ashworth scoring.

Conclusion: The combination of 3-D kinematic joint motion analysis and EMG can demonstrate significant abnormalities in simple movement tasks in children with cerebral palsy. These abnormalities are quite dramatically normalized by selective dorsal rhizotomy. This technique shows promise in the quantitation of spasticity and may aid in screening and evaluating treatment.
Separation of Craniopagus Twins*

Keyvan Abtin, MD, Marion L. Walker, MD (Salt Lake City, UT)

The incidence of conjoined twins is roughly 1:50,000 live births worldwide with only 2% of these as craniopagus variant. Among the craniopagus twins, there are variations among the location of conjoined skull as well as the structures shared intracranially. Multimodality radiographic studies are required to assess the complexity of such structures for surgical separation. One relatively common finding in these twins is the presence of a common or partly anastomosed venous drainage system, which would potentially complicate the separation procedure. We have had experience in separation of three sets of craniopagus parietalis-type twins over the past twenty years. Our experience has guided us in staging the separation procedure in order to minimize the complications, especially those associated with this shared venous system. This may also allow for establishment of collateral flow in between stages of the surgery and thus possibly decrease the risk of venous infarction. Other possible approaches should include the use of cardiopulmonary bypass and hypothermic circulatory arrest. In our latest set of twins, they both are doing well at age 22 months, approximately 10 months after the final stage of their separation, although both required shunts for CSF diversion. We will share our experience and findings.
Surgery of Chiari I malformations (ChiM1) must account for both ventral and
dorsal compression of the cervicomedullary junction (CMJ). Plain radiographic mea-
surements quantify rostral displacement of the odontoid, but fail to detect ventral
compression (VC) of the CMJ without rostral migration of the odontoid. A method
of quantifying VC using a sagittal magnetic resonance image (MR) of the cranio-
cervical junction was developed so as to objectively measure VC in these patients and
correlate this with imaging and clinical features. The MR of 40 consecutive patients (1.3
to 27.4 years old; mean 8.6 years) with ChiM1 who were referred to our pediatric
neurosurgical unit over a three-year period were reviewed. A line (B-C2) was drawn
from the basion to the caudal/posterior aspect of the C2 body. The distance of a
perpendicular (pB-C2) from the line B-C2 through the odontoid tip to the ventral
CMJ dura was determined and served as an objective measurement of VC. Subject-
ive grades of VC (absent, mild, moderate) on the sagittal MR were correlated with
pB-C2 distances, patient age, distance of tonsillar descent, odontoid relation to
McRae 5 and Chamberlain 5 lines, and incidence of syringomyelia and scoliosis. Ten,
19, and 11 patients had absent (set A), mild (set B), and moderate (set C) VC,
respectively. Means of set A characteristics compared to sets B and C were younger
age (6.3 vs. 8.9 and 10.3 years), shorter pB-C2 distances (4.1 vs. 7.2 and 9.1 mm),
and less tonsil descent (7.1 vs. 12.9 and 18.5 mm), respectively, (p<0.05). No
difference existed in the incidence of syringomyelia or scoliosis among sets (p>0.1).
No one with pB-C2 < 5.5 mm had evidence of VC. All with a pB-C2> 6 mm had
MR evidence of VC. Greater pB-C2 values correlated strongly with the presence of
VC (p<0.001). Despite 75% (30/40) of patients having VC, none had an odontoid
above McRae 5 line, and only three had an odontoid greater than 4 mm above
Chamberlain 5 line, indicative of basilar invagination. Of 12 patients with pB-C2 of
9mm or greater, four had the ventral vector reduced by either traction (n=2), or
transoral odontoidectomy (n=2). One child with a pB-C2 of 10 mm but no basilar
invagination experienced clinical deterioration following PFD and fusion, requiring
a ventral decompression. The remaining seven were treated successfully with PFD
alone. VC with or without basilar invagination is very common in children and
young adults with ChiM1. The pB-C2 provides an objective measurement of VC.
The majority of patients with moderate VC are treated successfully with PFD alone.
In children and young adults with ChiM1 and a pB-C2 of 9 mm or greater however,
strong consideration should be given to either indirectly (e.g. traction and fusion) or
directly (e.g., ventral decompression) addressing the VC so as to prevent neurological
deterioration following PFD.
56 Toward a Simpler Surgical Management of the Chiari I Malformation

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

Objectives: 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. These include various shunting procedures, duroplasty, obex plugging, and resection of the cerebellar tonsils. Our practice has been to avoid these adjuvants, and to perform a simple occipital craniectomy, C-1 laminectomy, and dural opening. The dura mater is left open and overlain with oxidized cellulose.

Methods: To evaluate the efficacy of this more limited procedure, a retrospective review was performed of the medical records of 31 consecutive patients treated over a 6-year period. Twenty-six (84%) of these patients had an associated spinal cord syrinx; all underwent the same procedure.

Results: Follow-up ranged from 6 to 72 months, with all patients having at least one postoperative MRI. Twenty-three of the 26 patients (88.5%) who presented with a syrinx had significant resolution of the syrinx on follow-up scans with concomitant improvement of presenting signs and symptoms. Of the remaining three patients, one had progressive hydrocephalus and received a ventriculoperitoneal shunt, with symptom resolution. In the other two patients the syrinx did not diminish, although they remained clinically stable; one received a syringopleural shunt, and the other has been followed for only 6 months without change. Postoperative morbidity includes a 26% incidence of headaches, of which half resolved within 5 days and only one persisted beyond 2 weeks. Nausea and vomiting occurred in 16%. Neither of these figures significantly exceed those of other large surgical series in which the dura mater was closed with a patch graft. Three patients (9.7%) did have a postoperative CSF leak; all responded to bedside suturing without further sequellae.

Conclusions: This study indicates that a simple bone and open dural decompression of the cervicomedullary junction is a safe, effective operative treatment for Chiari I malformation in children. Shunts, duroplasty, obex plugging, and tonsillar resection do not appear to improve the outcome when our series is compared to others in which such adjuvants were used.
The Chiari Zero Malformation

Bermans J. Iskandar, MD (Madison, WI), Gary L. Hedlund, DO, Paul A. Grabb, MD, W. Jerry Oakes, MD (Birmingham, AL)

We describe 5 cases of syringohydromyelia without hindbrain herniation. Preoperative MRI scans with and without Gadolinium-DTPA revealed no evidence of spinal cord tumor, arachnoiditis, or spinal dysraphism. Craniocevical decompression was performed in all patients, after which there was resolution of the symptoms in the 4 symptomatic patients, and marked reduction in the size of the syrinx in all 5 patients. We hypothesize the rare occurrence of syringohydromyelia resulting from a Chiari-like pathophysiology but lacking a hindbrain hernia. Such patients may benefit from a craniocevical decompression.
Chiari II Malformation: Lesions Found Within the Fourth Ventricle

Joseph Piatt, MD, Anthony D. Agostino, MD (Portland, OR)

Objective and Importance: Decompression of the Chiari II malformation may be undertaken to treat symptoms and signs of hindbrain compression, to treat associated syringomyelia, and to prevent complications of surgery for scoliosis. Whatever the indications, the author's usual practice has been to open the fourth ventricle to restore normal CSF flow patterns. A variety of structural lesions have been discovered at this site. Fourth ventricle pathology associated with the Chiari II malformation has received scant attention in the literature.

Clinical Presentation: Structural lesions associated with the Chiari II malformation have been identified in 7 patients. During the 42-month period encompassing the operations of the 6 patients treated surgically, only 7 other patients were explored without the discovery of some associated structural lesion. The patients ranged in age from 2 to 26 years. The following lesions were identified: glial or arachnoidal cysts (3 patients), glial or choroidal nodules (3 patients), and subependymoma (1 patient). These lesions were all situated in the roof of the fourth ventricle adjacent to or interspersed with the choroid plexus. Only the cystic lesions were identified by preoperative imaging. In only one case did the associated lesion, a 2 cm cyst, seem to contribute to the patient's clinical presentation.

Conclusion: Structural lesions of the fourth ventricle associated with the Chiari II malformation are common in patients who are submitted to decompression. Whether these lesions are dysplasias of developmental origin or reactive lesions related to chronic compression and ischemia is unknown. They do not necessarily require biopsy or excision.
Treatment of Intracranial Aneurysms in the Pediatric Population

Howard A. Riina, MD, Alejandro Berenstein, MD (New York, NY), Eugene S. Flamm, MD, Leslie Sutton, MD (Philadelphia, PA)

Introduction: Intracranial aneurysms in the pediatric population are rare, comprising approximately 0.5% - 4.5% of all aneurysms. Many of these aneurysms are complex in nature, and have significant morbidity and mortality despite recent improvements in surgical care.

Methods: We retrospectively analyzed patient characteristics, treatment and outcome data in this cohort. Twenty-four pediatric patients with complex intracranial aneurysms were treated from 1974 to the present. Etiology includes saccular, infectious, traumatic, iatrogenic and lesions associated with connective tissue disorders.

Results: Age ranged from 3 months to 20 years, and 75% of patients were males. Fifteen patients underwent surgical treatment of their aneurysm. Seven patients received endovascular treatment. Thirteen patients treated with surgery (86.6%) had a good outcome with either a transient neurologic deficit or no deficit. Six patients treated endovascularly had a good outcome (85.7%), 1 patient with underlying systemic disease underwent successful endovascular treatment, but expired from medical complications. One patient expired prior to treatment. One patient had a good outcome after spontaneous thrombosis of the aneurysm.

Conclusions: Factors that predict outcome of pediatric patients with intracranial aneurysms include location, size, neurologic condition at presentation, mass effect, subarachnoid hemorrhage, and vasospasm. Recent improvements in endovascular technology allows this approach in many patients, even in extremely young infants. Decisions regarding treatment approach must be determined on a case by case basis with consideration of the available technology and experience of the physicians involved.
Cerebral Aneurysms in Children: A Series of 39 Patients*

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

Objectives: We present a group of 39 pediatric patients with intracranial aneurysms that were treated at our institution over a 15-year period.

Methods: Medical records and radiographic studies for 39 consecutive patients were reviewed.

Results: The patients ranged in age from 6 months to 19 years (mean 10.9). Of the 39 cases, 11 were traumatic and 2 were mycotic. Thirty-five of the cases were ruptured at the time of diagnosis. There were 2 cases with multiple aneurysms for a total of 41 lesions. Subarachnoid hemorrhage was the most common mode of presentation (28) with the average Hunt-Hess grade being I-II. The location of the lesions was as follows: middle cerebral, 8; internal carotid, 6; anterior communicating, 5; posterior cerebral, 5; posterior communicating, 4; pericallosal, 3; anterior cerebral, 2; choroidal, 2; posterior inferior cerebellar, 2; basilar, 2; vertebral, 1; and frontopolar, 1. Treatment was accomplished through various combinations of clipping, wrapping, excision, bypass, and endovascular techniques. Intraoperative complications included rupture in 6 cases, progressive cerebral edema in 2, and perforating arterial injury in 4 patients. Postoperative complications were as follows: new hemiparesis, 2 patients; monoparesis, 2; cranial nerve palsies, 3; delayed ischemic stroke, 3; and infection, 1. Clinical vasospasm was encountered in 7 of our patients, but no cases were observed in those younger than age 9 (12 cases). Postoperative angiography was obtained in 35 cases with excellent to good results in 33 and fair in 2. Long-term outcome was excellent in 18, good in 8, and fair in 11 with one death and one case lost to follow-up.

Conclusion: Analysis of our data suggested a predilection for the posterior circulation, larger size, more complex architecture, and a decreased risk of clinical vasospasm in the younger age group. This series and a review of the literature suggest that aneurysmal disease in children may be distinct from the same lesions in adults.
Pediatric Arteriovenous Malformations: A Multi-Modality Approach

Kevin M. Cockroft, MD, Mary L. Marcellus, RN, Michael P. Marks, MD, Stephen L. Huhn, MD, Gary K. Steinberg, MD, PhD (Stanford, CA)

We reviewed the clinical and radiographic features of 51 consecutively treated pediatric patients (<18 yrs) with high-flow AVMs treated between 1988 and 1996. The mean age at presentation was 10.3 years (range 2 mo - 18 yrs). Most patients presented with intracranial hemorrhage (28 of 50, 56%). The remainder presented with progressive neurological deficit (10, 20%), seizures (5, 10%) or headache (6, 12%), while one patient (2%) was asymptomatic. The majority were Spetzler-Martin grade III - VI (59%). Mean time from presentation to treatment was 12 months (range 0 - 84 mo). Therapy included microsurgery, endovascular embolization and/or stereotactic radiosurgery. Nine patients (18%) were treated with all 3 modalities, 21 (41%) with 2 and 21 (41%) with one modality. Higher grade AVMs (III - V) underwent multi-modality therapy more frequently (67% vs 54%) and had more stages of treatment (3.3 vs 1.8) when compared to lower grade lesions. The mean duration of therapy was 15 months (range 0 - 113) and for 20 patients (39%) treatment remains ongoing. Radiologic follow-up was available in 43 of 51 patients. Of these, 18 patients (42%) demonstrated complete angiographic obliteration while 21 (49%) showed significant (>10%) reduction in AVM volume. Clinical follow-up was available for 47 patients (mean 26 mo, range 1 - 132 mo). Forty-three patients (91%) had good to excellent outcomes. One patient (2%) had a poor outcome and 3 (6%) died.

AVMs in the pediatric population can be particularly challenging to treat and onset of therapy is often delayed. Despite this, these results demonstrate that good clinical and radiographic results can be achieved with a flexible multi-modality approach.
62 Use of Intraoperative Angiography in Pediatric Vascular Abnormalities

Sanjay Ghosh, MD, Michael L. Levy, MD, Stephen L. Giannotta, MD, J. Gordon McComb, MD (Los Angeles, CA)

We reviewed our experience with intraoperative angiography in 13 patients with cerebral aneurysms and 14 with arteriovenous malformations who presented over the past 5 years. We have incorporated the use of intraoperative angiography into all pediatric vascular cases and will describe the indications, benefits and complications of these procedures.

There were a total of 14 males and 13 females in the study. Mean age was 9 years. For those patients with ruptured aneurysms, surgery was completed on a mean of 5.6 days following rupture. There were 4 internal carotid artery bifurcation aneurysms, 1 pericallosal aneurysm, 2 middle cerebral artery trifurcation aneurysms, 1 posterior communicating artery aneurysm, 1 vertebral artery aneurysm, 1 basilar aneurysm, 1 P1 aneurysm and 2 P2 posterior cerebral artery distribution aneurysms. No patient undergoing intraoperative angiography had a vascular complication related to the intervention. A groin hematoma was documented in only 1 patient. No arterial dissections or thromboses were reported for any patients in the series and no infectious complications were noted. There were no technical problems. All patients were able to undergo intraoperative imaging, which greatly facilitated the operative outcome. Clip placement was modified in 4 patients as a result of the angiogram and complete resections of the arteriovenous malformations were maximized in 3 patients. Operative duration was increased. In no case was residual AVM noted at follow-up.

In conclusion, intraoperative angiography is a safe and reliable procedure for use in the treatment of intracranial vascular lesions in children. Although it does increase the duration of the operative intervention, no additional morbidity or mortality was related to its use and outcomes could only be maximized through the use of concurrent intraoperative imaging.
fMRI Assessment of the Relationship Between Eloquent Cortical Function to Cerebral Lesions

Elaine T. Kiriakopulos, MD, James T. Rutka, MD, PhD, James M. Drake, MD, PhD, Harold J. Hoffman, MD, Robin P. Humphreys, MD, David J. Mikulis, MD (Toronto, Ontario, Canada)

Objective: This study was designed to determine the utility of fMRI in localizing the proximity of eloquent cortex to cerebral lesions in pediatric neurosurgical patients.

Methods: fMRI was performed using a 1.5T conventional MR system to localize motor, sensory, speech and visual areas in neurosurgical patients (n=25). The age range of this pediatric patient population was 22 months to 18 years. Activation paradigms included motor, sensory, language and photic stimulation. Statistical analysis (p<0.0001) of the functional data consisted of t-test comparisons between resting and task images.

Results: In 23/25 patients fMRI activation maps were produced. Two patients were unable to complete the activation protocol. In 23/23 patients the relationship between eloquent cortex and lesions was clearly demonstrated and aided in surgical planning. Intraoperative electrocortical stimulation was correlated with fMRI data to examine the reliability of fMRI brain maps.

Conclusion: The collection of accurate data using fMRI paradigms designed for children is demonstrated. We highlight the utility of single slice data in localizing eloquent cortex. This represents the largest single collection of pediatric cases studied with fMRI and includes the youngest reported patient studied, at 22 months of age.
Limbic Resection Through Parahippocampal Gyrus for Treatment of Mesial Temporal Lobe Epilepsy in Children: Preliminary Outcome

T.S. Park, MD, Blaise F.D. Bourgeois, MD, Edwin W. Dodson, MD, Susa Arnold, MD, Bruce A. Kaufman, MD (St. Louis, MO)

Introduction: Among accepted surgical options for treatment of medically refractory mesial temporal lobe epilepsy is amygdalo-hippocampectomy (AH). AH has been performed via the sylvian fissure, the superior temporal sulcus, the middle temporal gyrus and the fusiform gyrus. Although AH via the sylvian fissure and middle temporal gyrus is well known, the operation results in extensive injury of the temporal stem. In contrast, AH via the parahippocampal gyrus that we have described (1) can spare the fusiform gyrus and the rest of temporal lobe.

Material and Results: We have thus far performed the trans-parahippocampal AH on 15 children (age: 10-19 years). Dominant temporal lobe was involved in 10 patients. Pathological examination revealed mesial temporal sclerosis in 7 patients and a glioma in one patient. In a follow-up of 1 to 36 moths, 87% were seizure free and 13% had greater than 80% reduction of seizures. Morbidity included transient memory impairment in one and dysnomia in one patient.

Conclusion: Our experience indicates that the trans-parahippocampal AH can be safely performed; it is particularly suitable for pediatric patients; and seizure outcome following the operation is promising.

Temporal lobectomy is among the most successful surgical techniques for control of chronic seizures. There are several differences, however, between adults and children with epilepsy of temporal lobe origin. In contrast to the adult population where the most common pathology is mesial temporal sclerosis, tumors and heterotopias are frequently encountered in children. Zones of developing language, especially in bilingual children, may be more widely variable than in adults requiring extensive localization studies. Over the last seven years, we have completed temporal lobectomy in 47 children, ages 2 months to 19 years. Preoperative studies included MRI, scalp and telemetric EEG, intracarotid amobarbital injection in all children over 5 years of age, ictal and inter-ictal SPECT and neuropsychological testing. Subdural grid and strip electrodes were placed for electrocorticography and language localization in 28 patients. Preoperative MRI revealed temporal lobe mass lesions in 22 patients and architectural dysplasia in another six patients. Diffuse white matter gliosis and loss of hippocampal neurons was encountered in the other patients. Each tumor and region of heterotopia was removed in its entirety and at least 1 cm of the hippocampus was resected during each procedure.

All but one patient remains seizure free with mean follow-up of 28 months, and anticonvulsants have been withdrawn in 31 patients. There have been no postoperative language deficits; postoperative infections occurred in 2 patients following invasive grid monitoring for 9 days.

Temporal lobectomy is a very successful technique for lesion or non-lesion epilepsy of temporal lobe origin. When MRI imaging and EEG recording data are concordant, we suggest that invasive monitoring should be reserved for the localization of dominant language cortex in patients too young for awake resections. Invasive subdural strip and grid monitoring is also warranted when scalp electrode recording has not sufficiently localized the ictal focus.
Corpus Callosotomy in the Treatment of Intractable Childhood Epilepsy

Nejat Akalan, Assoc. Pro., Güzide Turanlı, Assoc. Pro., Yavuz Renda, Professor, Vural Bertan, Professor (Ankara, Turkey)

Objective: Corpus callosotomy for the management of intractable epilepsy is an uncommonly performed operation. The indications and surgical principles are controversial. The purpose of this study is to analyze the results of corpus callosotomy in 15 children with intractable epilepsy.

Methods: A retrospective review of children was performed undergoing anterior two-thirds corpus callosotomy and anterior comissurotomy. Fifteen patients were identified between 1994 and the present. The mean age of the patients was 7.2 years with the range being 3 to 15 years. Evaluation of the cases including neurodiagnostic work-up, EEG-video monitoring, neuropsychological testing and surgical decision were based on a protocol established by our pediatric epilepsy group.

Results: All cases had drop attacks prior to surgery. In addition, 10 cases further had a combination of either complex partial or simple partial seizures and 5 had tonic-clonic seizures refractory to anti-epileptic medication. Slight to moderate mental retardation was present in 8 cases. The number of seizures ranged from 60 to 800 per month with a duration of 2 to 8 years. Of 15 cases, 6 had cryptogenic Lennox-Gastaut syndrome. The follow-up period ranged from 7 to 41 months (mean=3±22 months). The results were evaluated according to Spencer's classification. Three (20%) cases were seizure free after surgery while 8 (53%) had drastic decrease in seizure frequency. Four (27%) had no improvement.

Conclusion: Corpus callosotomy can provide a significant seizure control in intractable drop attacks and improvement in life quality in carefully selected cases.
Pediatric Hemispherectomies: Is a Shunt Needed?

Omar Jimenez, MD, Glen Morrison, MD, Prasana Jayakar, MD,
Michael Duchowny, MD, Maria Penate, RN (Miami, FL)

The role of hemispherectomy in the treatment of childhood epilepsy is well established. Less well established is the technique (anatomical, functional, corticectomy, etc.) and the need for a cerebral spinal fluid diversionary procedure (a shunt). Some pediatric neurological surgeons have said that most (or at least 50%) of their patients require and receive a shunt at the initial operative procedure or shortly thereafter.

This report is of 34 hemispherectomies from a series of 239 children who have undergone 391 operations for their intractable epilepsy. Forty-eight percent of these operations were cortical resections, 37% were the placement of subdural electrodes for extraoperative monitoring, and 9% were hemispherectomies.

There were 19 males and 15 females. Six children (18%) were less than 1 year of age, 16 (47%) between 1 and 5, 5 (15%) between 5 and 10, and 7 (20%) between 10 and 21 years of age. Thirty children had a “functional” hemispherectomy and the right side was operated on in 19 cases.

Twenty nine children were seizure free (85%) but some variable seizure activity was present in 5 children. The follow-up has been < 1 year in 4, 1-5 years in 14, and 5-10 years in 12. NOT ONE CHILD WAS SHUNTED!

There have been significant complications/untoward events. Operative complications included a subdural hematoma and a cerebral vascular accident. One child was reoperated at another institution and remains in coma. Four children have died (12%). One drowned in her pool some years later and one severely delayed child died of respiratory problems 2 years after surgery (seizure free). One child developed contralateral seizures and died of sepsis one month later. Finally, one child presented to a hospital in a distant city with what was most likely acute hydrocephalus and died without treatment.

In conclusion, this series does not support the concept of a need for a shunt in the vast majority of hemispherectomies done in childhood. This may, or may not, be related to the primary use of a functional, as opposed to anatomical, hemispherectomy. However, the development of a dynamically important hydrocephalus needs to be considered in all children following hemispherectomy and did develop as a delayed phenomenon in one of our cases and, had it been treated, the child may still be alive.
Ventriculoscopic Procedures Using Frameless Stereotactic Guidance

Stephanie L. Einhaus, MD, Michael S. Muhlbauer, MD, E. Christopher Troup, MD, Robert A. Sanford, MD (Memphis, TN)

Introduction: Recent improved technology has brought a resurgence of interest in using endoscopes to perform minimally invasive neurosurgical procedures. There are two main drawbacks in the use of a ventriculoscope: 1) There is a significant learning curve in mastering its use; and 2) It is easy for even experienced surgeons to become spatially disoriented, especially if the anatomy is distorted. Frameless stereotaxy is a logical adjuvant for these procedures. There is concern about significant shifting of anatomy and resultant misleading data when large fluid spaces are entered. The purpose of this study was to evaluate the use of frameless stereotaxy coupled with ventriculoscopy.

Methods: Ten patients with hydrocephalus complicated by ventricular loculations or arachnoid cysts were treated with ventriculoscopic techniques aided by “STEALTHStation” frameless stereotactic guidance. Throughout the procedure assessments were made comparing the “real time” anatomical target with the “predicted” target on the computer generated image.

Results: Six patients were treated for multiloculated ventricles and four for arachnoid cysts. The goals set preoperatively for all patients were achieved except in one patient in which it was elected to abandon the procedure for technical reasons. The accuracy of the “predicted” targets with the “real time” targets was remarkably good throughout each case and estimated to be <1 to 1.5 cm.

Discussion: Some shifting of anatomic structure does occur with ventriculoscopic procedures, but the mild loss of precision experienced with this combined technique was not found to significantly affect its usefulness.

Conclusion: Frameless stereotaxy can be a highly valuable adjuvant to complex ventriculoscopic procedures.
Concurrent 3-Dimensional Endoscopy: A Preliminary Laboratory Experience

Joseph Chen, MD, Michael L. Levy, MD, J. Gordon McComb, MD
(Los Angeles, CA)

We describe our experience in the neurosurgical pediatric skull based laboratory with a 3-dimensional endoscope and concurrent picture picture display into a head mounted display system for use in the subarachnoid space.

The stereo endoscope measures 4.7 mm in diameter and has a fixed working distance of 15 mm. The endoscope viewing angle is 30 degrees with a 70 degree field of view (FOV). A permanently mounted light guide is positioned on the shaft which rotates the viewing angle 360 degrees while maintaining a “12 o’clock” image orientation. The telescope consists of single rod lens channel and a stainless steel shaft of 150 mm in length. The stereo image resolution is 768 x 494 pixels using dual 1/4" charged coupling devices. The endoscope allows for a split image display with each image being projected to an independent LCD in the head mounted display system. Dual picture and picture interfaces into the HMD allow for concurrent visualization of the 3-dimensional image while simultaneously visualizing the microscopic anatomy through the via the optics of the operating microscope. The primary intention of the study was to establish operative corridors and allow for concurrent visualization of contralateral anatomy using the 3-dimensional scope and evaluating the use of the scope with regard to the ability of the surgeon to perceive 3-dimensional relationships as opposed to standard visualization techniques using television monitors and 2-D endoscopes.

We will describe the construction and intraoperative use of the endoscope in a series of 6 cadaver heads. We will further discuss the facility of use and the importance of the integration of 3-D endoscopic images in head mounted display systems to increase the facility of microsurgical manipulation and dissection.
Children with Central Diabetes Insipidus Secondary to Nonspecific Inflammatory Disorders of the Pituitary and Hypothalamus

Donald P. Atkins, MD, Robert A. Sanford, MD, Lela Ostby, RN  
(Memphis, TN)

Two children presenting with diabetes insipidus at our institution were found to have enhancing lesions of the hypothalamus on MRI. Although diabetes insipidus was permanent in both patients, each had resolution of the MRI abnormality over a period of years with no specific treatment. One underwent open biopsy shortly after discovery of the abnormality which revealed a chronic inflammatory infiltrate.

Only two similar cases were found on a literature review, one adult and one child, and in each the process was self-limited. Fifteen to thirty percent of nonsurgical diabetes insipidus is classified as idiopathic. MRI, however, is improving the sensitivity for diagnosing anatomical abnormalities in these cases. In a review of 17 adults, Imura found MRI abnormalities of the posterior pituitary or infundibulum in all patients. Two biopsies in his series revealed inflammatory infiltrate. The authors suggest that “infundibuloneurohypophysitis” is a self-limiting process which can be managed successfully medically.

Two cases of hypothalamic inflammatory lesions in children with diabetes insipidus will be presented. The literature related to inflammatory disorders of the pituitary and hypothalamus will be reviewed.
SCIENTIFIC POSTERS*

*Considered for Shulman Award.*
Chemical Analysis of Fluid from Intracranial Arachnoid Cysts

Todd A. Maugans, MD, J. Gordon McComb, MD, Michael L. Levy, MD (Los Angeles, CA)

Objectives: Arachnoid cysts become symptomatic via their progressive enlargement and attendant mass effect. The reasons for their growth have not been elucidated, however, the chemical composition of the cyst fluid may play a role in this process. Previous investigations of the composition of cyst fluid are few and reveal conflicting results. This study attempts to further characterize the chemical composition of intracranial arachnoid cyst fluid.

Methods: Fluid was collected from the intracranial arachnoid cysts of seven patients who underwent craniotomy and fenestration procedures during the past twenty-four months. The fluid was assayed for electrolytes, osmolality, glucose, total protein and cell count.

Results: Electrolyte content of the cyst fluid was similar to reported normative values for ventricular and cisternal cerebrospinal fluid (CSF) with mean values (mEq/L) as follows: sodium 145, potassium 4.0, chloride 116, bicarbonate 20. The osmolality and glucose contents were also similar to CSF with values of 284 mOsm/L and 57 mg/dL, respectively. In contrast, the total protein content was significantly higher than reported values for ventricular and cisternal CSF with a mean of 119 mg/dL. This elevation could not be attributed to contamination by blood.

Conclusions: The general chemical composition of fluid from intracranial arachnoid cysts resembles normal CSF. The elevation in total protein, however, may be implicated in the expansion of these collections. The walls of the cysts may act as semipermeable membranes and impede the efflux of proteins. The colloid osmotic gradient created would foster the retention of CSF and produce progressive enlargement.
Nonsurgical Treatment of Post-meningitis Subdural Empyema

Márcia C da Silva, MD, MSc, Christovão C. Xavier, MD, Andrea L. Oliveira, MD, Juliana E. Gurgel, MD, Valeria M. Lcal, MD (Belo Horizonte, Brazil)

Objective: The authors report on the incidence of subdural collections and empyemas on a series of 527 consecutive children admitted in a period of 20 months for the treatment of bacterial meningitis.

Methods: Collections were suspected due to persistent fever, delayed onset seizures, signs and symptoms of increased intracranial pressure or worsening of clinical status. Diagnosis was made based on CT scan and subdural tap findings. Fifty-one children presented with symptomatic subdural collections during treatment. Thirty-seven of them that presented with a contrast-enhanced capsule on CT scan and elevated protein and cell count on the subdural fluid were diagnosed as having subdural empyemas. Treatment consisted of changing the venous antibiotic to a third-generation cephalosporin that was maintained for another 4-6 weeks. Patients were followed closely with serial CT scans up to 24 months after treatment completion.

Results: The majority of children treated this way showed prompt clinical improvement plus a progressive decrease of the subdural collection until its complete disappearance. From the thirty-seven children only two had to be submitted to a surgical evacuation of the empyema due to failure of the nonsurgical treatment. No variable studied significantly influenced the efficacy of the clinical treatment.

Conclusion: Subdural empyemas due to bacterial meningitis in infancy can be treated in the majority of cases with venous antibiotics only, with good resolution of the collection. If surgical treatment is necessary, the delay does not seem to adversely affect the outcome of the children.
Evidence That Oxidative Stress Contributes to the Pathophysiology of Hydrocephalus in a Hydrocephalic Rat Model

Debra Socci, PhD (Orlando, FL), H. C. Jones, PhD (Gainesville, FL), Jogi V. Pattisapu, MD, FAAP (Orlando, FL), K. Blugstad, G. W. Arendash, PhD (Tampa, FL)

Oxidative stress can contribute to many neurological disease processes. The present study evaluated the potential role of reactive oxygen species (ROS) and oxidative stress in the pathophysiology of hydrocephalus. We used a rat model (HTx) with a hydrocephalus inheritance rate of 20-50% (with careful breeding). Normal and hydrocephalic brains of these rats (n= 5-8) were dissected, fresh frozen, and stored at 70°C. Left cortex, hippocampus, and cerebellum were then assayed for protein and ROS levels, using an assay measuring desacetylation and oxidation of 2’, 7’-dichlorofluorescin diacetate (DCFH-DA) to dichlorofluorescin (DCF).

The generation of fluorescent DCF from DCFH-DA was determined fluorometrically in sonicated tissue, after a 15 minute incubation. ROS levels were measured at 0, 30, 60, and 90 minutes, and at 24 hours post-incubation.

ANOVAR showed that there was significant interaction between groups for the hippocampus when the 0-24 hour time points were compared. Post-hoc t-tests revealed that at all the 0-90 minute time points, hydrocephalic rats had significantly more ROS compared to controls. ANOVAR showed no significant group differences in the left cortex or cerebellum. Linear regression analysis revealed that the rate of ROS formation was significantly higher in the hippocampus and cerebellum of hydrocephalic rats compared to controls. Thus, ROS generation and oxidative stress may be involved in the molecular pathophysiology of hydrocephalus in the HTx rat.

(Supported by Wade Center for Hydrocephalus Research.)
Two cases of Chiari II malformation associated with occipital encephaloceles are presented. In addition to the encephalocele, both patients had multiple features of the Chiari II malformation. In the posterior fossa, both patients had a distorted, low fourth ventricle with vermian peg and cervicomедullary kink. The tectal plate of the midbrain was beaked and the posterior fossa was small. Both patients developed hydrocephalus. An enlarged massa intermedia was seen in one patient. Both patients had syringomyelia and one also had a low conus medullaris located at the L3 body. Neither patient has become symptomatic from the Chiari malformation.

The unified theory of embryogenesis of the Chiari II malformation was proposed by McLone in 1989, and suggested that loss of distention of the rhombencephalic vessels following CSF decompression through the open neural tube defect resulted in abnormal induction effects on the surrounding posterior fossa mesenchyme. Subsequent development of the hindbrain within the small posterior fossa leads to both upwards and downwards hemiation and secondary hydrocephalus.

The cases presented provide further support for this mechanism, and also specifically preclude the possibility of the traction theory as the cause of the malformation, in that neither child had a spinal neural tube defect. The hydrocephalus/hydrodynamic theory is also not supported in view of the supratentorial malformations and upwards hemiation. The occurrence of the malformation in the setting of encephalocele also supports the notion that this is not a primary dysgenetic lesion of the posterior fossa, but rather that it is a epiphenomenon to the loss of CSF via either an open neural tube defect or a large, fluid-filled encephalocele. The literature is also reviewed.
6 Induction of Neuronal Nitric Oxide Synthase During the Late Stage of Congenital Hydrocephalus in the HTX Rat

Stephen L. Kinsman, MD, Donald Eslin, BA (Baltimore, MD), Hazel C. Jones, PhD (Gainesville, FL), Mary S. Lange, MA (Baltimore, MD), James P McAllister II, PhD (Cleveland, OH)

The long-term effects of congenital hydrocephalus include: damage to periventricular white matter, damage to cortical dendritic spines and synapses, and some neuronal loss. The mechanism of injury for this cellular damage is unknown. Nitric oxide synthase (NOS) can be induced with brain injury creating neurotoxic levels of nitric oxide. We hypothesized that an induction of neuronal NOS (nNOS) occurs with hydrocephalus. The hydrocephalic HTX rat provides a model to study brain injury in hydrocephalus. These rats develop, because of aqueductal stenosis, progressive hydrocephalus perinatally. By postnatal day 21 (P21), these rats exhibit moderate to severe hydrocephalus, usually with symptoms of learning problems and motor impairments. We investigated whether nNOS expression was altered by comparing P21 hydrocephalic HTX rats with their control littermates using immunocytochemistry for nNOS. Examination of coronal sections confirmed the presence of hydrocephalus. nNOS immunoreactivity (IR) showed increased fiber staining in hydrocephalic brains without evidence of neuronal loss, particularly in the basal ganglia. Using dark-field densitometric image analysis of nNOS IR axons we found 33.92 axon pixels/area (n=5) for hydrocephalic animals vs. 6.52 axon pixels/area (n=4) for controls, an over 5-fold increase (p<.001). Analysis of a section of cortex separated into upper and lower halves showed a 3.5-fold increase for the lower half of cortex, 5.06 axon pixels/area (n=4) vs. 1.45 (n=3) (p<.005). These results suggest that nNOS may be involved in the mechanism of injury produced by hydrocephalus.

(Supported in part by: 5T32 HD 07414-06.)
Extended Midline Incision for Nasal Dermoid Cyst Excision: Nasal Tip to Intracranial Extradural Extension

Michael W. Born, MD, Joel W. Winer, MD (York, PA)

Objective: To remove midline nasal dermoid cysts from nasal tip to skull base without the need of a bicoronal exposure.

Methods: C-arm fluoroscopy, microscope, and CT scan planning will be described for two cases with pre-, intra-, and postoperative radiographs and photographs.

Results: Reduced operative time, reduced hospital stay, no evidence of disease with follow-up greater than twelve months, and acceptable cosmetic result.

Conclusions: Using an extended midline incision, nasal dermoid cysts can be safely and completely excised, en bloc, from nasal tip to intracranial extradural extension. Cranial base and basal bony defect reconstruction techniques using Grafton will also be discussed.
Cavernous Sinus Dermoid Cysts

Paul M. Kanev, MD (Detroit, MI)

Dermoid cysts are rare intracranial lesions in children. Most are located within the anterior or middle cranial fossa, and patients typically present with headaches or seizures. There are two reported cases of intracavernous dermoid cysts; a 4-year-old male with isolated oculomotor nerve palsy and a 27-year-old woman with headaches. We report our experience with four children with intracavernous dermoids. Each child, 27 months to 18 years of age, presented with ptosis, pupil dilation, and ophthalmoplegia. CT and MRI revealed a smooth ovoid mass within the cavernous sinus.

CSF examination in three patients revealed fat droplets and inflammatory pleocytosis. Preoperative decadron improved ptosis but had no effect upon ophthalmoplegia. We utilized the Dolenc procedure for extradural removal of the orbital roof, anterior clinoid process and the bone overlying the superior orbital fissure. After dural opening, the petroclinoid ligament was coagulated and divided medial to the path of the third nerve, entering the plane between the dura propria and membranous wall of the cavernous sinus. The dermoid tumors were adherent to the third nerve within Dolenc’s anterior medial or lateral triangle. Cyst contents were evacuated and capsules were completely dissected from the third nerve. Ptosis, pupillary reactivity and ophthalmoplegia resolved postoperatively. Intracavernous dermoids must be considered in the differential diagnosis of ptosis and ophthalmoplegia in children. Using the Dolenc extradural approach to the cavernous sinus, these cysts may be completely resected with improved cranial nerve function.
9 The Role of Radical Surgery for Intracranial Melanotic Neuroectodermal Tumor of Infancy* 

Shervin Rahimidashti, BS, Mark L. Cohen, MD, Alan R. Cohen, MD (Cleveland, OH) 

Melanotic neuroectodermal tumor of infancy (MNTI) is a rare locally aggressive tumor that arises most commonly from the maxilla or mandible. Infrequently, it originates from the skull vault, and recent reports have described a favorable outcome following radical surgery. Some lesions are difficult to remove, particularly those located along the cranial midline or cranial base, or those with significant intracranial extension. Unfortunately, radiotherapy for MNTI is precluded by the patients' young age, and chemotherapy has been ineffective. Thus, the role for radical resective surgery assumes even more importance in these young patients for whom no other therapy exists. We report successful removal of an MNTI at the skull base that required excision of the involved meninges and major dural venous sinuses.

A two-month-old infant presented with a large firm exophytic retroauricular skull-based mass with intra- and extracranial extension. Craniectomy with gross total resection was carried out, and the pigmented tumor was peeled off the dura over the junction of the transverse and sigmoid sinuses. Within six weeks the tumor recurred and grew to the same size as preoperatively. Evaluation with CT, MRI and angiography showed involvement of the meninges, but patent dural venous sinuses. Repeat surgery was carried out with extensive craniectomy, and the dura was removed with wide margins including portions of the tentorium, patent transverse sinus and vein of Labbe. The patient made an excellent recovery in the short term, and is without neurologic deficit or radiographic evidence of tumor.

MNTI is a rare tumor that is encountered infrequently by the neurosurgeon. Because no effective adjuvant therapy exists, radical surgical resection is indicated, even when the tumor extends intracranially to involve structures that would not generally be sacrificed. Although MNTI is locally highly invasive, radical surgery may be associated with a favorable outcome and offers the potential for long-term cure.
Pituitary macroadenomas occur infrequently in children and can present with visual dysfunction or endocrinopathy. Occasionally, primary end-organ failure can cause reactive enlargement of the pituitary gland that may be clinically and radiographically indistinguishable from a pituitary macroadenoma. The authors describe an example of reactive pituitary hyperplasia from primary hypothyroidism that mimicked a pituitary macroadenoma in a child. Recognition of this entity, which responds to medical therapy, is important because it can spare patients unnecessary surgery.

A 13-year-old boy presented with 10 months of nausea and vomiting. Examination was remarkable only for dry skin. MRI demonstrated a large homogeneously enhancing sellar mass with suprasellar extension abutting the optic chiasm. The thyroid stimulating hormone (TSH) level was 827 mU/ml (normal 0.5-5.0 mU/ml) and the total thyroxine was less than 1.0 mg/dl (normal 5-12 mg/dl). A presumptive diagnosis of primary hypothyroidism was made on the basis of the markedly elevated TSH, and the patient was treated with thyroid replacement and received no other therapy. Symptoms resolved, laboratory values returned to normal, and repeat MRI showed complete resolution of the large enhancing pituitary mass.

Primary end-organ failure should be considered in the differential diagnosis of mass lesions of the pituitary gland, particularly in children in whom pituitary adenomas are distinctly uncommon. The clinical presentation of pituitary hyperplasia resulting from end-organ failure may be difficult to distinguish from primary pituitary neoplasms, especially if the symptoms of endocrine failure are subtle. Since pituitary hyperplasia can also mimic pituitary tumors radiographically, it is essential that children with abnormal sellar imaging undergo careful endocrinologic evaluation to exclude primary end-organ failure. Some suprasellar masses may be the result, rather than the cause, of endocrinopathy. Subtle MRI findings suggest the diagnosis of pituitary hyperplasia rather than neoplasia, and these findings are discussed. Recognition that pituitary enlargement can occur in response to primary end-organ failure is important, and can prevent unnecessary surgical exploration in patients not harboring pituitary neoplasms.
Bipolar, Suprasellar and Pineal, Germ-Cell Tumors: Report of Three Cases and Review of the Literature

Matthieu Vinchon, Annie Laquerriere, PhD, Jean-Pierre Vannier, PhD, Jean-Marc Kuhn, PhD, Pierre Freger, PhD (Rouen Cedex, France)

In rare occurrences, intracranial germ-cell tumors may be present in the pineal and suprasellar regions simultaneously. We report on three cases of bipolar germ-cell tumors (BGCT) operated recently at our institution.

These three young male patients presented with one to six months' duration intracranial hypertension, diabetes insipidus, and in two cases, Parinaud's syndrome. In two patients with negative tumor markers, the tumor was a germinoma. The third had high alphafeto-protein levels and was initially given chemotherapy. The alphafeto-protein normalized and the suprasellar tumor disappeared, however, the pineal tumor grew larger, and necessitated emergency surgery: the diagnosis was immature teratoma. At last follow-up control, all three patients have complete remission with no or minor deficits.

We found 52 other documented cases of BGCTs in the literature, on average 13.9 f 5.6 years old, with a male/female ratio of 2.9. Thirty-nine cases were germinomas, with a favorable outcome, and 13 cases were other tumor types. In rare cases, two different tumor types were found in the same individual. The pathogenesis of BGCT supposes metastasis from one site into the other. The strong polarity of the primary tumor as well as the metastases for these locations suggests local factors favoring the development of germ-cell tumors.

Histologic diagnosis is mandatory. We favor a direct surgical approach of the pineal tumor to reduce the risk of sampling error. Since the prognosis of these tumors is favorable after radiotherapy, we think radical surgery of the two lesions is not indicated.
Revision of Ventricular Catheter Obstruction with Bugbie Wire: Retrospective Review at Two-Year Follow-up

Stanley H. Kim, MD, Christopher Madden, MD, Henry Bartkowski, MD, Edward Kosnik, MD, Pramit S. Malhotra (Columbus, OH)

Obstruction of a ventricular catheter is the most common of shunt failures. Removal of an obstructed ventricular catheter can be complicated by intraventricular hemorrhage (IVH), which has been reported to occur as high as 31%. The technique of inserting a bugbie wire into the obstructed ventricular catheter and coagulating the adherent tissue has been used to decrease the risk of IVH. To determine the failure rate of revision of obstructed ventricular catheters and the incidence of IVH seen intraoperatively using the bugbie wire technique, a retrospective study was conducted of all children who had undergone revision of an obstructed ventricular catheter at Childrens Hospital in Columbus in 1995 with two year follow-up. A total of 60 patients underwent 82 shunt procedures by the same surgeon in 1995. Of these 60 patients, 36 patients were intraoperatively diagnosed with obstructed ventricular catheter without preoperative evidence of IVH. Of these 36 patients, 32 (88.9%) patients had restoration of dynamic cerebrospinal fluid (CSF) flow through the ventricular catheter after the bugbie wire was used without placing the catheter. The remaining 4 (11.1%) patients had either the ventricular catheter replaced or tied off after the bugbie wire had failed to restore dynamic CSF flow. IVH was not observed intraoperatively in all patients who had ventricular catheter revision with the bugbie wire. At two-year follow-up, 5 of 32 (15.6%) patients who had revision of a ventricular catheter with the bugbie wire without replacement of the catheter developed a total of 7 cases of recurrent ventricular catheter obstruction. The time to next revision was 148.2 ± 79.2 days. In conclusion, revision of obstructed ventricular catheter with the bugbie wire may minimize the incidence of IVH and subsequent catheter obstruction.
Volumetric Study of the Ventricular System in a Canine Model of Obstructive Hydrocephalus

Peter Chovan, MD, Charles P. Steiner, MS, James P. McAllister II, PhD, Martha J. Johnson, PhD, Mark G. Luciano, MD, PhD, Igor Ayzman, BA, Arcangela S. Wood, BA, Jean A. Tkach, PhD, Joseph F. Hahn, MD (Cleveland, OH)

In the large canine model of acquired obstructive hydrocephalus that we have developed recently, computer-assisted 3-dimensional morphometry has been performed on T1-weighted Spin Echo MRI images from adult dogs before and after the induction of hydrocephalus. To date, 7 hydrocephalic animals have been analyzed that survived 7-83 days (median=54) after receiving injections of cyanoacrylate glue into the anterior fourth ventricle. Measurements were obtained from lateral, 3rd, and 4th ventricles. The volumes of the left and right lateral ventricles were symmetrical before and after induction. Mean lateral ventricle volume increased 424% from a baseline of 0.63 cc to a post-induction value of 3.30 cc (p<0.01 with unpaired t-test). In contrast, the 3rd ventricle expanded 187% from a mean of 0.15 cc to 0.43 cc (p<0.05). The combined volume of the lateral and 3rd ventricles increased 369% from a mean of 0.78 cc to 3.69 cc (p<0.01). Evans ratios, which are used routinely in the clinical setting, were also obtained from linear measurements of the lateral ventricle width divided by brain width at the level of the foramen of Monro. These values exhibited only a 94% increase from mean baseline ratios of 0.17 to post-induction ratios of 0.33 (p<0.05). These findings indicate that in mechanically-induced obstructive hydrocephalus the relative expansion of the lateral ventricles is greater than that of the 3rd ventricle. In addition, volumetric measurements of the lateral and 3rd ventricles suggest that the extent of ventriculomegaly is 3-4 times greater than estimated by Evans ratios.

(Supported by Johnson & Johnson Professional, Inc., The Cleveland Clinic Foundation, The Leede Hydrocephalus Research Fund, and NASA CAMIS.)
Children with Hydrocephalus and Congenital Heart Disease: What Is Their Outlook?

Todd Crawford, MD, William C. Olivero, MD, William C. Hanigan, MD
(Peoria, IL)

Introduction: Children presenting with the combination of congenital hydrocephalus and congenital heart disease are infrequent but often pose treatment dilemmas. Each condition has associated co-morbidities and risks of therapy. We analyzed cases of combined hydrocephalus and heart disease in the pediatric population to determine the mortality, morbidity and prognosis.

Methods/Results: A retrospective chart review was performed at the Children's Hospital of Illinois at Saint Francis Medical Center in Peoria, Illinois, a tertiary referral center for pediatric cardiology and neurosurgery. A total of 11 children (5 male and 6 female) were identified and treated between 1987 and the present. Four presented at birth and the remaining 7 by 5 months of age. The etiology of hydrocephalus was Dandy Walker malformation (4), aqueductal stenosis (5), and idiopathic (2). The nature of the heart disease was AV canal (3), simple septal defects (3), complex septal defects (3), corrected transposition (1) and coarctation of the aorta (1). Follow-up of 6 months to 10 years revealed that 4 have died, 6 are developmentally delayed and one is developmentally normal.

Discussion: The incidence of congenital heart disease in children with hydrocephalus is not well documented but may range from 6-10%. Few studies to date have addressed the outcomes of these children. Over a 10-year period we have identified 11 such children with a mortality rate of 36% (4/11), a psychomotor retardation rate of 54% (6/11), and a normal outcome of only 9% (1/11). The combination of these diseases in children appears to portend a poor prognosis and should be discussed with the family before aggressive treatment is undertaken.
15 Atypical Presentations of Low-Pressure Shunt Malfunction

Lori A. McBride, MD, Michael D. Partington, MD, K.R. Winston, MD
(Denver, CO)

Intracranial hypotension due to cerebrospinal fluid (CSF) overdrainage has classically been associated with postural headache and low CSF pressure on lumbar puncture. This can occur spontaneously, after trauma, or after the insertion of a low-pressure or malfunctioning CSF shunt. We report a series of four patients with five episodes of unusual symptoms of intracranial hypotension due to shunt malfunction, including episodic unresponsiveness, vomiting, lethargy and memory deficits. Intracranial hypotension was documented in each patient by preoperative shunt tap or intraoperative shunt pressure measurement. In each patient, symptoms resolved after valve replacement. These findings are supported by the fact that atypical presentations of low CSF pressure have previously been reported following spontaneous CSF leaks or those due to dural tears after lumbar puncture. This report describes a new clinical entity: the first association of low-pressure shunt malfunction with unusual presentations of intracranial hypotension. We suspect that patients with CSF shunts who complain of these atypical symptoms are currently underdiagnosed.
Unusual Presentation of the Complete Currarino Triad with Unique Teratoma Histopathology*

Christopher A. Gegg, MD, Micam W. Tullous, MD, Kathleen Kagan-Hallet, MD (San Antonio, TX)

Objective and Importance: We present an unusual case of the complete familial Currarino Triad (an association between sacral agenesis, an anterior sacral mass, and anorectal malformations) in which the teratoma arose from the conus and contained mature neurons, ependymal cells, and glia. Presence of the complete triad is rare, and the histopathology in this patient is unique.

Clinical Presentation: The patient was a term neonate when discovered to have imperforate anus. Further workup revealed lumbosacral dysraphism with a presacral mass, a rectovaginal fistula, and a single pelvic kidney. Family pedigree revealed a familial transmission pattern: the patient had a second cousin with anal atresia and a first cousin with similar sacral anomalies. The motor level was L4 with trace L5 and there was absent sensation in the sacral dermatomes.

Intervention: A diverting colostomy was performed on day 14, and the infant returned at three months for near total resection through the previous abdominal approach. Only a subtotal resection was possible because the mass arose from the conus and was firmly adherent to the sacral nerve roots and iliac vessels. Postoperative complications—cerebrospinal fluid leak and renal failure—will be discussed. The residual tumor has not progressed on follow-up MRI at 18 months.

Conclusions: The complete Currarino Triad is rare and is familial in half the cases. In familial cases, the teratoma is usually benign and contains all three germ layers. Special features of the tumor in our case were the presence of mature neurons, ependymal canals, glia, and its origin from the conus. These are unique findings, and this is the first such case reported.
Post-Operative Use of Chalasia Wedge After Craniotomy and Morcellation of Skull for Craniosynostosis: A Technical Note

Stanley H. Kim, MD, Christopher Madden, MD, Henry Bartkowski, MD, Edward Kosnik, MD, Pramit S. Malhotra, MD (Columbus, OH)

We report an alternative means of positioning the child postoperatively when an extensive craniotomy and morcellation of skull is performed for various craniosynostosis in children under the age of 6 months. Chalasia wedge (Pedicraft) was originally used in infants as part of the treatment for gastroesophageal reflux. The wedge is triangular shaped with the dimensions of 20 inch in length by 18 inch in height by 29 inch in width with a slope of 30 degrees. It is made of anti-bacterial foam covered with vinyl and cotton quilt. The child is placed on the slope side of the wedge of the abdomen in head-up position and secured with velcro fasteners. A child that has begun to crawl may be too active to be restrained in the wedge. A potential advantage of using the wedge after an extensive morcellation procedure is to allow cranial expansion and remodeling of the skull to take place under gravity as the head of the child is supported in near up-right position. A total of eight patients with various craniosynostosis underwent morcellation of skull and were placed in the wedge for six weeks postoperatively. Optimal remodeling of the skull was observed at six-week follow-up. Further follow-up study with radiological imaging may support the cosmetic effect of using the wedge after an extensive morcellation procedure.
Spasticity and Strength at the Ankle in Children with Cerebral Palsy

Jack R. Engsberg, PhD, Kenneth S. Olree, MS, Sandy A. Ross, MHS, T. S. Park, MD (St. Louis, MO)

Spasticity and weakness are problems associated with children with cerebral palsy (CP). Recently, we quantified spasticity and strength in the hamstrings of these children. It has been reported that distal areas of the lower limb are often more affected than proximal areas. This investigation quantified spasticity and strength in the ankle plantarflexors in children with spastic diplegia CP (CP group). Both legs of 27 children with CP were tested on a KinCom dynamometer. For the spasticity measure, the machine rotated the passive ankle from maximum plantarflexion (PF) to maximum dorsiflexion (DF) at speeds of 10, 30, 60, 90, and 120°/s while recording the resistive PF torque. Work values for the torque-angle data were calculated. Linear regression was used to derive the slope of the line of best fit for the work-velocity data (i.e., the spasticity measure). For strength, the machine rotated the ankle from maximum DF to maximum PF at a speed of 10°/s while the child performed a maximum concentric contraction. Peak PF torque and work by the plantarflexors were determined. Unpaired t-tests were used to test for significant differences (p<0.05) between the CP group and an able-bodied control group (AB group, n=9). Spasticity in the plantarflexors for the CP group (x=0.019 J/(°/s), SD=0.016) was significantly greater than the AB group (x=0.008 J/(°/s), SD=0.006). Peak PF torque (CP x=0.57 Nm/kg, SD=0.35; AB x=1.88 Nm/kg, SD=0.63) and maximum PF work (CP x=0.25 J/kg, SD=0.25; AB x=1.28 J/kg, SD=0.50) were significantly less than the AB group. Our previously reported result for spasticity at the hamstrings (0.024 J/(°/s), SD=0.024) was not significantly different from the spasticity at the plantarflexors. This finding is not supported by the subjective reports in the literature indicating that spasticity is greatest at the distal joints. Our strength measures for peak knee flexion torque and maximum knee flexion work for a similar group of children were 42% and 19% of able-bodied norms, respectively. At the ankle, similar values were 30% and 20%, respectively. Thus, while the work values were similar between the knee and ankle, the peak values were less at the ankle. These peak values support the notion of greater involvement in the distal lower extremity joints.
Traumatic Brain Injury in Children Versus Adults: Differences & Outcomes

Omar F. Jimenez, MD, David Petrin, RN, Doreen Berguestein, RN, John Ragheb, MD (Miami, FL)

Introduction: The impression that children with severe traumatic brain injury have better outcomes than adults is not well supported by the literature. Our purpose is to directly compare outcomes for severe (GCS 8 or less), non-penetrating traumatic brain injury in children and adults at a single institution.

Methods: We retrospectively reviewed our institutional data trauma bank from 1993-1997 to assess survival and outcome for children (under 15 yrs) at time of discharge with traumatic brain injury of GCS 8 or less and compared them to young adults (16-19 yrs) and adults (20-94 yrs).

Results: There were 87 patients under the age of 15 yrs (31F; 46M), 51 patients from 16-19 years (8F; 43M), and 446 patients from 20-94 years (86F; 360M). The most common mechanism of injury under the age of 15 was pedestrian struck by automobile vs MVA for patients in the other age groups. The most common injury for children were skull fractures and contusions vs SDH and contusions in the other groups. Only 6% of patients under the age of 15 yrs required surgical intervention vs 27% for young adults and 32% for adults. For children, the mortality was 23% (92% mortality (11/12 patients) for children 1-2 years with GCS 3-5), 27% went home and 22% went to rehabilitation. For young adults, 11% died, 33% went home and 42% went to rehabilitation. For adults, 33% died, 15% went home and 42% went to rehabilitation. The average length of stay for children was 17 days vs 28 days for the other groups.

Conclusion: Patients under the age of 15 were less likely to have a surgical traumatic brain injury. The highest mortality for traumatic brain injury was for children 1-2 years of age with GCS 3-5. Children overall had a greater proportion discharged home and a shorter hospital stay. We propose that in the management of traumatic brain injury the outcome of young adults is similar to adults. Children, however, have a better prognosis.
Trends in Firearms Related Deaths in Children: A Public Health Crisis for Pediatric Neurosurgery

John Ragheb, MD, Andrew Jea, Dorene Beguiristain, RN, Mimi Sutherland, RN (Miami, FL)

During the 10-year period from 1985 through 1994 data from the U.S. Bureau of Alcohol, Tobacco and Firearms reports a 55% increase in the number of new firearms manufactured and imported into the United States on a per capita basis. During the last 6 years of this same period the Center for Disease Control reports a near parallel increase in the per capita firearms related death rate of 42% in teenagers age 15 to 19 years and a 9% increase for those age 10 to 14. These statistics greatly underestimate the magnitude of this problem as there are no national statistics for the number of non-lethal firearms injuries in children. This alarming epidemic in firearms related deaths raises an important public health issue particularly for those who care for the pediatric patients. Although, there are many potential explanations for the rise in firearms related deaths, the increasing presence of firearms in the home most certainly plays a role. Organized pediatric neurosurgery must therefore take the initiative in educating families with children of the dangers of firearms in the home and work to change how children view firearms. Programs from our institution, and resources available nationwide for family education, teenage conflict resolution, and to diminish the allure of firearms for young children will be presented.
Stereolithographic Models of Complex Tumors

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

Certain tumors, especially of the skull base, require the combined efforts of two or more teams of surgical specialists. We have been able to facilitate the coordinated planning of the teams by the use of stereolithographic models, three-dimensional reconstructions in one or two colors, made from high-resolution CT or MRI scans according to specified protocols. Thus far, models have been prepared for five patients, four of whom we operated upon. Models depicted: 1) a malignant fibrous histiocytoma of the right temporal fossa, cavernous sinus, maxillary sinus and infratemporal fossa, 2) a juvenile angiofibroma involving most of the nasopharynx, the left orbit, left maxillary sinus, left middle fossa, and the frontal fossae bilaterally, 3) a recurrent glossopharyngeal schwannoma, 4) a large clivus chordoma, and 5) an osteosarcoma involving vertebral bodies T6-9 and left ribs 7 and 8. We found the models to allow "hands-on" comparison of alternative approaches and sequences for the operations, in a manner that cannot be achieved by manipulation of the images at a conventional computer workstation. The models materially affected the extent of some operations, and helped to optimize the integration of the teams' efforts, minimizing intraoperative time and improving operative efficiency.
Benign Migratory Cerebellar Endotheliosis

John R. Mawle, MD, Patricia T. Franklin, MSN, James Schimschock, MD
(Portland, OR)

A seven-year-old male presented with the acute onset of headache, nausea and vomiting of two days duration. He was drowsy and bradycardic. CT scanning demonstrated a low dense area in the right cerebellar hemisphere compatible with edema or infarction and producing obstructive hydrocephalus. He was managed with mannitol and steroids and experienced dramatic overnight improvement. Follow-up MR scanning and MR arteriography and venography demonstrated neither arterial nor venous occlusive disease; residual areas of high signal in both cerebellar hemispheres were interpreted as gliosis. A workup for hypercoagulable I.

The child was well for eighteen months, until he again presented listless and vomiting. On this occasion he had similar radiographic findings, but in a previously uninvolved area of the left cerebellum. Angiography demonstrated a distinct blush in the left tonsil. He was treated with steroids alone, and symptoms promptly resolved.

A biopsy of very vascular cerebellar tissue showed proliferation of the microvasculature and endothelial hyperplasia.

The relationship of this condition to hemangioma, telangiectasia and vascular pathology has not been previously described, and we propose the term “benign migratory cerebellar endotheliosis.”
23 Volumetric Reduction of a Choroid Plexus Carcinoma Using Preoperative Chemotherapy

Mark M. Souweidane, MD, James H. Johnson, Jr., MD, Eric Lis, MD
(New York, NY)

Objective and Importance: The best treatment of choroid plexus carcinoma in children remains controversial. However, a gross total resection is the best prognostic indicator. Achieving a complete resection can be challenging given the vascularity of these tumors. Thus, any modality that maximizes the potential for a complete resection while decreasing the surgical risks would be beneficial. Chemotherapy has been reported to decrease the estimates of intraoperative blood loss. We report for the first time an objective response of tumor volume using preoperative chemotherapy.

Clinical Presentation: A thirteen-month-old female child who presented with gait difficulty was found to have hydrocephalus and an intraventricular tumor. She underwent shunting and an open biopsy, the pathology being consistent with a choroid plexus carcinoma.

Intervention: The patient underwent three courses of intravenous chemotherapy consisting of etoposide (4 mg/Kg), cyclophosphamide (65 mg/Kg), vincristine (0.05 mg/Kg/dose), and cisplatin (3.5 mg/Kg). Comparisons were made between an MRI at diagnosis and an MRI ten days after completing chemotherapy. Tumor volume diminished from 95.13 cm³ to 67.02 cm³, a reduction of 29.5%. Subsequently, a staged surgical procedure resulted in a gross total excision of her tumor. A complete resection was confirmed with postoperative MRI and the child remains disease free at 15 months of follow-up.

Conclusion: The contention that preoperative chemotherapy may assist in tumor reduction prior to surgery is supported by our volumetric analysis. The role of chemotherapy in controlling disease remains open for debate, but should be strongly considered as a preoperative adjunct in the treatment of choroid plexus carcinoma.
Diffuse Subarachnoid Spread of Juvenile Pilocytic Astrocytoma (JPA)

Daniel M. Lieberman, MD, Susan M. Chang, MD, Kenneth D. Aldape, MD, Daniel B. Vigneron, PhD, Charles B. Wilson, MD (San Francisco, CA)

Often JPAs spread to the local subarachnoid space from an intraparenchymal nidus, and it appears that some become multicentric through metastasis via cerebrospinal fluid. By contrast, some oligodendrogliomas actually spread diffusely through the subarachnoid space and so extensively that they have been termed secondary leptomeningeal gliomatosis. We recently treated an 18-year-old boy who had diffuse leptomeningeal spread of a variant JPA. He presented with acute hydrocephalus and intracerebral hemorrhage centered in the left caudate nucleus, without evidence of tumor. He did well after placement of a ventriculoperitoneal shunt for 3 years, then again presented with hydrocephalus. Repeat MR imaging revealed a heterogeneously enhancing, contiguous mass in the left lateral, third, and fourth ventricles, with circumferential extension throughout the spinal cord. MR spectroscopy showed an abnormal metabolite pattern, suggestive of tumor, with zones of necrosis. The intraventricular component of the tumor was biopsied through a frontal craniotomy. The tumor was composed of loose and compact areas of astrocytes with fibrillary morphology and Rosenthal fibers, typical of JPA. Many of the neoplastic cells expressed glial fibrillary acidic protein. In addition, there were patches with patterns of oligodendroglial differentiation and prominent nuclear atypia, as has been reported in JPA. The patient was partially treated with craniospinal axis irradiation, but he withdrew from treatment and died after 6 months. To our knowledge, this is the first reported example of diffuse subarachnoid spread by JPA. In addition, this case illustrates the utility of MR spectroscopy to detect the spread of JPA.
25 Posterior Fossa Craniotomy: An Effective Alternative to Craniectomy*  

Shekar N. Kurpad, MD, PhD, Alan R. Cohen, MD (Cleveland, OH)  

We describe a simple midline approach to posterior fossa lesions in children that involves removal and replacement of a bone flap. This approach offers several advantages over conventional suboccipital craniectomy.

After wide bilateral exposure of the occipital bones from the foramen magnum to the superior nuchal line, two burr holes are placed far laterally such that they are situated just inferior to the transverse sinus on each side. Additional paramedian burr holes may be placed but are usually not necessary. The dura is carefully stripped and the bone flap is elevated using a high speed drill and footplate. The lateral cuts are made first, with care to avoid injuring the annular sinus at the foramen magnum. The superior cut crossing midline is made last, using a dissector to gently depress the dura and avoid injury to the occipital sinus and torcular Herophili. When required, the craniotomy is extended with rongeurs. At the end of the procedure, the bone flap is resecured using titanium plates and screws. Any resulting gap is easily bridged by the plates.

During the past 3 years we performed midline posterior fossa craniotomies on 23 patients undergoing exploration for a variety of lesions including tumors, cysts and one traumatic epidural hematoma. There were no complications with this procedure. Specifically, there were no instances of infection, hemorrhage, or postoperative brain swelling necessitating reoperation. One patient underwent reoperation for recurrent astrocytoma two years after the initial procedure, and presence of the bone flap considerably simplified the exposure. Furthermore, there was complete fusion at the superior margins of the bone flap, suggesting that replaced flaps may heal well in this location.

Elevation of the bone flap is facilitated by the relatively small size of the midline bony keel in children. Posterior fossa craniotomy is simple and safe, and offers advantages over conventional craniectomy techniques, including preservation of anatomical planes and increased protection of the posterior fossa contents.
THE AMERICAN ASSOCIATION OF NEUROLOGICAL SURGEONS

AND

CONGRESS OF NEUROLOGICAL SURGEONS

JOINT SECTION ON

PEDIATRIC NEUROLOGICAL SURGERY

MEMBERSHIP ROSTER
Abbasy, Munir H., MD  
80 Congress St.  
Springfield, MA 01104-3427  
Active Member

Arkins, Thomas J., MD  
Neurological Associates  
60 Temple St.  
New Haven, CT 06510-2716  
Active Member

Benzel, Edward C., MD  
Univ. of New Mexico  
Div. of Neurosurgery  
Albuquerque, NM 87131  
Active Member

Abbott, Ira R., III MD  
Beth Israel Med. Ctr.  
170 E. End Ave./Neuro.  
New York, NY 10128  
Active Member

Aronin, Patricia A., MD  
521 Dubuar St.  
Northville, MI 48167-1419  
Active Member

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

Adelson, P. David, MD  
Children's Hosp. of Pittsburgh  
3705 Fifth Ave./Neurosurgery  
Pittsburgh, PA 15213-2524  
Active Member

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

Berger, Thomas S., MD  
Mayfield Neurological Clinic  
506 Oak St.  
Cincinnati, OH 45219-2507  
Active Member

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

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

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

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

Balis, Gene A., MD  
3000 E. Fletcher Ave.  
Suite #340  
Tampa, FL 33613-4845  
Active Member

Black, Peter McL., MD PhD  
Children's Hosp./Neuro.  
300 Longwood Ave.  
Boston, MA 02115  
Active Member

Amacher, A. Loren, MD  
Gelsemer Clinic  
Dept. of Neurosurgery  
Danville, PA 17822-0001  
Active Member

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

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

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

Bartkowski, Henry M., MD PhD  
Neurological Assoc.  
931 Chatham Ln.  
Columbus, OH 43221-2486  
Active Member

Boop, Frederick A., MD  
Arkansas Children's Hosp.  
800 Marshall St./Neurosurgery  
Little Rock, AR 72202-3510  
Active Member

Anderson, Jim D., MD  
Redwood City Offices  
1150 Veterans Blvd.  
Redwood City, CA 94063-2037  
Active Member

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

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

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

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

Bressler, Bruce C., MD  
720 S. Van Buren  
Green Bay, WI 54301-3588  
Active Member
Brockmeyer, Douglas L., MD
Primary Children's Med. Ctr.
100 N. Medical Dr.
Salt Lake City, UT 84113-1100
Active Member

Chapman, Paul H., MD
Massachusetts Gen. Hosp.
Fruit St., GR8502
Boston, MA 02114
Active Member

Dagi, T. Forcht, MD
993 F. Johnson Ferry Rd.
Suite #100
Atlanta, GA 30342-1620
Active Member

Brown, Jeffrey Alan, MD
Med. Coll. of Ohio
Dept. of Neurosurgery
Toledo, OH 43699-0008
Active Member

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

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

Bruce, Derek A., MD
7777 Forest Ln.
Suite B308
Dallas, TX 75230-2505
Active Member

Choux, Maurice, MD
Hôpital Des Enfants
De La Timone, Cedex 5
Marseille, 13385
France
International Member

Di Rocco, Concezio, MD
Univ. Cattolica/Neurochirurgia
Largo Gemelli 8
Rome, 00168
Italy
International Member

Buchheit, William A., MD
1015 Chestnut St.
Suite #1400
Philadelphia, PA 19107
Active Member

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

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

Cahan, Leslie D., MD
1505 N. Edgemont St.
Neurosurgery/Room #4141
Los Angeles, CA 90027-5209
Active Member

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

Dorsen, Michael, MD
The Neurosur. Group of Austin
2905 San Gabriel, #310
Austin, TX 78705-3541
Active Member

Canady, Alexa Irene, MD
Children's Hosp. of Michigan
3901 Beaumien
Detroit, MI 48201-2119
Active Member

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

Doty, James R., MD
11190 Warner Ave.
Suite #404
Fountain Valley, CA 92708-4018
Active Member

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

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

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

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

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

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

Chaparro, Michael J., MD
5070 Rolling Woods Tr.
Kettering, OH 45429-1109
Active Member

Crone, Kerry R., MD
3333 Burnet Ave.
Cincinnati, OH 45229-3039
Active Member

Duncan, Charles Cecil, MD
333 Cedar St., TMP 416
P.O. Box 206082
New Haven, CT 06520-8082
Active Member
<table>
<thead>
<tr>
<th>Name</th>
<th>Address</th>
<th>City, State, Zip</th>
<th>Phone</th>
<th>Active Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duncan, John A., MD</td>
<td>2 Dudley St., Suite #530, Providence, RI</td>
<td>02905-3236</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Fischer, Edwin G., MD</td>
<td>110 Francis St., Suite #3B, Boston, MA</td>
<td>02215-5501</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Gennuso, Rosemario, MD</td>
<td>727 Snyder Ave., Aromas, CA</td>
<td>95004</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Dunn, Mary E., MD</td>
<td>280 N. Smith Ave., #234, St. Paul, MN</td>
<td>55102-2420</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Flannery, Ann Marie, MD</td>
<td>1120 15th St./Neurosurgery, Augusta, GA</td>
<td>30912-4010</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>George, Richard E., Jr. MD</td>
<td>3506 21st St., #400, Lubbock, TX</td>
<td>79410-1200</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Edwarde, Michael S. B., MD</td>
<td>2100 Webster St., San Francisco, CA</td>
<td>94115-2373</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Foltz, Eldon L., MD</td>
<td>UCI Med. Ctr./Neurosurgery, P.O. Box 14091, Orange, CA</td>
<td>92613-4091</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Godersky, John C., MD</td>
<td>2841 De Barr Rd., Anchorage, AK</td>
<td>99508-2956</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Eisenberg, Howard M., MD</td>
<td>Div. of Neurosurgery, 22 S. Greene St., #S10B11, Baltimore, MD</td>
<td>21201-1734</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>French, Kathleen B., MD</td>
<td>3020 Hamaker Ct., Suite B104, Fairfax, VA</td>
<td>22031-2220</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Godrich, James T., MD PhD</td>
<td>Dept. of Neuro./Montefiore, 111 E. 210th St., Bronx, NY</td>
<td>10467-2401</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Ellenbogen, Richard G., MD</td>
<td>Walter Reed Army Med. Neuro. Svc, Georgia Ave., Bldg. II, Rm. 5C25, Washington, DC</td>
<td>20307-5001</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Fried, Arno H., MD</td>
<td>New York Med. Coll./Neuro. Menger Pavilion, Valhalla, NY</td>
<td>10595</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Gourmeneva, Liliana C., MD FRCS</td>
<td>Children’s Hosp., Bader 3, 300 Longwood Ave., Boston, MA</td>
<td>02115-5724</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Epstein, Fred J., MD</td>
<td>Beth Israel Med. Ctr., 170 E. End Ave., New York, NY</td>
<td>10128-7603</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Gahm, Norman H., MD</td>
<td>100 Retreat Ave., Suite #705, Hartford, CT</td>
<td>06106-2528</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Greene, Clarence S., Jr. MD</td>
<td>2650 Elm Ave., #218, Long Beach, CA</td>
<td>90802</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Erasmus, Mark D., MD</td>
<td>522 Lomas Blvd., N.E., Albuquerque, NM</td>
<td>87102-2454</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Galicich, Joseph H., MD</td>
<td>P.O. Box 276, Alpine, NJ</td>
<td>07620-0276</td>
<td></td>
<td>Lifetime Member</td>
</tr>
<tr>
<td>Guido, Laurance J., MD</td>
<td>174 Hubbell Ln., Fairfield, CT</td>
<td>06430-2148</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Faillace, Walter J., MD</td>
<td>Univ. of Florida, Jacksonville, FL</td>
<td>32209</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Gamache, Francis W., Jr. MD</td>
<td>Neuroscience Inst., 523 E. 72nd St., 7th Fl., New York, NY</td>
<td>10021-4099</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Gutierrez, Francisco A., MD</td>
<td>707 N. Fairbanks Ct, Chicago, IL</td>
<td>60611-4807</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Fell, David A., MD</td>
<td>Neurological Surg., Inc., 6767 S. Yale, Tulsa, OK</td>
<td>74136-3303</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Gaskill, Sarah J., MD</td>
<td>4499 Medical Dr., Suite #397, San Antonio, TX</td>
<td>78229-3735</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Haase, Jens, MD</td>
<td>Aalborg Hosp./Neurosurgery, Aalborg, Denmark</td>
<td>9100</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Name</td>
<td>Hospital/Address</td>
<td>City, State, Zip</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------</td>
<td>--------------------------------------------</td>
<td>------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hahn, Joseph F., MD</td>
<td>Cleveland Clinic</td>
<td>Cleveland, OH 44195-1004</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hahn, Yoon Sun, MD</td>
<td>Hope Children's Hosp. 4440 W. 95th St./Ped. Neuros. Oak Lawn, IL 60453-2600</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Haines, Stephen J., MD</td>
<td>MUSC/Dept. of Neurosurgery 171 Ashley Ave. Charleston, SC 29425-2272</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hakanson, Sten E., MD</td>
<td>Karolinska Sjukhuset Dept. of Neurosurgery Stockholm, S-10401 Sweden</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hamilton, Mark G., MD</td>
<td>Alberta Children's Hosp. 1820 Richmond Rd., S.W. Calgary, AB T2T 5G7 CANADA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hammargren, Lonnie L., MD</td>
<td>3160 S. Maryland Pkwy. Las Vegas, NV 89109-2305</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hammock, Mary Kathryn, MD</td>
<td>8620 Rolling Rd. Manassas, VA 20110-3828</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Handler, Michael H., MD</td>
<td>1010 E. 19th St. Suite #605, Tammen Hall Denver, CO 80218-1808</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Henlgan, William C., MD Phd</td>
<td>214 N.E. Glen Oak Ave. Suite #500 Peoria, IL 61603</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heathnor, Michael D., MD</td>
<td>1010 Edgemoor Rd., N. Charlotte, NC 28207-1885 Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heffez, Dan S., MD</td>
<td>2515 N. Clark St. Suite #800 Chicago, IL 60614-2720 Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Helbusch, Leslie C., MD</td>
<td>111 N. 84th St. Omaha, NE 68114-4101 Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemmy, David C., MD</td>
<td>20811 Watertown Rd. Suite J Waukesha, WI 53186-1871 Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hendee, Robert W., Jr. MD</td>
<td>4710 Lookout Mtn. Cove Austin, TX 78731-3654 Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hendrick, E. Bruce, MD</td>
<td>63 Leggett Ave. Etobicoke, ON M9P 1X3 CANADA Lifetime Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hoffman, Harold J., MD</td>
<td>Hosp. for Sick Children 555 University Ave., #1504 Toronto, ON M5S 1X8 CANADA Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Holbrook, Thomas J., Jr. MD</td>
<td>3 Medical Park Rd. Suite #310 Columbia, SC 29203-6873 Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hollenberg, Robert D., MD</td>
<td>McMaster Univ. Med. Ctr. Dept. of Surgery/Room #4U4 Hamilton, ON L8N 3Z5 CANADA Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hudgins, Roger, MD</td>
<td>5455 Maridan Mark Rd. Suite #540 Atlanta, GA 30342 Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Humphreys, Robin P., MD</td>
<td>Hosp. for Sick Children 555 University Ave., #1504 Toronto, ON M5S 1X8 CANADA Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>James, Hector E., MD</td>
<td>7930 Frost St. Suite #304 San Diego, CA 92123-2737 Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jane, John A., MD PhD</td>
<td>Univ. of Virginia Neuro/Hosp. Box 212 Charlottesville, VA 22908 Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jimenez, David F., MD</td>
<td>Univ. of Missouri Hosp.&amp;Clinic One Hospital Dr., N521 Columbia, MO 65212-0001 Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Johnson, Dennis L., MD</td>
<td>Milton Hershey Med. Ctr. P.O. Box 850/Div. of Neuro. Hershey, PA 17033-0880 Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Johnson, John K., MD</td>
<td>Greenville Neuro. Group 27 Memorial Medical Dr. Greenville, SC 29605-4407 Active Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Johnson, Mary M., MD</td>
<td>3223 Chatham Rd. Atlanta, GA 30305-1101 Lifetime Member</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Name</td>
<td>Address</td>
<td>City, State Zip</td>
<td>Position</td>
<td></td>
</tr>
<tr>
<td>---------------------</td>
<td>--------------------------------</td>
<td>-----------------</td>
<td>-------------------</td>
<td></td>
</tr>
<tr>
<td>Jones, Robert F. C., MD</td>
<td>21 Northington St.</td>
<td>Paddington, NSW 2021</td>
<td>Australia International Member</td>
<td></td>
</tr>
<tr>
<td>Klein, David M., MD</td>
<td>690 Farrelling Post</td>
<td>Pittsboro, NC 27312-8507</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Loefman, Morris D., MD</td>
<td>16311 Ventura Blvd.</td>
<td>Encino, CA 91436-2152</td>
<td>Lifetime Member</td>
<td></td>
</tr>
<tr>
<td>Joseph, Allen S., MD</td>
<td>7777 Hennessy Blvd.</td>
<td>Baton Rouge, LA 70808-4300</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Kleiner, Laurence I., MD</td>
<td>5304 Indian Grave Rd.</td>
<td>Roanoke, VA 24014-6608</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Longo-Cordero, Rafael, MD</td>
<td>Calle Rochester 911</td>
<td>San Juan, PR 00927-4812</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Kalsbeek, John E., MD</td>
<td>Riley Children's Hosp.</td>
<td>Indianapolis, IN 46202-5200</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Knierim, David S., MD</td>
<td>Loma Linda Univ Childrens Hosp</td>
<td>Loma Linda, CA 92835-2804</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Louis, Kenneth M., MD</td>
<td>3000 E. Fletcher Ave.</td>
<td>Tampa, FL 33613-4645</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Kanew, Paul M., MD</td>
<td>2790 W. Grand Blvd.</td>
<td>Detroit, MI 48202-2608</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Kosnik, Edward J., MD</td>
<td>Chatham Village Prof. Bldg.</td>
<td>Columbus, OH 43221-2417</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Luciano, Mark G., MD</td>
<td>Cleveland Clinic</td>
<td>Cleveland, OH 44195-1004</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Kasoff, Samuel S., MD</td>
<td>New York Med. Coll./Neuro.</td>
<td>Valhalla, NY 10595</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Krii, Mervin P., MD</td>
<td>158 Oakdale St.</td>
<td>Redwood City, CA 94062-1737</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Luerssen, Thomas G., MD</td>
<td>Riley Hosp. for Children</td>
<td>Indianapolis, IN 46202-5200</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Kaufman, Bruce A., MD</td>
<td>St. Louis Children's Hosp.</td>
<td>St. Louis, MO 63110</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Laurent, John P., MD</td>
<td>Clinical Care Ctr., MC 3-3435</td>
<td>Houston, TX 77030-2030</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Magid, Gail A., MD</td>
<td>1661 Saquel Dr.</td>
<td>Santa Cruz, CA 95065-1709</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Keating, Robert F., MD</td>
<td>Children's National Med. Ctr.</td>
<td>Washington, DC 20010-2401</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Laws, Edward R., Jr., MD</td>
<td>Univ. of Virginia/Neuro.</td>
<td>Charlottesville, VA 22908-0001</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Magram, Gary, MD</td>
<td>St. Christopher's Hosp.</td>
<td>Philadelphia, PA 19134</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Kelly, David L., Jr., MD</td>
<td>Bowman Gray Sch. of Med.</td>
<td>Winston-Salem, NC 27157-1029</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Lipow, Kenneth L., MD</td>
<td>267 Grant St.</td>
<td>Bridgeport, CT 06610-2805</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Manwaring, Kim Herbert, MD</td>
<td>Phoenix Children's Hosp.</td>
<td>Phoenix, AZ 85006-2513</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Kestrel, John R.W., MD</td>
<td>B.C.'s Children Hosp.</td>
<td>Vancouver, BC V6H 3V4</td>
<td>CANADA Active Member</td>
<td></td>
</tr>
<tr>
<td>Loeser, John D., MD</td>
<td>Univ. of Washington</td>
<td>Seattle, WA 98195</td>
<td>Active Member</td>
<td></td>
</tr>
<tr>
<td>Mapstone, Timothy B., MD</td>
<td>Div of Neurosurgery/CHOA</td>
<td>Birmingham, AL 35233-1711</td>
<td>Active Member</td>
<td></td>
</tr>
</tbody>
</table>
Marin, Arthur E., MD
Methodist Plaza
4499 Medical Dr., Suite #397
San Antonio, TX 78229-3713
Active Member

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

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

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

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

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

McAllister, James P., II, PhD
Cleveland Clinic Fdn., SBO
9500 Euclid Ave./Neurosurgery
Cleveland, OH 44195-0001
Associate Member

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

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

McCallum, Jack E., MD
800 6th Ave.
Suite #220
Fort Worth, TX 76104-2619
Active Member

Michelsen, W. Jost, MD
Montefiore Med. Ctr.
111 E. 210th St./Neuro.
Bronx, NY 10467-2490
Active Member

Muakass, Kamel F., MD
130 W. Exchange St.
Akron, OH 44302-1701
Active Member

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

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

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

McLanahan, C. Scott, MD
Charlotte Neuro. Assoc.
1010 Edgehill Rd., N.
Charlotte, NC 28207-1885
Active Member

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

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

McLaurin, Robert L., MD
250 William Howard Taft Rd.
Suite #210
Cincinnati, OH 45219-2664
Lifetime Member

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

Myles, S. Terence, MD
1820 Richmond Rd., S.W.
Calgary, AB T2T 5C7
CANADA
Active Member

McLone, David Gordon, MD, PhD
2300 Children's Plaza, Box 28
Chicago, IL 60614-3318
Active Member

Moel, Richard H., MD
3656 E. Eila Lee Ln.
Houston, TX 77027-4105
Lifetime Member

Nadell, Joseph M., MD
Children's Hosp.
260 Henry Clay Ave.
Naw Orleans, LA 70118-5720
Active Member

Meacham, William F., MD
Neuro. Assoc./4230 Harding Rd.
709 Medical Plaza, E.
Nashville, TN 37205
Lifetime Member

Montes, Jose L., MD
Montreal Children's Hosp.
2300 Tupper St./Room C-811
Montreal, PQ H3H 1P3
CANADA
Active Member

Nagib, Mahmoud G., MD
305 Piper Bldg.
800 E. 28th St.
Minneapolis, MN 55407-3799
Active Member
Nagle, Richard C., MD  
3000 Colby  
Suite #101  
Berkeley, CA 94705-2058  
Active Member

Partington, Michael David, MD  
1056 E. 19th Ave., B467  
Denver, CO 80218-1007  
Active Member

Pudenz, Robert H., MD  
574 Garfield Ave.  
South Pasadena, CA 91030-2254  
Lifetime Member

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

Pettisapu, Jogi V., MD  
22 W. Lake Beauty Dr.  
Suite #204  
Orlando, FL 32806-2037  
Active Member

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

Nirmel, Krishna, MD  
10 Union St.  
Natick, MA 01760-4710  
Active Member

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

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

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

Platt, Joseph H., Jr. MD  
3181 S.W. Sam Jackson Park Rd.  
Div. of Neurosurgery (L-472)  
Portland, OR 97201-3098  
Active Member

Reigel, Donald H., MD  
320 E. North Ave.  
7th Fl., South Tower  
Pittsburgh, PA 15212-4756  
Active Member

Oakes, W. Jerry, MD  
Children's Hosp. of Alabama  
1600 7th Ave., S., Suite #400  
Birmingham, AL 35233  
Active Member

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

Rekate, Harold Louis, MD  
Barrow Neurological Inst.  
2910 N. 3rd Ave.  
Phoenix, AZ 85013-4434  
Active Member

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

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

Reynolds, Arden F., Jr. MD  
Magan Med. Clinic  
420 W. Rowland St.  
Covina, CA 91723-2943  
Active Member

Pang, Dachling, MD  
Univ. of California, Davis  
2518 Stockton Blvd.  
Sacramento, CA 95817-2208  
Active Member

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

Roberts, Theodore S., MD  
Univ. of Washington/Neuro.  
CH-50, P.O. Box 5371  
Seattle, WA 98195  
Active Member

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

Portnoy, Harold D., MD  
1431 Woodward Ave.  
Bloomfield Hills, MI 48302-0550  
Active Member

Robinson, Walker L., MD  
New Jersey Neurosci. Inst.  
66 James St./Neurosurgery  
Edison, NJ 08818  
Active Member

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

Prats, Antonio R., MD  
P.O. Box 440757  
Miami, FL 33144-0757  
Active Member

Rosenblum, Bruce R., MD  
P.O. Box 159  
Colts Neck, NJ 07722-0159  
Active Member
<table>
<thead>
<tr>
<th>Name</th>
<th>Address</th>
<th>City, State, Zip</th>
<th>Active Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rosenthal, Alan, MD</td>
<td>Long Island Neuro. Assoc. 410 Lakeville Rd. New Hyde Park, NY 11042-1101</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Schneider, Steven J., MD</td>
<td>Long Island Neuro. Assoc. 410 Lakeville Rd. New Hyde Park, NY 11042-1101</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Simonds, Gary Robert, MD</td>
<td>237 Sunbury Rd. P.O. Box 14 Riverside, PA 17868-0014</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Rothman, Allen S., MD</td>
<td>1160 Fifth Ave. Suite #106 New York, NY 10029-6928</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Schut, Luis, MD</td>
<td>Children's Hosp. Philadelphia 34th and Civic Ctr. Blvd. Philadelphia, PA 19104</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Sklar, Frederick H., MD</td>
<td>1935 Motor St. Dallas, TX 75235-7794</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Ruge, John R., MD</td>
<td>630 S. Oak St. Hinsdale, IL 60521-4634</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Scott, R. Michael, MD</td>
<td>The Children's Hosp. 300 Longwood Ave., Bader 319 Boston, MA 02115-5724</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Smith, Harold P., MD</td>
<td>300 20th Ave., N. #506 Nashville, TN 37203-2131</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Rutka, James T., MD</td>
<td>Hosp. for Sick Children 555 University Ave./Neuro. Toronto, ON M5G 1X8</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Seljeskog, Edward L., MD</td>
<td>2805 Fifth St., S. Suite #110 Rapid City, SD 57701-7306</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Smith, Lenwood P., Jr. MD</td>
<td>3 Medical Park Suite #310 Columbia, SC 29203-6973</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Ruzicka, Petr O., MD</td>
<td>74-I Northfield Ave. West Orange, NJ 07052-1104</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Seiker, Robert G., MD</td>
<td>Western Pennsylvania Hosp. 4800 Friendship Ave./Neuro. Pittsburgh, PA 15224-1722</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>St. Louis, Phillip G., MD</td>
<td>1801 Cook Ave. Orlando, FL 32806-2913</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Sanford, Robert A., MD</td>
<td>Semmes-Murphy Clinic 930 Madison Ave., Suite #600 Memphis, TN 38103-3401</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Shallat, Ronald F., MD</td>
<td>3000 Colby St. Suite #101 Berkeley, CA 94705-2058</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Stein, Sherman Charles, MD</td>
<td>310 Spruce St. Philadelphia, PA 19106-4201</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Sato, Osamu, MD</td>
<td>1-5-40, Tamagawa Gakuen Machida Tokyo, 194 Japan</td>
<td></td>
<td>International Member</td>
</tr>
<tr>
<td>Shapiro, Kenneth N., MD</td>
<td>1935 Motor St. Dallas, TX 75235-7794</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Steinbok, Paul, MD</td>
<td>B.C. Children's Hosp. 4480 Oak St., Room A325 Vancouver, BC V6H 3V4  CANADA</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Scarff, Timothy B., MD</td>
<td>33 S.W. Upper Riverdale Rd. Suite #25 Riverdale, GA 30274-2626</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Shillito, John, MD</td>
<td>1109 Fearrington Post Caswell Square Pittsboro, NC 27312</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Storrs, Bruce B., MD</td>
<td>2211 Lomas Blvd., N.E. Dept. of Neurosurgery/ACC2 Albuquerque, NM 87131-5341</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Schiff, Steven J., MD</td>
<td>Children's National Med. Ctr. 111 Michigan Ave., N.W./Neuro. Washington, DC 20010-2916</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Simmons, James C., MD</td>
<td>190 Grove Park Rd. Memphis, TN 38117</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Stringer, Douglas L., MD</td>
<td>2011 Harrison Ave. Panama City, FL 32405-4545</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Name</td>
<td>Address</td>
<td>City, State, Zip</td>
<td>Phone</td>
</tr>
<tr>
<td>-----------------------------</td>
<td>----------------------------------------------</td>
<td>----------------------</td>
<td>-------</td>
</tr>
<tr>
<td>Stringer, Merle Preston, MD</td>
<td>2011 N. Harrison Ave.</td>
<td>Panama City, FL 32405-4545</td>
<td></td>
</tr>
<tr>
<td>Uscinski, Ronald H., MD</td>
<td>3301 Woodburn Rd., #209</td>
<td>Annandale, VA 22003-1229</td>
<td></td>
</tr>
<tr>
<td>Weiss, Martin H., MD</td>
<td>LAC/USC Med. Ctr., Box 786</td>
<td>Los Angeles, CA 90033</td>
<td></td>
</tr>
<tr>
<td>Sukoff, Michael H., MD</td>
<td>17602 17th St., #102-118</td>
<td>Tustin, CA 92680-1961</td>
<td></td>
</tr>
<tr>
<td>Venes, Joan L., MD</td>
<td>27517 Via Montoya</td>
<td>San Juan Capistrano, CA 92675-6364</td>
<td></td>
</tr>
<tr>
<td>White, Robert Joseph, MD PhD</td>
<td>MetroHealth Med. Ctr.</td>
<td>Cleveland, OH 44109-1998</td>
<td></td>
</tr>
<tr>
<td>Susan, Anthony F., MD</td>
<td>R.R. 3 Box 140-J</td>
<td>Burgess, VA 22432-9801</td>
<td></td>
</tr>
<tr>
<td>Ventureyra, Enrique C., MD</td>
<td>401 Smyth Rd.</td>
<td>Ottawa, ON K1H 8L1</td>
<td></td>
</tr>
<tr>
<td>Willman, Philip J. A., MD</td>
<td>3006 S. Alameda St.</td>
<td>Corpus Christi, TX 78404-2601</td>
<td></td>
</tr>
<tr>
<td>Sutton, Leslie N., MD</td>
<td>Child. Hosp. of Philadelphia</td>
<td>Philadelphia, PA 19104</td>
<td></td>
</tr>
<tr>
<td>Vries, John Kenric, MD</td>
<td>Univ. of Pittsburgh</td>
<td>Pittsburgh, PA 15261-0001</td>
<td></td>
</tr>
<tr>
<td>Wilson, Ronald J., MD</td>
<td>1313 Red River</td>
<td>Austin, TX 78701-1923</td>
<td></td>
</tr>
<tr>
<td>Taskman, Michael S., MD</td>
<td>15 Oakmont Ct.</td>
<td>San Rafael, CA 94901-2058</td>
<td></td>
</tr>
<tr>
<td>Wald, Steven L., MD</td>
<td>10 Grove Ln.</td>
<td>Shelburne, VT 05482-7232</td>
<td></td>
</tr>
<tr>
<td>Winer, Joel W., MD</td>
<td>1601 S. Queen St.</td>
<td>York, PA 17403-4830</td>
<td></td>
</tr>
<tr>
<td>Tew, John M., Jr. MD</td>
<td>Mayfield Clinic</td>
<td>Cincinnati, OH 45219</td>
<td></td>
</tr>
<tr>
<td>Waldman, John B., MD</td>
<td>Albany Med. Coll. Div. of Neurosurgery/A-61-NE</td>
<td>Albany, NY 12208</td>
<td></td>
</tr>
<tr>
<td>Winfield, Jeffrey A., MD PhD</td>
<td>Hill Med. Ctr.</td>
<td>Syracuse, NY 13210-1653</td>
<td></td>
</tr>
<tr>
<td>Tomita, Tadanori, MD</td>
<td>Children's Mem. Hosp.</td>
<td>Chicago, IL 60614-3318</td>
<td></td>
</tr>
<tr>
<td>Walker, Marion L., MD</td>
<td>Primary Children's Med. Ctr.</td>
<td>Salt Lake City, UT 84113-1100</td>
<td></td>
</tr>
<tr>
<td>Winston, Ken R., MD</td>
<td>1950 Ogden, B467</td>
<td>Denver, CO 80218-1022</td>
<td></td>
</tr>
<tr>
<td>Tulipan, Noel, MD</td>
<td>8533 McRory Ln.</td>
<td>Nashville, TN 37221-5905</td>
<td></td>
</tr>
<tr>
<td>Walsh, John Willson, MD</td>
<td>Tulane Univ. M/C/Neurosurgery</td>
<td>New Orleans, LA 70122</td>
<td></td>
</tr>
<tr>
<td>Wisoff, Jeffrey H., MD</td>
<td>New York Univ. Med. Ctr.</td>
<td>New York, NY 10016-6481</td>
<td></td>
</tr>
<tr>
<td>Turner, Michael S., MD</td>
<td>1801 N. Senate Blvd. Suite #535</td>
<td>Indianapolis, IN 46202-1228</td>
<td></td>
</tr>
<tr>
<td>Ward, John D., MD</td>
<td>Virginia Commonwealth Univ. Box 980631</td>
<td>Richmond, VA 23298-0631</td>
<td></td>
</tr>
<tr>
<td>Woodward, Meredith V., MD</td>
<td>7130 N. Sharon Ave., #100</td>
<td>Fresno, CA 93720-3328</td>
<td></td>
</tr>
<tr>
<td>Name</td>
<td>Address</td>
<td>Phone</td>
<td>Status</td>
</tr>
<tr>
<td>--------------------</td>
<td>----------------------------------------------</td>
<td>-------------</td>
<td>--------------</td>
</tr>
<tr>
<td>Yamada, Shokei, MD</td>
<td>Loma Linda Univ. Sch. of Med. Room #2539/Neurosurgery Loma Linda, CA 92350-0001</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Zampella, Edward J., MD</td>
<td>10 Parrott Mill Rd. P.O. Box 808 Chatham, NJ 07928-2744</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Zavala, L. Manuel, MD</td>
<td>2147 Mowry Ave. Suite D-1 Fremont, CA 94538-1724</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Zakalik, Karol, MD</td>
<td>Wm. Beaumont Hosp. 3535 W. 13 Mile Rd., Ste. #504 Royal Oak, MI 48073-6704</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Zanetti, Paul Henry, MD</td>
<td>5920 Saratoga Blvd. Suite #440 Corpus Christi, TX 78414-4108</td>
<td></td>
<td>Active Member</td>
</tr>
<tr>
<td>Zovickian, John G., MD</td>
<td>3000 Colby St. Suite #101 Berkeley, CA 94705-2058</td>
<td></td>
<td>Active Member</td>
</tr>
</tbody>
</table>