Section on Pediatric
Neurological Surgery
of
The American Association of
Neurological Surgeons

24th Annual Meeting

December 6-9, 1995

Ritz-Carlton, Huntington Hotel Pasadena, California





Jointly Sponsored by The American Association of Neurological Surgeons

The American Association of Neurological Surgeons

Section on Pediatric Neurological Surgery

24th Annual Meeting

Ritz-Carlton, Huntington Hotel

Pasadena, California



December 6-9, 1995



Jointly Sponsored by The American Association of Neurological Surgeons

This program is sponsored by The American Association of Neurological Surgeons.

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

The American Association of Neurological Surgeons designates this continuing education activity for 23.25 credit hours (with attendance of Breakfast Seminars) in Category I of the Physicians Recognition Award of the American Medical Association.

Program Summary

| Paolo Raimondi Lecturers 4 |
|---|
| Shulman Award Recipients4 |
| Hydrocephalus Foundation Award Recipients5 |
| Pediatric Section Chairmen 5 |
| Annual Winter Meeting Sites5 |
| Exhibitor Listing |
| Officers of the Section on Pediatric Neurological Surgery 8 |
| Program of the Pediatric Section |
| Poster Session |
| Scientific Program Abstracts |
| Scientific Program Posters |
| 1995 Membership Roster 103–114 |

Paolo Raimondi Lecturers

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

| | Kenneth Shulman Award Recipients |
|------|--|
| 1983 | Kim Manwaring: Neonatal Post-Hemorrhagic Ventriculomegaly: Management with Pulsed Lumbar Cisternostomy |
| 1984 | Arno Fried: A Laboratory Model of Shunt-Dependent Hydrocephalus |
| 1985 | Anne-Christine Duhaime: The Shaken Baby Syndrome |
| 1986 | Robert E. Breeze: CSF Formation in Acute Ventriculitis |
| 1987 | Marc R. DelBigio: Shunt-Induced Reversal of Periventricular Pathology in Experimental Hydrocephalus |
| 1988 | Scott Falci: Rear Seat-Lap Belts. Are They Really "Safe" for Children? |
| 1989 | James M. Herman: Tethered Cord as a Cause of Scoliosis in Children with a Myelomeningocele |
| 1990 | Christopher D. Heffner: Basilar Pons Attracts Its Cortical Innervation by Chemotropic Induction of Collateral Branch Formation |
| 1991 | P. David Adelson: Reorganization of the Cortical-Tectal Pathway Following Neonatal Cerebral Hemispherectomy in Cats |
| 1992 | David Frim: Effects of Biologically Delivered Neurotrophins in Animal Models of Neural Degeneration |
| 1993 | Monica C. Wehby: Metabolic Demonstration of Retained CNS Function in the Rabbit Model of Infantile Hydrocephalus |
| 1994 | Ellen Shaver: Experimental Acute Subdural Hematoma in Infant Piglets |

Hydrocephalus Foundation Award Recipients

- 1989 Eric Altschuler: Management of Persistent Ventriculomegaly Due To Altered Brain Compliance
- 1990 S.D. Michowiz: High Energy Phosphate Metabolism in Neonatal Hydrocephalus
- 1991 Nesher G. Asner: Venous Sinus Occlusion and Ventriculomegaly in Craniectomized Rabbits
- 1992 Marcia DaSilva: Reversal of High Energy Phosphate Metabolism Changes in Experimental Hydrocephalus After CSF Shunting
- 1993 Charles Bondurant: The Epidemiology of Cerebrospinal Fluid Shunting
- Monica C. Wehby Grant: The Rabbit Model for Infantile Hydrocephalus: Regional Differences in the Cortical Metabolic Response to Hydrocephalus and Shunting

Pediatric Section Chairmen

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

Annual Winter Meeting Sites

| 1972 Cincinnati | 1985 Houston |
|---------------------|-------------------------------|
| 1973 Columbus | 1986 Pittsburgh |
| 1974 Los Angeles | 1987 Chicago |
| 1975 Philadelphia | 1988 Scottsdale |
| 1976 Toronto | 1989 Washington, DC |
| 1977 Cleveland | 1990 San Diego & Pebble Beach |
| 1978 Philadelphia | 1991 Boston |
| 1979 New York | 1992 Vancouver, BC |
| 1980 New York | 1993 San Antonio, TX |
| 1981 Dallas | 1994 St. Louis, MO |
| 1982 San Francisco | 1995 Pasadena, CA |
| 1983 Toronto | 1996 Charleston, SC |
| 1984 Salt Lake City | |

Exhibitor Listing

The Section on Pediatric Neurological Surgery of The American Association of Neurological Surgeons gratefully recognizes the support of the following Exhibitors for the 1995 Pediatric Annual Meeting.

| the following Daniortors for | the 1773 | 1 cdiatric minuai micei | ing. |
|---|----------|--|------------------|
| Acra-Cut, Inc. 989 Main Street Acton, MA 01720 (508) 263-2210 | Booth 4 | Hall Surgical, Division of Zimr 11311 Concept Blvd. Largo, FL 34643 (813) 399-5112 | ner Booth 3 |
| Bio-Vascular, Inc. 2575 University Avenue St. Paul, MN 55114 (612) 603-3700 | Booth 23 | Hydrocephalus Association 870 Market Street Suite 955 San Francisco, CA 94102 (415) 776-4713 | Booth 31 |
| Camino/Heyer-Schulte NeuroCare 5955 Pacific Center Blvd. San Diego, CA 92121 (619) 455-1115 | Booth 25 | KLS - Martin, L.P. P.O. Box 50249 Jacksonville, FL 32250 (904) 641-7746 | Booth 12 |
| Clarus Medical Systems, Inc. 1000 Boone Avenue North, #100 Golden Valley, MN 55427 (612) 525-8400 | Booth 28 | Leica, Inc./Surgical Microscope Division 110 Commerce Drive Allendale, NJ 07401 (800) 526-0355 | Booths 14 and 15 |
| Codman/Johnson & Johnson Professional, Inc. 325 Paramount Drive Raynham, MA 02767-5199 (508) 828-3311 | Booth 8 | MedChem Products, Inc. 232 West Cummings Park Woburn, MA 01801 (617) 932-5900 | Booth 7 |
| Colorado Biomedical, Inc. 6851 Highway 73 Evergreen, CO 80439 (303) 674-5447 | Booth 21 | Mednext, Inc. 5490 Dexter Way West Palm Beach, FL 33407-22 (800) 863-8118 | Booth 24 |
| Cook Incorporated 925 South Curry Pike P.O. Box 489 Bloomington, IN 47402 (812) 339-2235 | Booth 13 | Midas Rex Instruments 3000 Race Street Fort Worth, TX 76111-4134 (817) 834-2601 | Booth 27 |
| Cordis Corporation 14201 NW 60th Avenue Miami Lakes, FL 33014 (305) 824-2651 | Booth 5 | Mosby/Williams & Wilkins 17960 Raymer St. Northridge, CA 91325 (818) 885-0491 | Booth 9 |
| Elekta Instruments, Inc. 8 Executive Park West Atlanta, GA 30329 (404) 315-1225 | Booth 16 | Neuro Navigational Corporatio 3180 Pullman Street Costa Mesa, CA 92626 (714) 557-9111 | n Booth 19 |

Booth 10 Valleylab, Inc. Booth 20 Phoenix Biomedical Corp. P.O. Box 80390 5920 Longbow Drive Valley Forge, PA 19484 Boulder, CO 80301-3299 (610) 539-9300 (303) 530-2300 Booth 6 Booth 18 **PMT Corporation** W.B. Saunders Company 1500 Park Road 11750 Baker Street, #12-395 Chanhassen, MN 55317 Costa Mesa, CA 92626 (612) 470-0866 (714) 297-5280 PS Medical Booths 1 and 2 Booth 22 Walter Lorenz Surgical, Inc. 125 Cremona Drive 1520 Tradeport Drive Jacksonville, FL 32218 Goleta, CA 93117 (805) 968-1546 (800) 874-7711 Radionics, Inc. Booth 11

Radionics, Inc. P.O. Box 438 22 Terry Avenue Burlington, MA 01803 (617) 272-1233

Officers of the Section on Pediatric **Neurological Surgery** of

The American Association of **Neurological Surgeons**

Chairman:

Harold L. Rekate

Secretary-Treasurer:

John P. Laurent

Executive Council:

A. Leland Albright Timothy B. Mapstone Arthur E. Marlin Paul Steinbok Kenneth R. Winston

Membership Committee:

Terrence Myles, Chairman

Rules and Regulations:

Leslie Sutton

Nominating Committee:

Arthur Marlin

Ad Hoc Committees

Committee for Resident Travel Fellowship: Kenneth R. Winston

Committee for Publication:

Richard Coulon

Committee for CPT Codes:

Arthur Marlin Peter Carmel

Committee for Child Advocacy:

Hector James

Committee for Guidelines:

Tom Luerssen Hector James

Program of the Section on Pediatric Neurological Surgery

24th Annual Meeting

Jointly Sponsored by The American Association of Neurological Surgeons

Program Schedule

WEDNESDAY, DECEMBER 6

| 12:00 NOON-5:30 PM | Nurses Seminar — Plaza Room | | |
|----------------------|--|--|--|
| 2:00 рм-5:00 рм | Monitoring/Adjudication Committee — Boardroom | | |
| 4:30 рм-8:00 рм | Registration — Ballroom Foyer | | |
| 6:30 рм-8:00 рм | Welcoming Reception — Viennesse Ballroom | | |
| THURSDAY, DECEMBER 7 | | | |
| 7:00 ам-5:00 рм | Registration — Ballroom Foyer | | |
| 7:00 ам-8:15 ам | Breakfast Seminar: Endoscopy — Salon I/II Faculty: Kim H. Manwaring, MD Kerry R. Crone, MD Alan R. Cohen, MD | | |
| 7:00 ам-8:30 ам | Continental Breakfast and Exhibits — Salon III | | |
| 8:30 AM-8:45 AM | Opening Remarks: Harold L. Rekate, MD Welcome: J. Gordon McComb, MD Salon I/II | | |

8:45 AM-10:00 AM

Scientific Session I — Salon I/II

Moderators: Derek A. Bruce, MD Arnold Menezes, MD

8:45 AM-9:00 AM

Epidemiology of Urban Pediatric Neurological Trauma: Prospective Evaluation of a Community-Based Injury Prevention Program
 E. Sander Connolly, Jr., MD, Anthony Virella, BA, Sonja Olsen, BA, Maureen Durkin, PhD, DrPH (New York, NY)

9:00 AM-9:15 AM

Nonaccidental Head Injury . . . "Who Done It?"
 Dennis L. Johnson, MD, Susan Jaskowski, RN, MSN, CCRN (Hershev, PA)

9:15 AM-9:30 AM

3. Single Photon Emission Computed Tomography in Relation to MRI and Long-Term Outcome of Pediatric Head Injury

Harvey S. Levin, PhD, Michael D. Devous, PhD, Derek Bruce, MD, Sandra B. Chapman, PhD, Harriet Harward, MS (Baltimore, MD)

9:30 AM-9:45 AM

4. Outcome Prediction After Pediatric Head Injury Using Proton Magnetic Spectroscopy

D. Kneirim, S. Ashwal, B. A. Holshouser, T. Serna, R. M. Perkin, D. B. Hinshaw, Jr. (Loma Linda, CA)

9:45 AM-10:00 AM

5. Hyperventilation and Cerebral Blood Flow in Pediatric Brain Injured Patients

Peter Skippen, MB, John Kestle, MD, Ken Poskitt, MD, Doug Cochrane, MD, Paul Steinbok, MD (Vancouver, BC)

10:00 AM-10:30 AM Coffee Break - View Exhibits — Salon III

10:30 AM-11:15 AM

Paolo Raimondi Lecturer: Robert H. Pudenz, MD

"Development of the Ventriculo-Arterial Shunt"

Salon I/II

11:15 AM-12:30 PM Scientific Session II — Salon I/II Moderators: Donald H. Reigel, MD

Thomas G. Luerssen, MD

11:15 AM-11:30 AM

6. Corticosteroids Fail To Improve Functional Outcome Following Penetrating Spinal Cord Injury in Children and Adolescents

Sean D. Lavine, MD, Michael L. Levy, MD, Lena S. Masri, MS, J. Gordon McComb, MD (Los Angeles, CA)

11:30 AM-11:45 AM

7. Pediatric SCIWORA in the MR Era: Evaluation, Treatment, and Outcome in Twenty-Eight Patients

R. J. Weil, R. Armonda, J. Dormans, A. C. Duhaime (Philadelphia, PA)

11:45 AM-12:00 NOON

8. Craniovertebral Abnormalities in Down's Syndrome Arnold H. Menezes, MD (Iowa City, IA)

12:00 NOON-12:15 PM

Antibiotic Prophylaxis in the Treatment of Traumatic CSF Fistulae Emad Eskandar, Bob Carter, David Frim (Boston, MA)

12:15 PM-12:30 PM

10. Treatment Options in the Management of Arachnoid Cysts Matthew E. Fewel, BA, Michael L. Levy, MD, J. Gordon McComb, MD (Los Angeles, CA)

12:30 PM-1:30 PM

Luncheon: Brain Tumor Study Update — Viennese Room

Speakers: Jeffrey H. Wisoff, MD Mitchel S. Berger, MD Alexander R. Sanford, MD

1:30 PM-3:00 PM

Scientific Session III — Salon I/II Moderators: Harold L. Rekate, MD Dennis L. Johnson, MD

1:30 PM-1:45 PM

11. Effects of Experimental Infantile Hydrocephalus and Surgical Decompression on Molecular Markers Associated with CNS **Plasticity**

William E. Bingaman, MD, Narongsong Boonswang, BA, Robert Connelly, MS, James P. McAllister II, PhD (Cleveland, OH)

1:45 PM-2:00 PM

12. Effects of Congenital Hydrocephalus on Molecular Markers of Neuronal Activation, Synaptogenesis, and Astrocytosis

James P. McAllister II, PhD, Robert W. Connelly, MS, Hazel C. Jones, PhD, Neil G. Harris, PhD (Cleveland, OH)

2:00 PM-2:15 PM

13. Cerebrospinal Fluid Absorptive Site of the Parasagittal Dura: A Cadaveric Study

R. J. Fox, K. E. Aronyk, K. C. Petruk, A. H. Walji (Edmonton, AB, Canada)

2:15 PM-2:30 PM

14. Cost Effective Hydrocephalus Management — The Strategic Value Method

Jogi V. Pattisapu, MD, Kay Taylor, RN, BSN, Steve Harr, RN, BSN, MBA (Orlando, FL)

2:30 рм-2:45 рм

15. Third Ventriculostomy in the Management of Hydrocephalus: A Preliminary Experience

Steven J. Schneider, MD (New Hyde Park, NY)

2:45 PM-3:00 PM

16. The Role of Ventriculoscopy in Shunt Avoidance, Reduction, or Elimination in Children with Hydrocephalus

Douglas Brockmeyer, MD, Lyn Carey, MD, Charles Lind, Marion L. Walker, MD (Salt Lake City, UT)

3:00 PM-3:30 PM

Coffee Break - View Exhibits - Salon III

3:30 PM-5:00 PM

Scientific Session IV — Salon I/II Moderators: A. Leland Albright, MD J. Parker Mickle, MD

3:30 PM-3:45 PM

17. The Copper Beaten Skull: A Correlation Between Intracranial Pressure, Skull Radiographs and CT Scans in Children with Craniosynostosis

G. F. Tuite, MD, J. Evanson, FRCR, W. K. Chong, MD, FRCR, D. P. Thompson, FRCS, W. F. Harkness, FRCS, B. M. Jones, FRCS, R. D. Hayward, FRCS (Houston, TX)

3:45 PM-4:00 PM

18. Dynamic Orthotic Cranioplasty (DOC): An Anthropometric Study of Its Effectiveness in the Treatment of Positional Plagiocephaly

Catherine E. Ripley, MA, Jeanne Pomatto, Kim H. Manwaring, MD, S. David Moss, MD, Harold L. Rekate, MD, Stephen P. Beals, MD, Edward F. Joganic, MD (Phoenix, AZ)

4:00 PM-4:15 PM

19. The Differential Diagnosis of Posterior Plagiocephaly

Joseph S. Gruss, Martin Huang, Theodore S. Roberts, John D. Loeser, Sterling Clarren, Wendy E. Mouradian, Michael L. Cunningham, Cathy J. Cornell (Seattle, WA)

4:15 PM-4:30 PM

20. Crouzon-Apert Syndrome: Frequency of Cranial Surgery and IQ Related to Time of First Surgery

Mahmoud Moradi, Derek A. Bruce (Dallas, TX)

4:30 PM-4:45 PM

21. Rigid Skeletal Fixation of the Immature Craniofacial Skeleton in the Non-Human Primate

Richard G. Ellenbogen, MD, Michael H. Mayer, MD, Jeffrey O. Hollinger, DDS, PhD, Lesley Wong, MD, Theodore M. Cole III, PhD, Joan T. Richtsmeier, PhD, Jenner Manson, John A. Bley, Jr., DVM, Dawn Yules, MD, Paul N. Manson, MD (Washington, DC)

4:45 PM-5:00 PM

22. A Combined Fronto-Orbital and Occipital Advancement Technique for Use in Total Calvarial Reconstruction

Ian F. Pollack, MD, H. Wolfgang Losken, MD, Dennis J. Hurwitz, MD (Pittsburgh, PA)

| 5:00 рм-5:30 рм | Business Meeting — Salon I/II |
|-----------------|--|
| 5:30 рм-6:30 рм | Consensus Conference on Hydrocephalus — Diplomat Room |
| 5:30 рм-6:30 рм | Neurosurgery On-Call – Pediatric Neurosurgery Meeting — Boardroom |
| 5:30 рм-8:00 рм | Head Injury Guidelines Committee — Plaza Room |

6:00 PM-7:30 PM

Shunt Design Trial Executive Committee — Counsulate Room

FRIDAY, DECEMBER 8

7:00 AM-5:00 PM

Registration — Ballroom Foyer

7:00 AM-8:15 PM

Breakfast Seminar: The Effect of Changes in Health Care Delivery of Pediatric Neurosurgery

- Salon I/II

Faculty: Bernard T. Farrari, MD, JD

7:00 AM-8:30 AM

Continental Breakfast and Exhibits — Salon III

8:30 AM-10:00 AM

Scientific Session V — Salon I/II

Moderators: Osamu Sato, MD Luis Schut, MD

8:30 AM-8:45 AM

23. The Effect of Early Craniocervical Decompression on Functional Outcome in Neonates and Young Infants with Symptomatic Chiari Malformations: Results from a Prospective Series

Ian F. Pollack, MD, Dorinne Kinnunen, BS, A. Leland Albright, MD (Pittsburgh, PA)

8:45 AM-9:00 AM

24. Bone Regrowth and Recurrence of Symptoms Following Decompression in the Infant with Arnold Chiari II Malformation Roger J. Hudgins, MD, William R. Boydston, MD, PhD (Atlanta, GA)

9:00 AM-9:15 AM

- 25. Impact of Urodynamic Evaluation on the Surgical Management of Spinal Cord Tethering
 - O. Vernet, J. P. Farmer, A. M. Houle, J. L. Montes (Montreal, PQ, Canada)

9:15 AM-9:30 AM

- 26. Chick Hindbrain Malformation Induced by Decompression of the Mesencephalic Vesicle
 - S. Robinson, R. G. Higbee, P. A. Knepper, D. G. McLone (Chicago, IL)

9:30 AM-9:45 AM

27. A Molecular Model of Secondary Neurulation Defects

Timothy M. George, MD, Michael McLone, Paul A. Knepper, MD, PhD, David G. McLone, MD, PhD (Chicago, IL)

9:45 AM-10:00 AM

28. A New Model for Understanding Encephalocele Pathogenesis

Ty J. Gluckman, BA, Timothy M. George, MD, Paul A. Knepper, MD, PhD, David G. McLone, MD, PhD (Chicago, IL)

10.00 AM-10.30 AM

Coffee Break and Exhibits — Salon III

10:30 AM-12:30 PM

Scientific Session VI — Salon I/II Moderators: Bruce B. Storrs, MD Glenn Morrison, MD

10:30 AM-10:45 AM

 Language Localization in the Dominant Hemisphere of Children Mitchel S. Berger, MD, George A. Ojemann, MD (Seattle, WA)

10:45 AM-11:00 AM

30. Can Epileptic Foci Be Suppressed with Electric Fields? Steven J. Schiff, MD, PhD (Washington, DC)

11:00 AM-11:15 AM

31. Hippocampal Activation Stops the Spread of Experimental Limbic Seizures

Mark R. Proctor, Rebecca Vaurio, Chris Hogan, Karen Gale (Washington, DC)

11:15 AM-11:30 AM

32. Subtemporal Transparahippocampal Amygdalohippocampectomy for Treatment of Mesial Temporal Lobe Epilepsy: A Novel Operation

T. S. Park, MD, Blaise F. D. Bourgeois, MD, Bruce A. Kaufman, MD (St. Louis, MO)

11:30 AM-11:45 AM

33. Corpus Callosotomy in Lennox-Gastaut Syndrome

Jeffrey P. Blount, MD, Bernard Maister, MD, Robert E. Maxwell, MD, PhD (Minneapolis, MN)

11:45 AM-12:00 NOON

34. Ablative Epilepsy Surgery in Children

Mark Lee, Joseph R. Smith, David J. Yeh, Yong D. Park (Augusta, GA)

12:00 NOON-12:15 PM

35. Results and Complications of Reoperation for Failed Epilepsy Surgery in Children

Ellen G. Shaver, MD, Glenn Morrison, MD, Antonio R. Prats, MD, A. Simon Harvey, MD, Patricia Dean, RN (Philadelphia, PA)

12:15 РМ-12:30 РМ

36. Seizure Outcome in Children with Arteriovenous Malformations Treated with Gamma Knife Radiosurgery

Peter C. Gerszten, MD, P. David Adelson, MD, Douglas Kondziolka, MD, John C. Flickinger, MD, L. Dade Lunsford, MD (Pittsburgh, PA)

12:30 рм-1:30 рм

Luncheon: Editor's Toil: "Separating the Wheat from the Chaft" — Viennese Room Speakers: Robin P. Humphries, MD Fred Epstein, MD

1:30 PM-2:15 PM

Scientific Session VII — Salon I/II Moderators: Joseph H. Piatt, MD Joan L. Venes, MD

1:30 PM-1:45 PM

37. Presence of Immunohistochemically Identifiable Tissue Plasminogen Activator in Cavernous Angiomata: A Potential Mechanism for Re-Hemorrhage and Lesion Growth

David M. Frim, Natasa Zec, Jeffrey Golden, R. Michael Scott (Boston, MA)

1:45 PM-2:00 PM

38. Dysregulation of Gene Expression in the Vasculature of Sturge-Weber Syndrome: Clues to the Pathogenesis of the Disease

R. L. P. Rhoten, MD, Y. G. Comair, MD, D. Shedid, MS, P. E. DiCorleto, PhD, C. De la Motte, PhD, M. S. Simonson, BS (Cleveland, OH)

2:00 PM-2:15 PM

39. The Management of Giant Aneurysms in Children and Adolescents

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

2:15 PM-3:30 PM Special Lecture: "Should There Be Certifi-

cation for Training in Pediatric Neurosurgery?"

Salon I/II

Speakers: R. Michael Scott, MD Martin H. Weiss, MD

3:30 PM-4:00 PM Coffee Break and Exhibits — Salon III

4:00 PM-5:00 PM Scientific Session VIII — Salon I/II

Moderators: John P. Laurent, MD T. S. Park, MD

4:00 PM-4:15 PM

40. Extent of Variation Between Centers in Electrophysiologic Techniques Used in Lumbosacral Selective Posterior Rhizotomy for Spastic Cerebral Palsy

Paul Steinbok, MBBS, FRCSC, John Kestle, BSc, MD, MSc, FRCSC (Vancouver, BC, Canada)

4:15 PM-4:30 PM

41. Electromyographic Monitoring of Cranial Nerves During Resection of Fourth Ventricular Tumors

Paul A. Grabb, MD, A. Leland Albright, MD, Robert J. Sclabassi, MD, PhD (Birmingham, AL)

4:30 PM-4:45 PM

42. Complications Associated with an Interhemispheric Operative Approach

Grant Shumaker, MD, Michael L. Levy, MD, Albert Penney, BS, J. Gordon McComb, MD (Los Angeles, CÅ)

4:45 PM-5:00 PM

43. Intraparenchymal Lesions in Patients with Leukemia or Lymphoma Felipe C. Albuquerque, MD, Marvin Nelson, MD, Corey Raffel, MD, PhD (Los Angeles, CA)

| 5:00 рм-6:00 рм | Endoscopic Shunt Surgery Trial — Ambassador Room |
|------------------|---|
| 5:00 рм-6:00 рм | Multi-Center Rhizotomy Study Group — Boardroom |
| 6:30 рм-7:30 рм | Reception — Viennese Terrace |
| 7:30 рм-11:30 рм | Annual Banquet — Viennese Ballroom |

SATURDAY, DECEMBER 9

| 7:00 am-12:00 noon | Registration — Ballroom Foyer |
|--------------------|--|
| 7:00 ам-8:15 ам | Breakfast Seminar: Update on Frameless Stereotaxy — Salon I/II Faculty: James M. Drake, MD Marion L. Walker, MD |
| 7:00 ам–8:30 ам | Continental Breakfast and Exhibits — Salon III |
| 8:30 ам-10:00 ам | Scientific Session IX — Salon I/II Moderators: Timothy B. Mapstone, MD Koreaki Mori, MD |

8:30 AM-8:45 AM

44. Surgical Management of Pediatric Thalamic Tumors: 10 Years of Experience

Jessie C. Huang, MD, Jeffrey H. Wisoff, MD, Fred J. Epstein, MD, Jeffrey C. Allen, MD (New York, NY)

8:45 AM-9:00 AM

45. Surgical Management of Pediatric Chiasmal Astrocytomas

Michael D. Medlock, Joseph R. Madsen, Liliana C. Goumnerova, Nancy J. Tarbell, Patrick D. Barnes, Douglas S. Anthony, R. Michael Scott (Boston, MA)

9:00 AM-9:15 AM

46. Anaplastic Change in Pilocytic Astrocytomas

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

9:15 AM-9:30 AM

47. Choroid Plexus Tumor Variants and Response to Surgical and Medical Management

Michael L. Levy, MD, Amy Goldfarb, BS, Grant Shumaker, MD, J. Gordon McComb, MD, Floyd H. Gilles, MD (Los Angeles, CA)

9:30 AM-9:45 AM

48. Childhood Ependymomas: Prognois Related to Location, Age, and History

Mammoud Moradi, Amir Friedman, Kenneth Shapiro, Charles Timmons, Derek A. Bruce (Dallas, TX)

9:45 AM-10:00 AM

49. Contemporary Management of Intracranial Ependymomas in Young Children (<3 Years)

R. Sanford, S. Einhaus, A. Gajjar, L. Kun (Memphis, TN)

10:00 AM-10:30 AM

Coffee Break and Exhibits — Salon III

10:30 AM-12:15 PM

Scientific Session X — Salon I/II Moderators: Leslie N. Sutton, MD David G. McLone, MD

10:30 AM-10:45 AM

50. Correlation of Chromosome 17p Loss with Clinical Outcome in Patients with Primitive Neuroectodermal Tumors

Seyed M. Emadian, PhD, MD, Jeffrey D. McDonald, MD, PhD, Dan Fults, MD (Salt Lake City, UT)

10:45 AM-11:00 AM

51. Glial Differentiation Predicts Poor Clinical Outcome in Central Nervous System Primitive Neuroectodermal Tumors

Leslie N. Sutton, MD, Anna J. Janss, MD, PhD, Anthony Yachnis, MD, Jeffrey H. Silber, MD, PhD, John O. Trojanowski, MD, PhD, Virginia M. Y. Lee, PhD, Giorgio Perilongo, MD, Lucy B. Rorke, MD, Peter C. Phillips, MD (Philadelphia, PA)

11:00 AM-11:15 AM

52. Medulloblastoma with Brain Stem Involvement: The Impact of Gross Total Resection on Outcome

R. A. Sanford, A. Gajjar, S. Einhaus, R. Heideman, A. Walter, Y. Li, J. Langston, C. Greenwald, M. Muhlbauer, J. Boyett, L. E. Kun (Memphis, TN)

11:15 AM-11:30 AM

53. Surveillance Scanning in Medulloblastoma

Dennis Shaw, Russ Geyer, Karen Lindsley, Jerrold Milstein, Mitchel S. Berger (Seattle, WA)

11:30 AM-11:45 AM

54. Value of Postoperative Surveillance Scans in the Management of Children with Some of the Common Brain Tumors

Paul Steinbok, MBBS, BSc, FRCSC, Stephen Hentschel, D. Douglas Cochrane, MD, FRCSC, John R. W. Kestle, BSc, MD, MSc, FRCSC (Vancouver, BC, Canada)

11:45 AM-12:00 NOON

55. Better Dead than Dull? Historical and Ethical Issues in the Treatment of Medulloblastoma

Jennifer C. Kernan, MD, Joseph H. Piatt, MD (Portland, OR)

12:00 NOON-12:15 AM

56. Outcome and Cost Analysis of Recurrent PNET

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

Poster Session

- 1. Anaplastic Astrocytomas of the Spinal Cord in Children Tania Shiminski-Maher, MS, CNRN, Cheryl Muszynski, MD, Diana Freed, BA, Douglas Miller, MD, David Zagzag, MD, Fred Epstein, MD (New York, NY)
- 2. Role of Oxygen Free Radicals and Adenosine in Leukocyte-Endothelial Adherence in the Newborn Pig Brain During Postasphyxic Reperfusion

Tae Sung Park, MD, Ernesto R. Gonzales, BSN, Raymond G. Maceren, BS, Aarti R. Shah, MS, Jeffrey M. Gidday, PhD (St. Louis, MO)

- 3. Design and Testing of a New Auto-Adjusting Flow Regulating Device for Shunting of CSF
 Newton Paes, MD (São Paulo, Brazil)
- 4. Pediatric Posterior Fossa Glioblastoma: MRI Findings W. Robinson, MD, Y. Numaguchi, MD, M. I. Rothman, MD, G. H. Zoarski, MD, M. Zagardo, MD, D. A. Kristt, MD (Baltimore, MD)
- Endoscopic Stereotaxic Neurosurgery: Frameless Stereotaxy with an Endoscope George I. Jallo, MD, I. Rick Abbott, MD (New York, NY)
- 6. Classification of Hydrocephalus and Outcome of Treatment Koreaki Mori, Junichi Shimada, Masahiro Kurisaka, Kiyoshi Sato, Kazuyoshi Watanabe (Kochi, Japan)
- 7. Tyrosine Hydroxylase Immunoreactivity Changes in the Nigrostrial Dopaminergic System in Experimental Hydrocephalus Shushovan Chakrabortty, Yuzuru Tashiro, James M. Drake, Toshiaki Hattori (Toronto, ON, Canada)
- 8. The Changes of Neuronal and Dendritic Calbindin-D Immunoreactivity in Cerebral Cortex, Striatum, and Hippocampus of the Hydrocephalic Rat Yuzuru Tashiro, Shushovan Chakrabortty, James M. Drake, Toshiaki Hattori (Toronto, ON, Canada)
- The Rabbit Model for Infantile Hydrocephalus: Preservation of Barrel Fields in Somatosensory Cortex Charles E. Olmstead, PhD, Katherine Y. Eng, Monica C. Wehby-Grant, MD, Warwick J. Peacock, MD, Robin S. Fisher, PhD (Los Angeles, CA)
- 10. Factors Influencing Outcome in Brain Tumor Surgery in Children Less Than Two Years of Age Keith S. Blum, DO, Steven J. Schneider, MD, Alan D. Rosenthal, MD (New Hyde Park, NY)

- 11. Head Registration Techniques in the Pediatric Age Group M. S. Alp, J. I. Ausman, M. Dujovny, F. T. Charbel, M. Misra, A. Goldberg (Chicago, IL)
- **12.** Chiari Crisis: Early Intervention and Long-Term Survival R. F. Keating, MD, J. T. Goodrich, MD, PhD (Bronx, NY)
- 13. Non-anastomotic Bypass Surgery for Childhood Moyamoya Disease Using Dural Pedicle Insertion Over the Brain Surface Combined with EGMS

Yasuko Hara, Reizo Shirane, Takashi Yoshimoto (Sendai, Japan)

- 14. Ventricular Peritoneal Shunt Malfunction Following Operative Correction of Scoliosis: Report of Three Cases Michael Egnor, MD, Wesley Carrion, MD, Susan Haralabatos, MD, Michael Wingate, BA (Stony Brook, NY)
- **15.** Seatbelt Injuries to the Pediatric Spine: A Report of 11 Cases Michael Egnor, MD, Wesley Carrion, MD, Susan Haralabatos, MD, Ira Chernoff, MD, Thomas Smith, MD (Stony Brook, NY)
- 16. Quantitative Analysis of Cerebrospinal Fluid Spaces in Children with Occipital Plagiocephaly
 Paul D. Sawin, MD, Michael G. Muhonen, MD, Arnold H. Menezes, MD (Iowa City, IA)
- 17. Use of MRI in the Evaluation of the Cervical Spine in the Multiply-Injured Young Child

R. A. Armonda, R. J. Weil, J. Dormans, J. Hunter, A. C. Duhaime (Philadelphia, PA)

- 18. Stereotactic Depth Electrodes for Localizing Seizure Foci in Children
 - P. David Adelson, MD, A. Leland Albright, MD, Peter C. Gerszten, MD, Patricia K. Crumrine, MD, John K. Vries, MD (Pittsburgh, PA)
- 19. The Role of Neuroendoscopy in the Treatment of Pineal Region Tumors
 - S. Robinson, A. R. Cohen (Cleveland, OH)
- 20. Treatment Results for 149 Medulloblastoma Patients from One Institution
 - Y. Khafaga, A. E. Kandil, A. Jamshed, M. Hassounah, E. DeVol, A. J. Gray (Riyadh, Saudi Arabia)
- 21. Brain Stem Gliomas Possibilities and Limits of Surgical Treatment

Siegfried Vogel, MD, Christiane May, MD (Berlin, Germany)

Scientific Abstracts

Epidemiology of Urban Pediatric Neurological Trauma: Prospective Evaluation of a Community-Based Injury Prevention Program

E. Sander Connolly, Jr., MD, Anthony Virella, BA, Sonja Olsen, BA, Maureen Durkin, PhD, DrPH (New York, NY)

Although neurological injury prevention programs are widely recommended, such programs rarely focus on underserved communities, and are seldomly evaluated. To evaluate whether a community-based prevention program begun in 1988 lessened neurotrauma, we prospectively studied (1983-1992) two cohorts of children aged 0-16 years living in Northern Manhattan for the incidence, type, and cause of neurological trauma based on hospital discharge records. In 973,927 person-years of overall observation, we identified 1479 pediatric neurological injuries (155/100,000/year; 21% of all injuries).

Spinal cord (1.74/100,000/y) and peripheral nerve (6.53/100,000/y) injuries were relatively rare (5%). Minor head injuries: isolated skull fractures (7.84/100,000/y) and minor concussions (<1h LOC, 71.94/100,000/y) accounted for the majority of injuries (51%), whereas severe injuries: severe concussion (>1h LOC; 1.2/100,000/v), cerebral laceration/contusion (9.69/ 100,000/y), and intracerebral hemorrhage (11.32/100,000/y), were less common (14%). Boys were more often affected, and this increased with age. Infants showed the highest incidence while young children (1-4 y) showed the lowest rates, with steady increases thereafter. Falls were most common before age 4, pedestrian MVAs most common in late childhood, and assaults most common in early adolescence. Following the preventative intervention, an 11%-37% decrease in the incidence of neurotrauma was noted in both the control and experimental populations, with targeted populations (age >5) experiencing only slightly greater improvements than nontargeted ones (20%-37% vs. 11%-30%). Decreases were limited to minor head trauma, and were unlikely to be due to the intervention. The epidemiological information provided here will, however, help in designing more effective community-based preventions in the future.

2. Nonaccidental Head Injury . . . "Who Done It?"

Dennis L. Johnson, MD, Susan Jaskowski, RN, MSN, CCRN (Hershey, PA)

The shaken baby syndrome is seldom witnessed and the victims remain silent because they have not developed the ability to talk. Criminal prosecution of the alleged perpetrator is simplified by a confession but often hangs on circumstantial and forensic evidence which attempt to deduce the time and place of the injury. What is the time interval between lethal infant shaking and onset of symptoms? "Who done it?"

Soft tissue injuries can be aged by the color of the bruise but the sensitivity is judged in days rather than hours, and the shaken infant syndrome is often associated with no external signs of trauma. Retinal hemorrhages can be dated ophthalmoscopically but only in terms of days to weeks postinjury. Intracranial blood can also be aged by MRI. Blood which is less than 6 hours old can be differentiated from blood which is greater than 6 hours but less than 24. The ability to stage hemorrhage temporally may be important to the diagnosis of shaken baby syndrome, but lack timing precision. Even postmortem findings provide only crude estimates of the time of injury.

Infants are often abused because they are irritable and crying. Crying provokes shaking, and shaking continues until the infant stops breathing and the crying stops. In a study of 28 children who suffered significant head injuries, the diagnosis of child abuse was based on intracranial hemorrhage, retinal hemorrhage, and a discordant history. Clinical or radiographic evidence of apnea was found in 21/28 (75%) and provided critical timing information. Apnea defines the sentinel event and establishes "who done it."

3. Single Photon Emission Computed Tomography in Relation to MRI and Long-Term Outcome of Pediatric Head Injury

Harvey S. Levin, PhD, Michael D. Devous, PhD, Derek Bruce, MD, Sandra B. Chapman, PhD, Harriet Harward, MS (Baltimore, MD)

Cognitive deficit and behavioral disturbance can persist in children following severe closed head injury (CHI) despite normal or minimally abnormal magnetic resonance imaging (MRI). Although our group has shown that the volume of prefrontal lesions contributes to predicting cognitive impairment, interpretation of our MRI and outcome data is consistent with disturbance of a prefrontal "system." Consequently, we postulated that single photon emission computed tomography (SPECT) would disclose a broader zone of cerebral dysfunction than the areas of abnormal signal on MRI. Twenty-six CHI (GCS: x=8.8, SD=3.8) patients (age at study: x=12.2, SD=2.8) had SPECT within 2 months of their MRI after a mean postinjury interval of 3.8 years (SD=1.3). The Vineland Adaptive Behavior Composite score, California Verbal Learning Test, Verbal Fluency, and Wisconsin Card Sorting Test assessed outcome. Four groups were identified based on comparing the MRI-SPECT findings: (1) Consistent Cortical Group (n=7) in whom SPECT included and extended beyond the region of abnormal signal on MRI; (2) Inconsistent Cortical Group (n=10) in whom the hypoperfused region on SPECT was noncontiguous with the area of abnormal signal; (3) Positive SPECT-Normal MRI Group (n=8); (4) Normal SPECT and Normal MRI (n=1). Combining data for Groups 1 and 2 disclosed MRI evidence of white matter lesions in 12 patients, whereas MRI findings were confined to the cerebral cortex in 13 patients. Patients with white matter lesions had more impaired memory (p<.02) than the cases whose SPECT and MRI findings were confined to the cerebral cortex, but these groups did not differ on the other outcome measures.

4. Outcome Prediction After Pediatric Head Injury Using Proton Magnetic Spectroscopy

D. Kneirim, S. Ashwal, B. A. Holshouser, T. Serna, R. M. Perkin, D. B. Hinshaw, Jr. (Loma Linda, CA)

We studied 19 children (ages 2-150 months) 3 to 31 days after traumatic brain injury (nonaccidental [n=11]; other trauma [n=8]) to determine whether proton magnetic spectroscopy (¹H-MRS) was useful in predicting neurological outcome. ¹H-MRS data (occipital gray/parietal white matter; 1.5T; 8 cm³ Vol, STEAM sequence TE=20 ms, TR=3000 ms) were expressed as ratios of metabolite peak areas. Clinical data included admission Glasgow Coma Scale, pH, glucose, and the number of days unconscious at the time of ¹H-MRS. Outcome was measured using the Glasgow Outcome Scale. Patients with bad outcomes (severe disability, vegetative state, death; n=12) were compared to patients with good or mild/moderate disability outcomes; n=7). We performed a discriminant analysis to assess ability to predict outcome in patients stratified by outcome (good/moderate vs bad) and also by age (<18 months vs ≥18 months). We were able to predict outcome as follows:

| | All patients (n=19) | Pts <18 mos (n=12) | Pts ≥18 mos (n=7) |
|--------------------------------|---------------------|---------------------|---------------------|
| Clinical data alone | 88% (fp=0%; fn=0%) | 100% (fp=0%; fn=0%) | 83% (fp=50%; |
| fn=0%) | | | |
| ¹ H-MRS (occipital) | 77% (fp=9%; fn=20%) | 73% (fp=60%; fn=0%) | 100% (fp=0%; fn=0%) |
| Clin + H-MRS (occ) | 100% (fp=0%; fn=0%) | 100% (fp=0%; fn=0%) | 100% (fp=0%; fn=0%) |

fp = % false positive cases, that is the % of patients who had an abnormal variable yet had a good outcome.

'H-MRS (occipital) alone in patients ≥18 mos was very accurate in predicting outcome (100% with no false +/-) and in all patients the combination of clinical and spectroscopy variables similarly predicted outcome. The data suggest that 'H-MRS is a useful technique in conjunction with the clinical assessment to predict neurological outcome after severe traumatic brain injury in children.

fn = % false negative cases, that is the % of patients who had a normal variable yet had an abnormal outcome.

Hyperventilation and Cerebral Blood Flow in Pediatric Brain Injured Patients

Peter Skippen, MB, John Kestle, MD, Ken Poskitt, MD, Doug Cochrane, MD, Paul Steinbok, MD (Vancouver, BC)

Hyperventilation is a common component of the management of children with acute cerebral insults. Recent cerebral blood flow (CBF) studies in adults have demonstrated regional and global cerebral ischemia with hyperventilation. We observed regional CBF responses to changes in PaCO₂ in pediatric brain injured patients. Resuscitation, intubation, ventilation, diagnostic CT scans, and surgical interventions were performed according to standard practice. Xenon CT scans were performed at various levels of PaCO₂ at admission, at 24-72 hours after injury, and at greater than 72 hours after injury. Jugular venous bulb catheters were used to measure cerebral oxygen extraction (CEO₂).

Thirteen children underwent a total of 77 Xenon CT scans. All had a Glasgow Coma Scale score less than 8. Three required evacuation of a hematoma.

Hyperventilation-induced global oligemia (CBF < 20 ml/100 gm per minute) in two patients. The proportion of patients with regional oligemia on their scan was significantly higher with a lower PaCO₂ (McNemar chi square = 7.11, p=0.008). The agreement between oxygen extraction data and the presence of regional oligemia was good (actual agreement = 75%, kappa = 0.50).

Changes in cerebral blood flow with hyperventilation were common. Changes within the "acceptable range" induced oligemia. The role of hyperventilation in pediatric brain injury requires further study.

6. Corticosteroids Fail To Improve Functional Outcome Following Penetrating Spinal Cord Injury in Children and Adolescents

Sean D. Lavine, MD, Michael L. Levy, MD, Lena S. Masri, MS, J. Gordon McComb, MD (Los Angeles, CA)

Treatment with high-dose intravenous methylprednisolone has become the standard of care for acute nonpenetrating spinal cord injury since the results of the multicenter second National Acute Spinal Cord Injury Study (NASCIS) were published in 1990. By design, NASCIS excluded patients with spinal injury secondary to gunshot wounds (GSW); therefore, the efficacy of corticosteroids in the management of penetrating spinal cord injury is unclear.

A recent study by our institution demonstrated that gunshot wounds equal motor vehicle injuries as the most frequent cause of pediatric spinal injury, and that GSWs were responsible for 50% of adolescent (12-16 years) spinal cord injury rehabilitation admissions. We have previously reported the failure of corticosteroids to improve functional outcome in adults with penetrating spinal cord injury, and are reporting the results of our analysis in children and adolescents.

We have retrospectively reviewed 252 patients who presented to our institution from 1990-1995, 70 of which were less than 20 years of age. Twenty-four received corticosteroid per the NASCIS protocol and 46 had management that excluded the use of corticosteroids. Evaluation of their neurological status was performed on admission, and after transfer for rehabilitative care at 3, 6, 12, and 24 months. The population was 91% male, mean age 17 years, with mean hospitalization 105 days.

Outcome variables included: independence status, degree of deficit and preservation of function on discharge and revealed no differences between groups. The variable most predictive of outcome was degree of deficit on admission (p 0.0001).

We conclude that the use of intravenous corticosteroid per the NASCIS protocol does not significantly increase complication rate nor does it improve functional outcome in children and adolescents sustaining spinal cord injury from gunshot wounds.

7. Pediatric SCIWORA in the MR Era: Evaluation, Treatment, and Outcome in Twenty-Eight Patients

R. J. Weil, R. Armonda, J. Dormans, A. C. Duhaime (Philadelphia, PA)

Rationale: To assess mode of injury, presentation, MR findings, treatment, and long-term outcome in 28 children who presented with spinal cord injury without plain radiographic abnormality (SCIWORA).

Methods: We retrospectively reviewed the hospital and outpatient records of 28 children with SCIWORA presenting between 2/89 and 4/95 and completed long-term followup interviews with 24 (86%).

Results: Of the 28 patients, 24 (86%) were male; mean age was 9.6 years (range 14 months to 16 years). Mechanism of injury included sports (13 patients), motor vehicle accidents (9 patients), falls (5 patients), and assault (1 patient). Hyperextension was more common than hyperflexion. Symptoms included neck pain, weakness, and/or parasthesias; in 7 patients symptom had resolved by arrival at the hospital (average 2 hours after injury). Physical findings included weakness (13 patients), sensory abnormalities (11 patients), upper motor neuron signs (6 patients), and gait disturbance (4 patients).

Twenty-seven patients underwent MRI evaluation, of which 19 showed no abnormality. The other 8 patients showed spinal cord contusion (4 patients) and/or soft-tissue abnormality (hemorrhage, ligamentous injury, edema) (6 patients).

Treatment included methylprednisolone in 16 patients, prolonged cervical immobilization with collar or brace (6-8 weeks) in 7 patients with cervical MRI abnormalities, and briefer immobilization (2-6 weeks) for 3 additional patients with persistent neck pain.

Follow-up flexion-extension plain films in patients treated with immobilization showed no instability. All but one patient with a complete cord injury recovered to a Frankel Grade E. At follow-up (mean 28 months, range 2-76 months) all other patients had a normal exam, had resumed normal activities including sports, and had suffered no new neurologic symptoms or deficits.

Conclusions: Children with SCIWORA have a good outcome unless a complete cord injury is present. Those with a normal MRI and resolution of symptoms appear to do well without prolonged immobilization. In this series no child suffered a second injury or known late instability at a mean of 28 months after injury; however, the optimum duration of immobilization for children with deficits or MRI findings cannot be determined from this study.

8. Craniovertebral Abnormalities in Down's Syndrome

Arnold H. Menezes, MD (Iowa City, IA)

A prospective analysis of all symptomatic Down's syndrome patients with craniovertebral (CV) pathology was undertaken to identify associated abnormalities, management options, and outcome (1979-1995).

Thirty-nine patients (3-42 years) underwent neurodiagnostic definition of the lesions, reducibility, and neurovascular encroachment. Eleven were ineligible for surgery (severe cardiac or neurobehavior dysfunction) and had a rotary O-C1-C2 luxation. Four had acute rotary abnormality after respiratory infection achieving ligamentous reconstitution with immobilization. Twenty-four underwent surgical therapy (13 male, 11 female). Occipito-atlantoaxial instability in 13 of the 24, 10 of the 13 having rotary component. Eleven had primary atlantoaxial instability. "Fixed C1-C2 subluxation" was the presentation in 10. Four previous C1-C2 fusions and O-C1-C2 fusion which failed were encountered. Os odontoideum was detected in five. Predental space ranged from 8-13 mm. Twenty-two of 24 lesions were reducible.

Two irreducible dislocations underwent ventral decompression and dorsal O-C3 fusion. A dorsal C1-C2 fusion was made in 11; primary dorsal O-C3 fusion in 11. Three required instrumentation for gross instability. Halo immobilization was maintained in all, three months for atlantoaxial and six months for occipitocervical fusions. One fusion failure occurred in a child with repeated varied infections. Spontaneous ventral fusion occurred in 4 of the 24 fusion patients, 2 with juvenile rheumatoid arthritis and 2 without.

Conclusions: 1) unrecognized occipito-atlantoaxial pathology is cause for atlantoaxial fusion failure; 2) "fixed subluxation" does not protect against neurological deterioration; 3) active CV synovitis is sometimes present; 4) os odontoideum signifies early trauma and adds to the pathology; and 5) appropriate CV fusion constructs with extended immobilization must be entertained.

Antibiotic Prophylaxis in the Treatment of Traumatic CSF Fistulae

Emad Eskandar, Bob Carter, David Frim (Boston, MA)

Open skull fracture and traumatic CSF fistula (otorrhea or rhinorrhea) are frequent complications of head injury in the pediatric population. The role of prophylactic antibiotics in the management of these injuries remains controversial with numerous studies showing conflicting results. We have reviewed the literature from 1970 to the present and found 11 studies in which patients with traumatic CSF fistulae received either prophylactic antibiotics or no treatment. A meta-analysis of these pooled results (total patient number, adult and pediatric, n = 800) revealed that there is a significantly higher risk of post-traumatic meningitis with no treatment when compared with a prophylactic antibiotic regimen (odds ratio 2.3:1, 95% confidence bounds 1.4 - 3.7). Based on this analysis, and a review of the most likely pathogens, we have formulated a 5-day antibiotic regimen (nafcillin, ceftriaxone, and metronidazole) for the prophylaxis of traumatic CSF fistula and open skull fracture. To date, we have managed 40 consecutive adult and pediatric patients with this protocol. We have encountered no CSF related infections (median follow up of 3 months) and minimal morbidity (one antibiotic related skin rash). Based on these results, we suggest that a short course of prophylactic antibiotics may be appropriate in the management of patients with open skull fractures or traumatic CSF fistulae.

10. Treatment Options in the Management of Arachnoid Cysts

Matthew E. Fewel, BA, Michael L. Levy, MD, J. Gordon McComb, MD (Los Angeles, CA)

Treatment options for the management of intracranial arachnoid cysts include craniotomy for fenestration or shunting. The records of 80 patients with intracranial arachnoid cysts treated from 1976 to 1995 were reviewed. These patients are divided into two groups, those initially treated from 1976 to 1986 (previously reported), and those from 1987 to 1995 to see if any improvement in outcome had occurred. There were 31 patients (20 males/11 females) with 34 cysts treated during 1976-86 and 49 patients (34 males/15 females) with 53 cysts treated from 1987-95. Mean age at presentation for all cases was 5 years (range 3 days-18 years). The most common cyst location was the middle fossa (40 cysts), followed by the posterior fossa (19 cysts), suprasellar region (10 cysts), interhemispheric (5 cysts), over the convexity (5 cysts), quadrigeminal cistern (4 cysts), and other locations (4 cysts). The average length of hospital stay for fenestration patients was 5½ days.

In our series, 24 cysts from 1976-86 and 43 cysts from 1987-95 were initially fenestrated. Fifty-four percent of those initially fenestrated from 1976-86 required no further treatment compared to 58% from 1987-95. The success rate among those patients without associated hydrocephalus was higher than those with hydrocephalus; 67% (1976-86) and 74% (1986-95) without hydrocephalus required no additional treatment versus 42% (1976-86) and 31% (1986-95) with hydrocephalus. Twenty-nine percent of those initially fenestrated from 1976-86 required a subsequent cyst-peritoneal shunt, compared to only 16% from 1987-95. Seventeen percent from 1976-86 required subsequent ventriculo-peritoneal shunt (VPS), compared to 21% from 1987-95. All of these VP shunts were placed in patients who initially had presented with associated hydrocephalus.

We consider the avoidance of a shunt as a primary goal in the care of these patients. Although not statistically significant, the second group of patients (1987-95) had a higher success rate for fenestration alone. We recommend that in patients without evidence of hydrocephalus, cyst fenestration be considered as the primary procedure, as 72% of the patients in the two series remain shunt free. In those patients with hydrocephalus, we still recommend cyst fenestration, although many will require a VPS.

11. Effects of Experimental Infantile Hydrocephalus and Surgical Decompression on Molecular Markers Associated with CNS Plasticity

William E. Bingaman, MD, Narongsong Boonswang, BA, Robert Connelly, MS, James P. McAllister II, PhD (Cleveland, OH)

A feline model of kaolin-induced hydrocephalus was used to investigate changes in gap-43 and cellular src (c-src) expression in the hydrocephalic developing central nervous system (CNS). The expression of these genes correlates with CNS plasticity and neuronal differentiation, thus changes during hydrocephalus (pre- and posttreatment) may occur.

Hydrocephalus was induced by intracisternal injection of kaolin in 10-day-old kittens. Animals were stratified into acute (25-day survival), treated (shunted: 35-day and 85-day survival), and chronic (85-day survival) hydrocephalic groups. Each experimental group was compared with saline injected age-matched control animals. Ribonucleic acid (RNA) and total protein were isolated from primary visual cortex (area 17) and analyzed by northern and western blotting techniques for expression of gap-43 and c-src messenger RNA (mRNA) and protein.

Results demonstrate reduced levels of gap-43 mRNA and protein in acute hydrocephalic animals (19% and 20%, less respectively). Ventriculoperitoneal shunting returned these levels to near those of control animals at 35 days of age (6% less and 12% more, respectively). In 85-day-old shunted and chronic hydrocephalic animals, gap-43 mRNA levels were slightly reduced compared to controls (13% less), while protein levels were increased (45% increase in shunted animals and 58% increase in chronic hydrocephalic animals).

Our preliminary results with c-src expression are limited to western analysis of protein levels. These include a 16% increase in acute hydrocephalic animals, a 6% increase in 35-day-old shunted animals, a 19% increase in 85-day-old shunted animals, and a 25% increase in 85-day-old chronically hydrocephalic animals.

These results suggest that synaptogenesis is impaired during acute hydrocephalus. Gap-43 protein may be up-regulated in an attempt to restore synaptic connections in the hydrocephalic brain. VP shunting lessened but did not abolish this response, suggesting that synaptogenesis is impaired early and recovery takes place over an extended period of time. Interestingly, gap-43 protein levels increase in both shunted and chronically hydrocephalic animals, implying that CNS plasticity may be resilient to injury from long-standing infantile hydrocephalus. C-src upregulation may reflect a generalized neuronal protection mechanism, however, more work in characterizing this response is necessary.

12. Effects of Congenital Hydrocephalus on Molecular Markers of Neuronal Activation, Synaptogenesis, and Astrocytosis

James P. McAllister II, PhD, Robert W. Connelly, MS, Hazel C. Jones, PhD, Neil G. Harris, PhD (Cleveland, OH)

In an attempt to identify when congenital hydrocephalus first influences neuronal and glial elements in the brain, the present study has utilized the H-Tx rat to examine RNA expression and protein levels of fos, GAP-43, and glial fibrillary acidic protein (GFAP). These molecules are reliable markers for neuronal activation, axonal and synaptic growth, and astrocytosis, respectively. In this model, ventriculomegaly develops prenatally due to aqueductal stenosis and progresses rapidly during the first postnatal month, with eventual death at 4-6 weeks after birth. In the present study, fresh frozen tissue from the frontal neocortex, (sensorimotor), occipital neocortex (auditory and visual), and combined neostriatum and basal forebrain (basal ganglia) was obtained from 2-4 hydrocephalic and 2-4 agematched normal littermates at 6, 12, and 21 days of age, This tissue was processed for Northern blot analysis of RNA and Western blot analysis of protein. In all areas, statistically significant changes did not occur until day 21. when most animals were severely hydrocephalic. The occipital neocortex was more affected than the frontal, with fos expression declining to 55% (p<0.05) of controls. Although GAP-43 declined to 37% of controls in the occipital neocortex, this change was not statistically significant (p=0.08). GFAP exhibited the most dramatic changes, with 47%-52% increases at 12 days in the cortex and 105%-155% increases at 21 days. The basal ganglia followed a similar pattern of 22 and 43% fos decreases and 106 and 167% GFAP increases at 12 and 21 days, respectively. Taken together, these preliminary data suggest that GFAP may be a reliable marker for the pathophysiological effects of hydrocephalus, and that the ability of neurons to respond to stimuli and the proliferation of growth cones and synapses may not be impaired until relatively late stages of this disorder.

13. Cerebrospinal Fluid Absorptive Site of the Parasagittal Dura: A Cadaveric Study

R. J. Fox, K. E. Aronyk, K. C. Petruk, A. H. Walji (Edmonton, AB, Canada)

Cerebrospinal fluid (CSF) absorption occurs primarily via arachnoid granulations (AG) in the superior sagittal sinus (SSS) and lacunae laterales (LL) in parasagittal dura. Anatomic descriptions of this region suggest a network of intradural channels, but lack demonstrations of their extent and relationship to AG.

The SSS and parasagittal dura of 20 formalin-fixed adult cadavers, and 15 fresh autopsy specimens of ages 18 weeks gestation to 80 years were studied using light microscopy (LM), scanning and transmission electron microscopy (SEM, TEM), and corrosion casting. Intradural injections into the parasagittal region were performed in two formalin-fixed and four fresh autopsy adults using normal saline and corrosion casting medium.

Extensive, laterally-branching networks of intradural channels of 0.02 to 2.0 mm diameter were noted in all specimens. They connected to the SSS through regularly spaced trunks in its sidewall, or into LL, and extended up to 3 cm laterally. Their size and frequency increased with age. They were lined with endothelium similar to that of the LL and SSS, with positive factor VIII antibody staining and tight junctions. The LL were a coalescence of channels. The dural underside was trabeculated where these channels were abundant. AG were inserted between these trabeculae in sheets. In regions of dura where channels were sparse or absent, the dural underside was consistently smooth and lacked AG. The underlying cortical veins opened directly into the SSS, and were generally unrelated to the intradural channels.

Intradural parasagittal injections readily accessed the SSS via the parasagittal intradural channels with pressures of 0-20 cm water at an infusion rate of 1.5 mL/minute.

These channels appear to represent important pathways of CSF conduction to the SSS, and may represent a potential site for purely intracranial CSF diversion.

14. Cost Effective Hydrocephalus Management — The Strategic Value Method

Jogi V. Pattisapu, MD, Kay Taylor, RN, BSN, Steve Harr, RN, BSN, MBA (Orlando, FL)

Approximately 25,000 shunt operations are performed annually in the United States. It is predicted that a child with hydrocephalus will require 2.2 shunt operations over a 10-year period. Each hospitalization for shunt revision costs approximately \$20,000–\$25,000. Over the past 3 years, we have analyzed the cost of hydrocephalus management and altered our shunting techniques appropriately.

This study is based on financial review of 335 shunt operations (35%–40% of total surgical cases) with progressive planning and alteration of our shunt management protocol. Complication and infection rates compared favorably with the reported literature. We also obtained cost estimates from four Pediatric Neurosurgery programs. CFIS and IAMETER data were analyzed to assess patient care costs and hospital charges.

It is difficult to compare cost analysis systematically, since there are many variables which factor into the final algorithms. In 1992, the average cost for shunt malfunction with revision was \$20,902. Certain item charges could not be reduced/deducted due to manufacturers' costs/fixed prices (shunt apparatus, drugs, radiographic studies, etc.). However, significant cost reduction was achieved in ER management, OR expenses/supplies, and postoperative care.

Overall, expense reduction to \$15,601 (a 25.4% decrease) was realized after appropriate strategic value practice changes. Specific cost reduction measures including diminishing unnecessary preoperative evaluations (x-ray, laboratory, etc.), minimizing intraoperative supplies (sutures, drugs, instrumentation, etc.), and altering postoperative management practices (monitored Special Care beds vs. ICU, perioperative antibiotics, increased education/awareness, etc.). Decreased complication and infection rates have also contributed to lowering costs.

These introspective changes have positively impacted on our pediatric neurosurgical practice, with improved efficacy and cost-efficient hydrocephalus management

15. Third Ventriculostomy in the Management of Hydrocephalus: A Preliminary Experience

Steven J. Schneider, MD (New Hyde Park, NY)

Third ventriculostomy (TV) is quickly emerging as a successful treatment modality in the management of select hydrocephalus patients. Between 1991-1995, 300 neuroendoscopic procedures were performed and 48 patients were considered for TV. Selection criteria was initially based on MRI examinations and more recently on Cine-MRI. Evidence of absent or insufficient aqueductal flow or patent aqueduct with outlet of the fourth obstruction along with some degree of dilatation of the third ventricle were considered as possible candidates for TV. Patient age ranged from 1 week to 48 years (average 9 years). The etiology of hydrocephalus was congenital aqueductal stenosis in 11 patients, tumor in 17, post-intraventricular hemorrhage in 10, postinfection in 6, and myelodysplasia in 4. Four patients had been previously radiated. Successful TV was performed in 38 of 48 patients based upon postoperative criteria including: amelioration of symptoms, normalization of ICP, and reduction of ventricular size and flow on Cine-MRI. In 4 failed procedures, repeat attempts were successful in 2. In 4 patients harboring shunt systems, 3 were successfully removed. Complications included a partial third nerve paresis in 1 patient, transient diabetes insipidus in 1. transient parinauds in 2, and memory deficit in 1. Mean follow-up was 1 vear. The evolution, impact, and efficacy of our surgical experience in TV is discussed in detail.

16. The Role of Ventriculoscopy in Shunt Avoidance, Reduction, or Elimination in Children with Hydrocephalus

Douglas Brockmeyer, MD, Lyn Carey, MD, Charles Lind, Marion L. Walker, MD (Salt Lake City, UT)

From July 1992 to December 1994 we evaluated and treated 386 patients with hydrocephalus at our institution. One thousand one hundred twentyfive shunt-related procedures were performed on those patients, for an average of 3.2 procedures per patient. Seventy-four patients underwent a total of 98 ventriculoscopic-aided procedures (8.7% of the total number of procedures). We performed 49 third ventriculostomies, 14 septal fenestrations, and 36 cyst fenestrations. The indications for surgery included: post-intraventricular hemorrhage (IVH) (with or without loculated fluid cavities), 23; aqueductal stenosis (AS), 16; myelomeningocele (MMC), 15; supratentorial tumor, 4; arachnoid cyst, 3; posterior fossa tumor (PFT), 3; tectal plate tumor (TPT), 4; postmeningitis, 2; unilateral hydrocephalus, 1; congenital hydrocephalus, 1; and congenital pelvic tumor with spina bifida, 1. We were able to avoid placing shunts in 14 patients by performing 12 third ventriculostomies, and 1 septal fenestration. For 21 patients, their overall shunt burden was reduced after performing 19 cvst fenestrations, 11 septal fenestrations, and 1 third ventriculostomy. We were able to eliminate shunts entirely in 7 patients by performing 7 third ventriculostomies. There were no complications directly related to ventriculoscopy. However, 3 ventriculoscopy patients had serious CSF infections resulting in prolonged hospital stays and eventual shunting. Thirty-two of 74 patients eventually required shunting for an overall 44% failure rate. We conclude that ventriculoscopy is useful for the avoidance, reduction, and elimination of shunts in children with hydrocephalus.

17. The Copper Beaten Skull: A Correlation Between Intracranial Pressure, Skull Radiographs and CT Scans in Children with Craniosynostosis

- G. F. Tuite, MD, J. Evanson, FRCR, W. K. Chong, MD, FRCR,
- D. P. Thompson, FRCS, W. F. Harkness, FRCS, B. M. Jones, FRCS,
- R. D. Hayward, FRCS (Houston, TX)

The copper beaten appearance of the skull, as well as other skull radiograph and CT scan findings in children with craniosynostosis, is often interpreted by clinicians as evidence of elevated ICP. However, a correlation between radiographic findings and measured ICP has not been previously demonstrated, and their usefulness in detecting elevated ICP has not been defined.

To address these issues, 123 children with craniosynostosis who had skull radiographs and ICP monitoring were studied. To assess the specificity of certain radiographic findings to craniofacial patients, skull radiographs of craniofacial patients were compared to those of age- and sex-matched controls. In craniofacial patients, findings on skull radiographs were compared to CT scans of the brain. Radiographic findings were then correlated with sleeping ICP, which was measured using a Camino fiberoptic ICP monitor. All radiographs were independently analyzed by two radiologists who were blinded to clinical and ICP data.

Diffuse copper beating, erosion of the dorsum sella, and suture diastasis were more common in craniosynostosis patients than in controls (p<0.05), but the presence of copper beating was no more common in children with craniosynostosis. ICP was higher when diffuse copper beating, dorsum sellar erosion, suture diastasis, or narrowing of basal cisterns were present (p<0.05). While some skull radiograph and CT findings did correlate with ICP, their sensitivity in detecting elevated ICP was universally low and their use to screen for elevated ICP in children with craniosynostosis is not recommended.

18. Dynamic Orthotic Cranioplasty (DOC): An Anthropometric Study of Its Effectiveness in the Treatment of Positional Plagiocephaly

Catherine E. Ripley, MA, Jeanne Pomatto, Kim H. Manwaring, MD, S. David Moss, MD, Harold L. Rekate, MD, Stephen P. Beals, MD, Edward F. Joganic, MD (Phoenix, AZ)

Dynamic orthotic cranioplasty (DOC) was developed to treat positional deformation of the head in infants. These deformations occur as a result of external forces on an infant's craniofacial skeleton and often result in complex multistructural asymmetries of the cranial vault, skull base, and face.

DOC is a proprietary semirigid thermoplastic construct incorporating a low durometer polyurethane foam liner that is designed to apply dynamic corrective forces to cranial prominences while leaving voids in flattened areas for growth to occur. The custom cranial remodeling band is fabricated over a corrected positive model derived from a plaster of paris impression taken from the infant's head.

During the period 1988-1995, DOC was utilized for the correction of positional plagiocephaly in 697 infants across 3 centers. This study was performed to investigate the effectiveness of DOC in the treatment of 535 patients at the Phoenix center.

Results were determined by longitudinal anthropometric measurements of pre- and posttreatment patients through 24 months of age. Analysis of the resultant data show a) plagiocephalic infants treated with DOC consistently correct to or toward normocephaly, b) there is no relapse following treatment regardless of age, c) the pattern of correction is predictable, d) there is no evidence for spontaneous correction, e) DOC corrects skull base asymmetry, and f) rapid correction is more readily achieved during early infancy. Clinical observation and preliminary anthropometric data from other centers employing DOC substantiate these findings, thus validating the universality of this design and treatment protocol.

19. The Differential Diagnosis of Posterior Plagiocephaly

Joseph S. Gruss, Martin Huang, Theodore S. Roberts, John D. Loeser, Sterling Clarren, Wendy E. Mouradian, Michael L. Cunningham, Cathy J. Cornell (Seattle, WA)

The diagnosis and treatment of posterior plagiocephaly is one of the most controversial aspects of craniofacial surgery. The features of true lambdoid synostosis versus those of deformational plagiocephaly secondary to positional molding are inadequately described in the literature and poorly understood. This has resulted in many infants in several craniofacial centers across the country undergoing major intracranial procedures for nonsynostotic plagiocephaly. The purpose of this study was to describe the detailed clinical, imaging and operative features of true lambdoid synostosis and contrast them with the features of deformational plagiocephaly. During a 4-year period form 1991 to 1994, 102 patients with posterior plagiocephaly were assessed in a large multidisciplinary craniofacial program. During the same period, 130 patients with craniosynostosis received surgical treatment. All patients were examined by a Pediatric Dysmorphologist, Craniofacial Surgeon, and Pediatric Neurosurgeon. Diagnostic imaging was performed where indicated. Patients diagnosed with lambdoid lambdoid synostosis and severe and progressive positional molding underwent surgical correction using standard craniofacial techniques. Only 4 patients manifested the clinical, imaging, and operative features of unilambdoid synostosis, giving an incidence among all cases of craniosynostosis of 3.1%. Only 3 among the 98 patients with positional molding required surgical intervention. All the patients with unilambdoid synostosis had a thick ridge over the fused suture, identical to that found in other forms of craniosynostosis, with compensatory contralateral parietal and frontal bossing, and an ipsilateral occipitomastoid bulge. The skull base had an ipsilateral inferior tilt, with a corresponding inferior and posterior displacement of the ipsilateral ear. These characteristics were completely opposite to the finding in the 98 patients who had positional molding with open lambdoid sutures, and prove conclusively that true unilambdoid synostosis exists as a specific but rare entity. Awareness of the features of unilambdoid synostosis will allow more accurate diagnosis and appropriate treatment of posterior plagiocephaly in general, and in particular will avoid unnecessary surgical intervention in patients with deformational posterior plagiocephaly.

20. Crouzon-Apert Syndrome: Frequency of Cranial Surgery and IQ Related to Time of First Surgery

Mahmoud Moradi, Derek A. Bruce (Dallas, TX)

Thirty-nine children with Apert or Crouzon disease were studied with prospectively collected data collated retrospectively as to date of first surgery, frequency of cranial surgery, and IQ. There are 19 children with Apert disease and 18 with Crouzon syndrome, the current mean age of the children is 12.9 years and 16.7 years, respectively. The mean age at first surgery for the children with Apert disease was 4.4 ± 2.2 months and for the Crouzon children 11.8 ± 13 months. The average number of operations on the cranium was 2.4 ± 0.8 and 3.1 ± 5.0 , respectively. This was a surprise that the children with Crouzon disease had their first operation later in the first year but on average required more cranial operations than the Apert children. In part this was biased by one child who had 20 operations. The mean IQ of the two groups was significantly different with the Apert children having substantially lower IQ than the Crouzon children, 75.1 ± 12.1 vs 92.3 ± 19.5, and the later failing in the normal IO range. There was a clear trend showing that surgery prior to the first 3 months of life was associated with a higher number of repeat cranial operations and a lower IQ. This data is interpreted as arguing against strip craniectomy early in life for these complex syndromes and in favor of a planned craniofacial operation as late in the first year as possible. We found no support for the notion that very early suture release is required to preserve IO indeed the reverse may be true. These results and their implications will be discussed.

21. Rigid Skeletal Fixation of the Immature Craniofacial Skeleton in the Non-Human Primate

Richard G. Ellenbogen, MD, Michael H. Mayer, MD, Jeffrey O. Hollinger, DDS, PhD, Lesley Wong, MD, Theodore M. Cole III, PhD, Joan T. Richtsmeier, PhD, Jenner Manson, John A. Bley, Jr., DVM, Dawn Yules, MD, Paul N. Manson, MD (Washington, DC)

The purpose of this study was to determine if rigid skeletal fixation impedes subsequent growth in the maturing non-human primate calvaria, and if so, are these effects seen in animals undergoing fronto-orbital advancement.

A supraorbital bar and frontal bone flap were fashioned during the phase of rapid skull growth. Thirty-seven immature non-human primates (*Macaca fasicularis*) were randomly divided into five treatment groups: 1) osteotomy and rigid fixation; 2) osteotomy and wire fixation; 3) osteotomy, supraorbital bar, and forehead advancement with rigid fixation; 4) osteotomy, supraorbital bar, and forehead advancement with wire fixation; 5) control group.

Fifty three-dimensional landmarks describing anatomically relevant loci on the craniofacial complex were collected. Morphometric comparisons suggested that crania from all four treatment groups differ from normal in their proportions. Form changes were most extensive for Group 4, where many of the dimensions of the orbits, midface, and anterior cranial base were markedly reduced. The greatest reductions were concentrated on the facial and orbital components of the zygomatic bone. The remaining groups showed patterns of form difference that were similar to Group 4, in that the dimensions of the zygomatic bones were reduced; however, the changes in Groups 1, 2, and 3 were not as extensive as for Group 4 (i.e., the anterior basicranium appeared relatively unaffected in these groups. In all four operative groups, the proportions of the neurocranium appeared relatively unchanged.

In conclusion, skeletal fixation resulted in specific alterations of the facial and orbital components of the zygomatic bone and skull. The zygomatic bones were smaller, more significant with wire fixation than rigid fixation, reducing the height of the lateral orbit and cheek. These differences were noted in the advancement groups as well, again more significant with wire than with rigid fixation. While these differences may be a function of the procedure, rigid fixation appeared to overcome the limitations of technique. Skeletal fixation did not significantly impede the normal growth of the neurocranium.

22. A Combined Fronto-Orbital and Occipital Advancement Technique for Use in Total Calvarial Reconstruction

Ian F. Pollack, MD, H. Wolfgang Losken, MD, Dennis J. Hurwitz, MD (Pittsburgh, PA)

The management of infants with syndromic bilateral coronal synostosis and resultant brachyturricephaly poses a significant therapeutic challenge. The application of total calvarial reconstruction to the treatment of this problem has represented a major recent innovation that has substantially improved the cosmetic results in this patient population. However, rigid fixation of the reconstructed calvarium is often required in order to maintain the correction achieved and to provide protection for the underlying brain. The requirement for extensive fixation constitutes a significant disadvantage for the use of this procedure in infants and young children. During the past three years, we have developed an approach to the treatment of this problem that incorporates a series of tongue-in-groove osteotomies to provide increased stability to advancements of both the frontal and occipital regions in conjunction with cranial height reduction, while minimizing the need for metallic fixation. With this approach, the reconstructed skull is sturdy enough to resist the compressive force applied by the weight of the child's head immediately after surgery, but retains the ability to progressively expand. The cosmetic results have been extremely gratifying in the seven patients treated according to this technique with immediate normalization of the vertical and horizontal cephalic indices; two of these patients had undergone prior anterior reconstructions. Median blood loss for the operation was 450 ml, approximately two to three times the typical blood loss in an anterior calvarial procedure. Operating time ranged from 420 to 580 minutes (median: 475 minutes), approximately twice the length of an average anterior calvarial reconstruction. The median hospital stay was seven days. No morbidity or mortality has been encountered. At a median follow-up of one year, each child has maintained a good to excellent cosmetic result and has demonstrated progressive calvarial expansion along normal percentiles without recurrence of the brachyturricephalic head shape. Operative indications and technical caveats to this approach will be discussed.

23. The Effect of Early Craniocervical Decompression on Functional Outcome in Neonates and Young Infants with Symptomatic Chiari Malformations: Results from a Prospective Series

lan F. Pollack, MD, Dorinne Kinnunen, BS, A. Leland Albright, MD (Pittsburgh, PA)

The indications for hindbrain decompression in neonates and young infants with spinal dysraphism who exhibit brainstem dysfunction in association with a Chiari malformation have remained controversial. This largely reflects the fact that the postoperative outcome in such patients has often been poor, which has supported the belief that much of the brainstem compromise in these patients is congenital and inherently irreversible. However, in a previous retrospective review of our operative results between 1975 and 1989, we noted that the brainstem dysfunction in these children was often an acquired phenomenon that was reversible with prompt operative intervention. Accordingly, we hypothesized that with early craniocervical decompression, an excellent outcome could be achieved in most neonates and young infants with symptomatic Chiari malformations. Based on this premise, we prospectively treated all patients since 1989 with urgent decompression after other causes for brainstem dysfunction had been ruled out. All children underwent a limited suboccipital craniectomy, cervical laminectomy extending beneath the inferior extent of the cerebellar tissue. and dural decompression. The outcome in these patients has been favorable in comparison to previous studies. Ten of the 13 children treated according to this protocol recovered normal or nearly normal brainstem function shortly after decompression; one child had persistent unilateral lower cranial nerve paresis. None of these children required a tracheostomy for ventilatory support and only one required a temporary gastrostomy. The other three children all exhibited bilateral vocal cord paralysis and severe apnea by the time of decompression and failed to have any meaningful recovery of function. We conclude that early recognition of the symptoms of brainstem compromise in neonates and young infants with spinal dysraphism coupled with urgent evaluation and decompression are effective in producing prompt resolution of the brainstem dysfunction in most affected patients. Conversely, the prognosis for recovery is poor in children with bilateral vocal cord paralysis by the time of decompression.

24. Bone Regrowth and Recurrence of Symptoms Following Decompression in the Infant with Arnold Chiari II Malformation

Roger J. Hudgins, MD, William R. Boydston, MD, PhD (Atlanta, GA)

The Chiari malformation is the leading cause of death in infants with myelodysplasia. While controversial, early operative decompression may halt or reverse the progression of symptoms. If symptoms recur after initial improvement, attention is directed to assuring shunt patency and the absence of hydromyelia and not to reassessment of the area of bone decompression. Three infants are presented who initially improved after Chiari decompression only to have recurrence of symptoms several months after surgery. In each case, regrowth of bone at the site of previous decompression was documented radiographically and at surgery. In one case, regrowth of bone occurred twice. Each child stabilized or improved following further decompressive surgery. A theory of bone regrowth and a possible means of prevention are discussed. If recurrence of Chiari symptoms occurs after decompressive surgery in infants, thin section CT with done windows through the operative site should be performed to exclude bone regrowth and recurrent compression.

25. Impact of Urodynamic Evaluation on the Surgical Management of Spinal Cord Tethering

O. Vernet, J. P. Farmer, A. M. Houle, J. L. Montes (Montreal, PQ, Canada)

To determine the usefulness of urodynamic studies in the management of children with suspected tethered cord, we retrospectively reviewed case records of 25 patients evaluated both pre- and postoperatively with this diagnostic adjunct. All patients were also evaluated with MRI or myelogram-CT. Seven patients underwent primary cord untethering whereas 18 patients with prior myelomeningocele closure underwent secondary untethering. The primary surgical indication was urological (11 cases), neurological (7 cases), both urological and neurological (4 cases), or orthopedic (3 cases) deterioration. All patients were operated on with microsurgical techniques and intraoperative nerve roots stimulation. At a mean followup of 2 years, 6 out of 11 patients with urological deterioration improved in terms of social continence, 2 stabilized and 3 worsened. The 7 patients with neurological deterioration improved. The 4 patients with both urologic and neurological presentation improved except 2 who deteriorated urologically. The 3 patients with orthopedic deformities remained stable. With respect to urodynamic investigation, and there was significant increase in the total bladder and "20 below" capacities postuntethering whereas the increase in "30 below" capacity did not reach statistical significance. We conclude that urodynamic studies are useful both diagnostically and in the follow-up of patients with tethered cord, that urodynamic disturbances often precede clinical deterioration, and that untethering of the cord influences favorably the urodynamic status in most patients.

26. Chick Hindbrain Malformation Induced by Decompression of the Mesencephalic Vesicle

S. Robinson, R. G. Higbee, P. A. Knepper, D. G. McLone (Chicago, IL)

A better understanding of the pathogenesis of the hindbrain anomaly of the Chiari II malformation may lead to improved management of these patients. Normal hindbrain development results from a complex interaction of the neuroectoderm, mesoderm, and the primitive ventricular system. During normal vertebrate development after the neural tube closes, the neurocele (the primitive central canal of the spinal cord) occludes. Neurocele occlusion distends the primitive ventricular system during the period of rapid brain enlargement.

We hypothesized that disruption of adequate distention of the primitive ventricular system during the period of rapid brain enlargement would hinder normal development of the hindbrain. In this study, the mesencephalic vesicle of stage 26 (incubation day 5) chick embryos was decompressed by puncture or puncture and aspiration of cerebrospinal fluid. The embryos were processed for histological study on incubation day 16, and the midsagittal section of each embryo hindbrain was analyzed morphologically. Criteria developed from control embryos were used to classify the embryos with a defective hindbrain. A consistent hindbrain defect was induced in 52 percent of the experimental embryos. When compared to controls, the defective hindbrain was characterized by: a smaller cerebellar width, a smaller proportion of the posterior fossa occupied by the cerebellum, a narrower basal angle (all p<.001), and a wider cerebellar angle (p<.01). The defective hindbrain also showed cerebellar dysplasia.

The results demonstrate that a lack of adequate primitive ventricular distention during rapid brain enlargement can alter hindbrain formation. These findings are consistent with the theory that the hindbrain malformation results in part from inadequate distention of the primitive ventricular system due to delayed or defective neurocele occlusion.

27. A Molecular Model of Secondary Neurulation Defects

Timothy M. George, MD, Michael McLone, Paul A. Knepper, MD, PhD, David G. McLone, MD, PhD (Chicago, IL)

Secondary neurulation in humans is responsible for the formation of the conus medullaris and the filum terminale. Abnormalities in human secondary neurulation may be responsible for the formation of a tethering lesion such as the fatty or thickened filum terminale. The events leading to the formation of a normal secondary neural tube are believed to be governed by early regulating genes. In this study, we show that antisense oligonucleotide treatment to the early regulating gene, Pax-3, given during several phases of chick secondary neurulation, caused tail defects and hindlimb anomalies. The degree of the defect mirrored the timing of oligonucleotide administration, with the more extensive defects occurring earlier in the treatment course. At the cellular level, secondary neurulation defects revealed a lack of transition of primitive tailbud mesenchymal elements to neuroepithelial characteristics. The findings in this study suggest that 1) molecular mechanisms involved in secondary neurulation can be studied in isolation from primary neurulation; and 2) secondary neural tube defects can be generated by the use of antisense oligonucleotide technology.

28. A New Model for Understanding Encephalocele Pathogenesis

Ty J. Gluckman, BA, Timothy M. George, MD, Paul A. Knepper, MD, PhD, David G. McLone, MD, PhD (Chicago, IL)

The pathogenesis of encephaloceles remains unclear. Current theories on the etiology of encephaloceles suggest either a defect of cranial neural tube development or a defect in mesodermal development overlying the rapidly growing embryonic brain. Experimental evidence using mammalian models treated with a variety of teratogens supports the latter theory. Encephaloceles, therefore, are postulated to result from herniation of rapidly growing brain through defective mesoderm.

In order to determine the etiology of encephaloceles, we utilized a modification of Auerbach's method as an *in vitro* model system of chick embryos, to examine the period of rapid brain growth (days 3-5 of incubation). Surgical lesions in the primitive cranium over the mesencephalic vesicle, with or without decompression of the brain, were performed. Control embryos were either not manipulated or had sham procedures. As such, we sought to determine if disruption of mesoderm and/or brain decompression during the period of rapid brain growth would generate encephaloceles.

The results of the study revealed that lesioned embryos treated at day 5 that had evidence of marked decompression showed the presence of encephaloceles in 21 (60%) of the 35 embryos in the treatment groups. However, unmanipulated embryos or embryos lesioned without decompression revealed no signs of encephalocele (0/105) during treatment days 3-5 of incubation. Decompressed embryos treated at days 3-4 showed no evidence of encephaloceles (0/37).

Our findings suggest that there is a critical time point when a disturbance of the tissues overlying the rapidly growing brain, associated with brain decompression, leads to the formation of an encephalocele. Future investigations are aimed at evaluating the developmental changes in the mesoderm, surface ectoderm, and neural tissue during this critical time period.

29. Language Localization in the Dominant Hemisphere of Children

Mitchel S. Berger, MD, George A. Ojemann, MD (Seattle, WA)

We have reviewed our experience gained over the past several years with language mapping in the pediatric population undergoing surgery for epilepsy with or without an associated tumor. This data will be described in terms of naming, reading and speech localization in the pediatric brain, along with the methodology to achieve this at all ages.

There were 28 patients who underwent detailed language mapping of the dominant hemisphere. Nearly half of these children had an intrinsic tumor involving the temporal lobe. Ages ranged from 3 to 14 years and adolescents above this age range were excluded from analysis: < 10 years, n = 14; 10-12 years, n = 6; 13-14 years, n = 8. Eleven children were 7 years of age or younger. All but 5 patients had language mapping determined via subdural grid stimulation. Electrocorticography (ECoG) was used to set the stimulation current for language mapping following identification of after-discharge potentials. A series of slides with common objects and brief written leader phrases were alternated with and without stimulation, following determination of the baseline naming error rate. Broca's area was localized to areas associated with counting arrest. Cortical and subcortical mapping was used to localize sensory and motor pathways when needed.

Preoperative WADA testing demonstrated a strong correlation between left-handedness and right-sided or bilateral hemisphere, i.e., atypical, speech, and right hemiparesis, acquired before the age of 3 years, and atypical speech. Language mapping was accomplished in nearly all cases with a stimulation current that ranged between 4-6 milliamps (60 Hz, 1 msec). Most children had more than one cortical site essential for language, although not always within the same lobe. Each site was tightly localized to a cortical area no greater than 5 to 8 mm. Counting arrest was always seen in a cortical region contiguous with face motor cortex. Permanent language deficits did not occur if the resection did not come within 10 mm of an essential cortical site.

Language mapping of young children may be reliably and safely accomplished with stimulation coupled with ECoG techniques, and object naming plus counting paradigms. Composite hemispheric language maps will be shown along with describing the methodology to perform this type of functional mapping during epilepsy and/or tumor surgery in the pediatric population.

30. Can Epileptic Foci Be Suppressed with Electric Fields?

Steven J. Schiff, MD, PhD (Washington, DC)

The functional complications of resective surgery for intractable epilepsy can be significant. We ask whether epileptic foci can be paced with electrical stimulation, similar to arrhythmic cardiac tissue. In addition, we seek to demonstrate the feasibility of noninvasively pacing epileptic tissue using electric fields.

Hippocampal tissue slices are prepared from 125-150 gm Sprague Dawley rats, and perfused at 35°C in a perfusion chamber. In the presence of moderately elevated potassium (8.5 mM) such temporal lobe tissue is spontaneously active, generating epileptic spikes in CA3 that drive intermittent seizure-like events in CA1. Stimuli are delivered either directly into the tissue with a tungsten microelectrode, or through a dipole electric field delivered to the perfusion chamber with a submerged subdural electrode strip (PMT Corporation). Electric field pulses are delivered in 0.15-1.0 msec square wave pulses, with field strengths of up to 100 mV/mm.

We have demonstrated recently that such tissue can be paced with direct injection of current into the tissue, and that there exists a frequency band between 1.0-1.3 Hz which suppressed seizure generation in CA1 (7. Neurophysiol 73: 876-879, 1995). In this series of experiments, we show that electric field pulses can similarly entrain such tissue, and can replicate the seizure suppressive effects near 1 Hz. There is a clear dose response curve for the entrainment threshold, with a multiplicative relationship between pulse duration and field amplitude necessary for entrainment.

These observations offer the intriguing prospect of a novel technology that may be rapidly introduced into clinical trials to test feasibility in human epilepsy.

Supported by NIH 1R29-MH50006-03, and ONR #N00014-95-1-013.

31. Hippocampal Activation Stops the Spread of Experimental Limbic Seizures

Mark R. Proctor, Rebecca Vaurio, Chris Hogan, Karen Gale (Washington, DC)

Although extirpation of the hippocampus is performed for the treatment of temporal lobe epilepsy, there is little information concerning the functional role of the hippocampus in modulating limbic seizures *in vivo*. This study explores the role of the hippocampus using rat models of limbic motor seizures analogous to human complex partial seizures. Lesions and drug micro-injections were used to modify hippocampal function.

Animals with excitotoxic lesions of the hippocampus appeared neurologically normal, but when challenged with the GABA, antagonist bicuculline, either focally administered in the prepiriform cortex (90 ng) or systemically administered (0.3-0.4 mg/kg IV), they exhibited more severe seizures than sham operated controls. Similarly, hippocampal inactivation by focal infusion of the GABA, agonist, muscimol, resulted in significant potentiation of limbic seizures. Conversely, hippocampal activation by focal injection of bicuculline (120 ng) or the glutamate agonist, kainic acid (234 pmol), evoked wet dog shakes and hippocampal seizures (confirmed by implanted depth electrodes) but attenuated limbic motor seizures induced either focally from prepiriform cortex or by systemic bicuculline.

These results suggest the novel concept that the intact hippocampus serves to regulate, not generate, seizures. Activation of intrinsic hippocampal circuitry, even to the point of local seizure discharge, can 'protect' the larger limbic network from generating seizure activity. It is therefore possible that the relatively late maturation of hippocampal circuitry during postnatal development contributes to enhanced seizure susceptibility during infancy. Moreover, children may benefit from surgery which spares the hippocampus if performed prior to the onset of seizure-induced hippocampal neuronal reorganization.

Sponsored by the Epilepsy Foundation of America with support from McNeil Pharmaceuticals and by HHS Grant NS 28130.

32. Subtemporal Transparahippocampal Amygdalohippocampectomy for Treatment of Mesial Temporal Lobe Epilepsy: A Novel Operation

T. S. Park, MD, Blaise F. D. Bourgeois, MD, Bruce A. Kaufman, MD (St. Louis, MO)

Introduction: Amygdalohippocampectomy (AH) is widely employed for intractable mesial temporal lobe epilepsy. To date, the main surgical approaches to the mesial temporal structures have been via the sylvian fissure and the superior or middle temporal gyrus. Other AH procedures require division of the temporal stem; and when performed in the dominant temporal lobe, speech disturbance may follow. Also, the transsylvian AH permits only a limited cortical incision which makes the operation technically difficult. We have improved the technique for AH by utilizing a subtemporal transparahippocampal approach to the hippocampus and amygdala.

Results: Six patients (5 children and 1 adult) underwent the operation at our pediatric epilepsy center. Under general anesthesia, the amygdala and anterior 2-3 cm of hippocampus were exposed and eventually resected through a subpial removal of the adjacent parahippocampal gyrus. The uncus was also removed. The fusiform gyrus was left unresected. Five patients underwent the operation in the dominant temporal lobe. In a follow-up (1 to 9 months), five patients were seizure-free, and one had >90% reduction of seizures. Postoperative morbidities were contralateral homonymous quadrantanopsia in two patients and memory decline in one patient. One patient awaits a postoperative visual field test. No patient experienced postoperative dysphasia.

Conclusions: This novel AH technique appears to offer an excellent alternative and several advantages over other AH techniques. Besides that the temporal stem is not divided in this procedure, the access to the hippocampus is not limited, and postoperative dysphasia can be avoided. This procedure may benefit particularly children since it can be performed without language mapping and Wada test.

33. Corpus Callosotomy in Lennox-Gastaut Syndrome

Jeffrey P. Blount, MD, Bernard Maister, MD, Robert E. Maxwell, MD, PhD (Minneapolis, MN)

Introduction: The Lennox-Gastaut syndrome is characterized by a range of intractable seizure types and a characteristic EEG pattern and accounts for 2%-3% of new cases of pediatric epilepsy. Anterior corpus callosum section has been advocated as a useful procedure in the treatment of this notoriously refractile syndrome. We report our experience with a large group of patients with Lennox-Gastaut syndrome who have undergone corpus callosotomy.

Methods: The medical records of 41 patients who underwent corpus callosotomy at less than 12 years of age at the University of Minnesota/MINCEP between 1988 and 1993 were retrospectively reviewed. A subset of 18 patients were diagnosed with the Lennox-Gastaut syndrome and ≥12 month follow-up was available for 14 patients who form the focus of this report.

Data: Fourteen patients (9 male, 5 female) with Lennox-Gastaut syndrome were reviewed. Average age of seizure onset was 1.8 years (range 3 months-6 years). Average age at surgery was 7.2 years (range 3.2-11.6 years). Two-thirds callosal section was performed in 6 patients and complete callosal section performed in 8. The most common seizure types preoperatively were atonic/akinetic (9 patients), atypical absence (8 patients), generalized tonic-clonic (7 patients) and tonic (6 patients). There were no perioperative complications. Postoperatively, one patient stopped having seizures and three ceased having drop attacks entirely. Atonic/akinetic seizures demonstrated the greatest (80%) reduction in frequency although generalized tonic-clonic and atypical absence also improved.

Conclusions: Corpus callosum section is an effective technique for reducing the frequency and severity of drop attacks in the Lennox-Gastaut syndrome. EEG patterns associated with favorable outcomes will be reviewed.

Research was supported in part by NIH-NINDS Grant P50 NS16308 and the Peyton Society.

34. Ablative Epilepsy Surgery in Children

Mark Lee, Joseph R. Smith, David J. Yeh, Yong D. Park (Augusta, GA)

Ablative epilepsy surgery is increasingly becoming an accepted treatment in children. Thus, we find it timely to review our experience and analyze for factors associated with seizure-free outcome. Sixty children (age range at surgery, 1.5 to 18 years) underwent ablative epilepsy surgery at our institution between 1981 and 1993. Seizure outcome was reviewed after at least one year follow-up for all patients. Seizure outcome was graded as seizurefree, rare seizure (1-3 per year), >90% seizure reduction, and <90% seizure reduction. Patients were analyzed in 2 groups: those undergoing anteromesial temporal (AT) resections and those undergoing extratemporal (XT) resections. Patients were further analyzed for age at surgery and pathology. Thirty-four children underwent AT resections. There were no surgical complications among these patients. Twenty-four of these children are seizure-free (71%), 3 children have rare seizures, 3 children have >90% seizure reduction and 4 children have <90% seizure reduction. Children ≤12 years had a better outcome than children >12 (9/11 seizure-free (82%) versus 15/23 (65%), p<0.05). There was no difference in outcome between AT lesional (10/13 seizure-free, 77%) and nonlesional patients (14/21, 67%). Twenty-six children underwent XT resections. There were 2 transient neurological deficits in these patients. Sixteen of these children are seizure-free (61%), 3 children have >90% seizure reduction and 7 children have <90% seizure reduction. Children undergoing XT resections with lesions had a better outcome than children without lesions (14/18 seizure-free (78%) versus 1/8 seizure-free (13%), p<0.05). There was no difference in outcome between children ≤12 years (7/12 seizure-free, 58%) and those >12 years (9/14 seizure-free, 64%). In conclusion, 71% of our AT patients and 61% of our XT patients are seizure-free after at least 1 year follow-up. Younger children (≤12 years) have a better seizure outcome among children undergoing anteromesial temporal resections. Children with lesions have a better seizure outcome among children undergoing extratemporal resections.

35. Results and Complications of Reoperation for Failed Epilepsy Surgery in Children

Ellen G. Shaver, MD, Glenn Morrison, MD, Antonio R. Prats, MD, A. Simon Harvey, MD, Patricia Dean, RN (Philadelphia, PA)

The outcome and complications in children who undergo reoperation for failed epilepsy surgery have not been well documented. This retrospective study evaluated 20 children who underwent a second resective surgery for recurrent seizures. The mean age at first operation was 8 years. The mean seizure-free interval after the first procedure was 3 months.

The mean interval between surgeries was 2.5 years, and the mean age at reoperation was 11 years. Four categories of patients were identified: a) extension of the initial resection was performed in 8 patients; b) 5 patients underwent lobectomy or corticectomy in a region remote from the original surgical site; c) multilobar resection which may have included further resection of the initial procedure was accomplished in 4 patients; d) hemispherectomy was performed in 3 patients.

Seizure-free outcome in 16 patients who were more than one year since last surgery was 56%. Patients with reoperation in the same lobe as the first procedure (group a) had a 71% seizure-free rate, while only 33% of patients in groups b and c were free from seizures at follow-up evaluation. Significant unexpected neurological deficits occurred in three patients who underwent multilobar resection at reoperation. The complications are discussed in detail.

Reoperation for intractable partial epilepsy is beneficial in selected children. Factors which may contribute to a seizure-free outcome include an epileptogenic focus found in the same lobe as the original focus or limited to the temporal lobe, or an extratemporal focus anatomically related to a structural lesion. Patients who require multilobar resections may have higher risk of postoperative neurological deficit than those patients with reoperation in one lobe. These factors may be useful in counselling parents of children considering reoperation for recurrent epilepsy.

36. Seizure Outcome in Children with Arteriovenous Malformations Treated with Gamma Knife Radiosurgery

Peter C. Gerszten, MD, P. David Adelson, MD, Douglas Kondziolka, MD, John C. Flickinger, MD, L. Dade Lunsford, MD (Pittsburgh, PA)

In children, seizures are the second most common presentation of arteriovenous malformations (AVMs). Stereotactic radiosurgery has been advocated as a safe and effective alternative treatment of these lesions, but seizure control after radiosurgery for AVMs has not been specifically addressed in children. Between 1986 and 1994, 72 children under the age of 18 years were treated with Gamma knife radiosurgery for AVMs at our institution. Fifteen patients (21%) had seizures as part of their clinical course. Ages varied from 2 to 17 years (median 16 years). Seizure types included: generalized tonic-clonic (8), focal motor or sensory (4); partial complex (2); and mixed (1). Spetzler-Martin grades included: II (7); III (4); IV (2); VI (2). Following radiosurgical treatment, imaging revealed complete obliteration in 6, significant reduction in 8, and no response in 1 patient (mean follow-up 47 months).

Following radiosurgery, 11 of 13 patients (85%) were seizure free and off anticonvulsant therapy. Two patients had >90% improvement in their seizures but continue on medication. Two of the 72 patients (3%) developed seizures after radiosurgery and remain on medication. Seizure outcome was not associated with location, hemorrhagic presentation, or complete obliteration of the lesion.

The etiology for the antiepileptic effect of radiosurgery for AVMs is unknown. Different theories include the elimination of the steal phenomenon, specificity of effect on epileptic neurons, and effects on the neuronal cell membrane. Our experience in children has found that stereotactic radiosurgery, as a noninvasive alternative to microsurgery, is associated with a good outcome for not only the anatomic abnormality (the AVM), but the functional disorder (seizures) as well.

37. Presence of Immunohistochemically Identifiable Tissue Plasminogen Activator in Cavernous Angiomata: A Potential Mechanism for Re-Hemorrhage and Lesion Growth

David M. Frim, Natasa Zec, Jeffrey Golden, R. Michael Scott (Boston, MA)

The mechanisms of growth and re-hemorrhage of cavernous angiomata of the brain remain unknown. One possible mechanism of lesion growth is repetitive re-hemorrhage, in-growth of neovascularity during clot organization, and maturation of new vessels into a larger cavernous angioma. A histopathological similarity has been noted between the organizing clot surrounding hemorrhagic cavernous malformations and the organizing phase of the membranes surrounding chronic subdural hematoma. A thrombolytic process intrinsic to the membranes surrounding chronic subdural hematoma has been localized to immunocytochemically identifiable tissue plasminogen activator (TPA) located in the vascular endothelium of the membrane neovasculature. By analogy, we sought to identify TPA in the organizing tissues surrounding hemorrhagic cavernous angiomata.

Seven cavernous malformations, surgically removed and pathologically confirmed by standard staining techniques, were immunohistochemically stained for TPA. Five of the 7 lesions (71%) contained vascular endothelial cells which stained for TPA. The 2 lesions which did not contain TPA were associated with well organized clot cavities. There was a trend toward the most abundant TPA containing endothelium to be found in lesions associated with clots in an intermediate stage of organization. This finding may represent a local thrombolytic process responsible for the frequent hemorrhagic nature of cavernous angiomata. Alternatively, since local elaboration of TPA is common to both chronic subdural membranes and the clot cavity surrounding cavernous angiomata, this finding may represent a more global response of cerebral tissues to hemorrhage and clot reabsorbtion.

38. Dysregulation of Gene Expression in the Vasculature of Sturge-Weber Syndrome: Clues to the Pathogenesis of the Disease

R. L. P. Rhoten, MD, Y. G. Comair, MD, D. Shedid, MS, P. E. DiCorleto, PhD, C. De la Motte, PhD, M. S. Simonson, BS (Cleveland, OH)

Relatively little is known about the pathogenesis and subsequent biologic behavior of Sturge-Weber syndrome (SWS), a congenital, neurocutaneous syndrome characterized by leptomeningeal-based cerebrovascular lesions and facial nevi. We have investigated the expression of genes that regulate the development and phenotype of the cerebrovascular endothelium in four pediatric patients who underwent modified functional hemispherectomy for medically intractable epilepsy from SWS. Immunohistochemistry of the cerebral vasculature from the surgical specimens demonstrates markedly increased expression of laminin, fibronectin, and intercellular adhesion molecule (ICAM) compared to the vasculature of the normal human control. The immunoreactivity of the SWS vascular lesion endothelial cells (ECs) to proliferating cellular nuclear antigen (PCNA) and vascular endothelial cell growth factor (VEGF) was diminished compared to the controls. Cultured SWS sample endothelial cell strains from the surgical specimens revealed increased levels of vascular cell adhesion molecule (VCAM) and E-selectin (ELAM) compared to normal control endothelial cells from human umbilical vein and aorta. Expression of the potent endotheliumderived vasoconstrictor and mitogenic peptide, endothelin-1 (ET-1) was markedly elevated in the SWS lesions compared to the normal controls. ET-1 immunoreactivity was particularly high in the pial lesions, as well as in the underlying brain vasculature. Analyses of steady state mRNA by reverse transcription and the polymerase chain reaction (RT-PCR) revealed increased levels (two-fold) of preproET-1 mRNA transcripts in SWS vessels compared to normal human controls.

Taken together, these results in SWS lesions demonstrate dysregulation of genes that control endothelial cell phenotype and show an alteration in the endothelial cell genetic program. Further study of these candidate genes should yield insights into molecular mechanisms leading to the formation of the cerebrovascular lesions in these patients.

39. The Management of Giant Aneurysms in Children and Adolescents

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

Although only 1% to 2% of all aneurysms occur in children, giant aneurysms have been documented to occur more frequently than in adults and have a male preponderance. We have reviewed our series of pediatric giant aneurysms and have defined 11 cases that presented over the past 7 years that were considered to be giant in nature.

There were seven male and four female patients with a mean age of 12 years (3 months to 17 years). Three cases were traumatic in origin, four presented with acute rupture, one presented with headache and two with progressive cranial nerve palsies. One was found incidentally. All aneurysms were considered to be giant. Surgical treatment included endovascular coiling with subsequent trapping via a combined retrolabyrinthine-extreme lateral infratemporal transcondylar extended approach with staged clip placement in one, trapping with STA-MCA bypass in one, trapping with C₁-C₃ carotid bypass in one, hypothermic cardiac arrest in one, petrous carotid to intracavernous trapping in one and clip ligation in six. Eight patients had no change in their neurologic state while three developed new deficits postoperatively. Complications included bone flap infection, sepsis, and clip slippage in individual patients. Three patients had associated vasospasm.

We conclude that significant advances in operative approaches involving the skull base, refinements in endovascular techniques, and hypothermic arrest have allowed for the management of giant complex vascular lesions in children and adolescents with a reduced morbidity.

40. Extent of Variation Between Centers in Electrophysiologic Techniques Used in Lumbosacral Selective Posterior Rhizotomy for Spastic Cerebral Palsy

Paul Steinbok, MBBS, FRCSC, John Kestle, BSc, MD, MSc, FRCSC (Vancouver, BC, Canada)

The extent of variation between centers in the electrophysiologic techniques used in lumbosacral selective posterior rhizotomy (SPR) for spastic cerebral palsy was studied using a questionnaire survey.

Thirteen centers completed the questionnaire, and the responses were analyzed for those 11 centers in which the extent of posterior root section was guided by intraoperative electrophysiologic responses.

Consistent techniques included: use of unipolar stimulating electrodes (10/ 11): stimulation < 4 cm from the root exit foramen (9/11); separation of posterior roots into three to five rootlets each (9/11); tetanic stimulation frequency of 50 Hz (8/11); tetanic stimulation at 100% of threshold (8/11); recording from multiple lower limb muscles simultaneously (11/11); and using contralateral spread of the response as a major criterion of electrophysiologic abnormality (10/11). There was more variation (8/11 concurrence) with respect to the use of a constant current versus constant voltage stimulator; the location of the cathode of the stimulating electrode relative to the anode; the definition of the threshold for a response; whether threshold was determined from stimulation of a posterior root or individual rootlets; the type of recording electrodes (needle versus surface); and the relative importance of electrophysiologic versus clinical findings in determining how much of each posterior root to cut. In five centers, SPR would proceed without the benefit of electrophysiologic guidance if the equipment should fail intraoperatively, and this had happened in three.

The results indicate significant variation in critical aspects of electrophysiologically guided SPR, calling into question the value of the electrophysiologic guidance in SPR.

41. Electromyographic Monitoring of Cranial Nerves During Resection of Fourth Ventricular Tumors

Paul A. Grabb, MD, A. Leland Albright, MD, Robert J. Sclabassi, MD, PhD (Birmingham, AL)

We reviewed the results of intraoperative electromyography (EMG) of the lateral rectus (LR) and facial muscles in 17 children whose preoperative scans showed compression or infiltration of the fourth ventricular floor by tumor to determine how LR and facial EMG correlated with postoperative sixth (VI) and seventh (VII) nerve morbidity. Seventy muscles were monitored: bilateral LR (VI) and facial (VII) muscles in all children and bilateral cricopharynegeus (X) in one child.

Of the 70 monitored muscles, 10 new cranial neuropathies occurred in 7 children (VI, n=4; VII, n=5; X, n=1). Of these 10 new cranial neuropathies, 6 had correlative intraoperative EMG activity in 1 of 4 VI palsies, 4 of 5 VII palsies and 1 of 1 X palsies. Four children with new neuropathies had no correlative EMG activity. Of 60 monitored cranial nerves that remained clinically unchanged, 48 had no EMG activity. Twelve cranial nerves (VI, n=3; VII, n=9) had EMG activity but no deficit. All LR EMG activity (n=4) had concomitant facial nerve EMG activity (n=1), facial paresis (n=1), or both (n=2). In one child intraoperative EMG monitoring assisted in preserving the extrapontine facial nerve embedded in ependymoma. Facial EMG activity reduced the aggressiveness of tumor removal from the fourth ventricular floor.

Facial paresis was most strongly predicted by LR nerve EMG activity. LR EMG activity did not correlate with abducens paresis. Facial EMG activity did not correlate strongly with facial paresis. The relatively low incidence of facial nerve paresis (n=5) compared to the incidence of facial EMG activity (n=13) may relate to the reduction in surgical aggressiveness when facial EMG activity was surgically induced. While LR nerve EMG activity was associated with postoperative facial paresis it provided no useful intraoperative information. Facial EMG monitoring was useful for safely resecting a tumor that surrounded the extrapontine facial nerve and likely served as a warning to the surgeon that the floor of the fourth ventricle was being manipulated, and may thereby have reduced the risk of cranial neuropathies.

42. Complications Associated with an Interhemispheric Operative Approach

Grant Shumaker, MD, Michael L. Levy, MD, Albert Penney, BS, J. Gordon McComb, MD (Los Angeles, CA)

The interhemispheric operative approach is used to reach midline and paramedian lesions. This study reviews the complications of a consecutive series of 85 pediatric patients from 1986 through 1995 operated on via an intrahemispheric corridor with the patient placed in the lateral decubitus position. The mean age of the patient population was 7½ years (range 1 month to 19 years) and consisted of 45 males and 40 females. Minimal hemispheric retraction pressure was needed through the use of gravity and CSF aspiration.

Postoperative complications included transient hemiparesis in 24, seizures within the first twenty-four hours of surgery in 10, transient mutism in 2, and abnormalities of fluid and electrolyte balance in 15. In all patients, the hemiparesis resolved and none had a permanent deficit. No patient went on to have a chronic seizure disorder and the mutism resolved in the two patients with this problem.

We also examined the effect of sacrifice of superficial or deep intracerebral veins on outcome. No patient showed evidence of cortical hemorrhage associated with the sacrifice of bridging veins or hemispheric retraction. Four patients did develop a delayed paresis which subsequently resolved.

We conclude that the interhemispheric approach with the patient in a lateral decubitus position provides a safe and direct access to midline lesions in a pediatric population with the main complication being a moderate incidence of transient hemiparesis.

43. Intraparenchymal Lesions in Patients with Leukemia or Lymphoma

Felipe C. Albuquerque, MD, Marvin Nelson, MD, Corey Raffel, MD, PhD (Los Angeles, CA)

Pediatric patients with leukemia and extraneural lymphoma are at risk for developing a variety of intracranial lesions. Metastases of their original tumor, primary brain tumors, and opportunistic infections may be encountered. At Children's Hospital of Los Angeles, an average of 70 patients per year are diagnosed with leukemia/lymphoma. We have identified 16 leukemia/lymphoma patients, compiled over 13 years, with radiographic evidence of intraparenchymal brain disease. Twelve patients had acute lymphocytic leukemia, 2 had non-Hodgkin's lymphoma, 1 had acute nonlymphocytic leukemia, and 1 had acute myelogenous leukemia. Of this group, 7 had brain metastases, 5 had intraparenchymal infections, and 3 had primary brain tumors. Biopsy of the remaining patient's lesion revealed focal gliosis. Patients with metastases presented an average of 17 months after diagnosis of leukemia/lymphoma. In contrast, patients with primary tumors or infections presented at 68 and 71 months, respectively. Diagnosis was obtained by biopsy in 4 patients, lumbar puncture (LP) in 4, craniotomy in 3, autopsy in 3, cranial sinus exoneration in 1, and opthalmologic examination in 1. Pediatric patients with leukemia/lymphoma presenting early in their course with an intraparenchymal lesion most likely have a metastasis. Diagnosis can be established by LP, or in those with a negative LP, by stereotaxic biopsy. Patients presenting later most likely have an infection or a new, primary neoplasm. If LP fails to yield a diagnosis, a protocol of opthalmologic examination followed by stereotaxic biopsy or craniotomy should be employed.

44. Surgical Management of Pediatric Thalamic Tumors: 10 Years of Experience

Jessie C. Huang, MD, Jeffrey H. Wisoff, MD, Fred J. Epstein, MD, Jeffrey C. Allen, MD (New York, NY)

The authors reviewed 51 pediatric patients with thalamic tumors who were treated at the Divisions of Pediatric Neurosurgery and Pediatric Neurooncology at New York University Medical Center from 1985 to June 1995. There were 31 males and 20 females. The age of diagnosis ranged from 6 months to 20.6 years with a mean age of 9.3 years. The most common presenting symptoms were hemiparesis (51%), headache (45%), vomiting (37%), lethargy (22%), and ataxia (22%). Forty-six patients underwent surgery. Seven (15%) had gross total resection, 16 (35%) had subtotal resection, 8 (16%) had partial resection, and 15 (33%) had only biopsy. Although immediate postoperative morbidity was considerable (20%), most was transient and improved rapidly. In addition to craniotomy or biopsy, 38 (75%) patients required ventriculo-peritoneal shunt at the time of diagnosis or sometime during the course of their illness. The histological diagnosis was obtained in 45 of the 46 patients, which included anaplastic astrocytoma (27%), glioblastoma multiforme (15%), low grade glioma (16%), juvenile pilocytic astrocytoma (8%), and primitive neuroectodermal tumor(8%), anaplastic ependymoma (4%), anaplastic mixed glioma (4%), and others (12%). Malignant tumors consisted of 61% of our series. Eight (16%) patients received chemotherapy only, 2 (49%) received radiation therapy only, 23 (45%) received both, and 18 (35%) received neither. Of the 51 patients, 29 (57%) including 12 patients with malignant tumors are still known to be alive with average follow-up of 4 years. Ninety percent of these survivors lead an independent life of good quality. The authors discuss the evolution of thalamic tumor surgery over the past decade as well as the current treatment guidelines.

45. Surgical Management of Pediatric Chiasmal Astrocytomas

Michael D. Medlock, Joseph R. Madsen, Liliana C. Goumnerova, Nancy J. Tarbell, Patrick D. Barnes, Douglas S. Anthony, R. Michael Scott (Boston, MA)

Sixty-one patients <18 years old with chiasmal astrocytomas were evaluated between 1975 and 1994. The average age at diagnosis was 72 months. The most common presentations were visual difficulties in 18, neurofibromatosis in 10, and endocrine dysfunction in 9. The mean tumor diameter was 31 mm and 84% showed enhancement. There was involvement of the optic nerve in 41, hypothalamus in 38, optic tract in 15, optic radiations in 2, and 13 extended beyond the visual axis. Forty-one patients received conventional irradiation and 10 received stereotactic radiotherapy. Nineteen patients had chemotherapy at a mean age of 24 months.

Craniotomy was performed in 37 patients, 20 of these being operated on by a single surgeon (RMS). The astrocytoma was low grade in 35, anaplastic in 1, and unknown in 1. Temporary morbidity included coma (1), cranial nerve palsies (2), diabetes insipidus (2), visual deterioration (2), subgaleal effusions (3), and thermoregulatory dysfunction (1). Serious permanent morbidity included visual deterioration (6), hypopituitarism (3), stroke (2), memory loss (2), and diabetes insipidus (1).

Among 38 patients followed >5 years, 5 were normal, 20 were mildly impaired, 8 were markedly impaired, and 5 were dead. Late complications included deterioration to glioblastoma (2), spinal subarachnoid seeding (1), and two presumably radiation-induced problems, a sphenoid wing meningioma (1) and moyamoya syndrome (2). The posterior extent of the tumor at presentation was not a poor prognostic sign. No treatment was definitive. Large, exophytic or cystic tumors are amenable to surgical palliation although the attendant risks must be weighed.

46. Anaplastic Change in Pilocytic Astrocytomas

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

An examination of our 10-year series of pilocytic astrocytomas reveals that this disease entity is not as benign as commonly thought. We reviewed 36 cases of pilocytic astrocytomas operated on from 1984 through 1995. The mean age at initial presentation was 8 years (range 15 months to 14 years). These patients were followed for an average of 5½ years. None of these patients were given chemotherapy or radiation therapy after their initial surgery for pilocytic astrocytoma. Nineteen children (53%) required ventriculo-peritoneal shunting.

Twenty-three patients (64%) had a gross total resection with no residual tumor seen on immediate postoperative imaging studies. Three of these children had tumor recurrences at 2 to 5 years after their initial surgery, which were treated with re-excision. All three patients are tumor-free, with follow-up ranging from 4 to 10 years.

Nine of 13 patients with residual or recurrent tumor underwent re-excision. The second operation was undertaken an average of 1 year (range 1 month to 2½ years) after the first. In 4 of these children (11% of the series), the recurrent tumor was classified as anaplastic astrocytoma. These 4 children received radiation and chemotherapy, with no evidence of disease progression with follow-up of 2 to 5 years. Repeat blinded histopathological examination of these tumors confirmed both diagnoses. However, it was noted that 3 of the 4 pilocytic astrocytomas which subsequently showed anaplastic change initially displayed increased perivascular cellularity.

These results stress the need for continued follow-up of patients with pilocytic astroctyomas, and describe a histological feature which might indicate a more aggressive disease course.

47. Choroid Plexus Tumor Variants and Response to Surgical and Medical Management

Michael L. Levy, MD, Amy Goldfarb, BS, Grant Shumaker, MD, J. Gordon McComb, MD, Floyd H. Gilles, MD (Los Angeles, CA)

We sought to evaluate certain outcome variables associated with choroid plexus tumors. To accurately assess the relationship of these variables upon outcome, we only evaluated those 17 patients who presented to our institution since the inception of magnetic resonance (MR) imaging, all of whom underwent serial MR studies.

Eight patients had choroid plexus papillomas and 5 had choroid plexus carcinomas. There were 4 patients who had tumors with histologic features consistent with a papilloma, but in addition had more invasive characteristics and were classified as anaplastic papillomas. The mean age at diagnosis for the group was 27 months (range 2 months to 9 years). Ten tumors were in a lateral ventricle, 2 in the third, 4 in the fourth and 1 tumor was present in the cerebello-pontine (CP) angle. The placement of a ventriculo-peritoneal shunt was required in 3 patients preoperatively and 6 postoperatively. One patient required a subdural peritoneal shunt. Gross total resections were obtained in all but 3 patients, one having the CP angle tumor, the second having extensive diffuse CNS disease which was diagnosed by CSF cytology, and the third having a recurrence. Two patients required a second surgery to achieve gross total removal. All patients with carcinomas received chemotherapy and one also underwent a course of radiation (3,780 cGy). Only one patient with an anaplastic variant underwent chemotherapy and none of the papillomas. Of the patients with carcinomas, 4 out of 5 died at 5 months to $3\frac{1}{2}$ years (mean of 17 months) after diagnosis. The fifth patient is without evidence of recurrence at 2 years. In the patients with either papillomas or anaplastic papillomas, all 12 have survived 4 months to 10½ years (mean of 5 years) with 2 lost to long-term follow-up at 3 and 5 years.

We suspect that some papillomas with invasive characteristics, i.e., anaplastic choroid plexus papillomas, have been classified in some studies with choroid plexus papillomas while in others with choroid plexus carcinomas. Anaplastic papillomas may deserve a separate category but if not, should be classified with the papillomas as their biologic behavior more closely mimics that tumor variant than a carcinoma.

48. Childhood Ependymomas: Prognois Related to Location, Age, and History

Mammoud Moradi, Amir Friedman, Kenneth Shapiro, Charles Timmons, Derek A. Bruce (Dallas, TX)

Between 1982 and 1995, 39 cases of ependymoma were evaluated and treated at the Childrens Medical Center in Dallas. This represented 7% (39 of 587) of tumors in children over the 13-year period. Five cases of spinal ependymoma, 2 patients with neurofibromatosis, and 1 ependymoblastoma were excluded from the analysis leaving 31 cases for analysis. Twenty-two infratentorial and 9 supratentorial tumors were analyzed by age, location, degree of surgical resection, and histology with recurrence and survival being the outcome measures. The supratentorial tumors were diagnosed in an older age group than the infratentorial tumors, 87 and 44 months, respectively. All patients had negative CSF cytology and normal myelogram at postoperative staging. There were no deaths in the perioperative period (30 days). Gross total resection was achieved at the first operation in 19 (61.3%) children, 54.5% of the posterior fossa and 77.85% of the supratentorial tumors. The remaining cases all had near total resection. In the supratentorial space, after gross total resection (2 operations in 2 patients). XRT chemo or both there were no recurrences and all 9 patients are alive and tumor free. The 22 infratentorial cases were divided into 2 groups, <3 years, 13, and >3 years, 9. The 5-year survival rates are 52% and 58%, respectively, an unexpected finding. Five-year survival rates for the posterior fossa tumors are better in the children who had total resection for both age groups than those who had subtotal resection; <3 years, 75% vs 30%; >3 years, 65% vs 52%, but tumor-free survival was not affected by degree of resection. Tumor pathology had no influence on outcome MISS-ING COPY?

49. Contemporary Management of Intracranial Ependymomas in Young Children (<3 Years)

R. Sanford, S. Einhaus, A. Gajjar, L. Kun (Memphis, TN)

Between 1982-1995, 25 infants <3 years of age presented with ependymomas. Median age was 12 months (range 6 months - 36 months). Location of the tumor was as follows: posterior fossa n=21 (4th ventricular - 13, cerebellar pontine angle (CPA) - 8); supratentorial n=3; and spinal n=1. Histology was as follows: ependymoma n=17 and anaplastic ependymoma n=8. All patients had initial attempt at total surgical resection of the tumor. The surgical extent of resection was confirmed by neuroimaging obtained in the immediate postoperative period. Gross total resection (GTR) was achieved in 10 patients; the remaining 15 patients had subtotal resection (STR). Following surgery, patients received initial chemotherapy with planned delayed irradiation on serial institutional protocols (n=22) or radiation therapy alone (n=3). Progression free survival (PFS) for the series is 40% and the overall survival is 60% at a median follow-up time of 50 months (range 3 months - 160 months). In the 10 patients with GTR, 5 have no evidence of disease and 5 have recurred. Of the 15 patients with STR, 5 have stable disease and 10 have recurred.

Morbidity is of special interest in the 8 patients with CPA tumors, all of whom had multiple cranial nerve palsies following surgery and needed placement of a gastrostomy tube and tracheostomy; however, 5 have been removed, 2 remain tracheostomy-dependent at 6 months and 1 at 2 years postoperatively. A substantial portion of infants with ependymoma can be totally resected. This approach, along with postoperative chemotherapy and irradiation, appears to result in a favorable outcome for infants with ependymoma.

50. Correlation of Chromosome 17p Loss with Clinical Outcome in Patients with Primitive Neuroectodermal Tumors

Seyed M. Emadian, PhD, MD, Jeffrey D. McDonald, MD, PhD, Dan Fults, MD (Salt Lake City, UT)

Primitive neuroectodermal tumor (PNET) is the most common malignant central nervous system tumor in children. A frequent chromosomal abnormality in PNET is loss of heterozygosity (LOH) on chromosome 17p, reported in 30% to 60% of cases. Although the p53 tumor suppressor gene (TP53) is located on 17p, a different gene located telomeric to TP53 is most likely the target for the 17p deletions in PNET. LOH on 17p has been reported to portend a poor prognosis in PNET patients regardless of the clinical stage. We carried out an LOH analysis in 21 patients with PNET using polymerase chain reaction and polymorphic microsatellite markers for 12 distinct loci on chromosome 17. Deletion mapping revealed seven patients (33%) with LOH on 17p. The smallest region of overlap among the seven deletions spanned a large region (17p13.1-17pter) encompassing >19 centimorgan genetic distance. This region lies distal to TP53 and is larger than previously reported. To assess the clinical relevance of LOH on 17p, we correlated LOH with staging parameters known to predict survival (extent of tumor resection and presence or absence of preoperative craniospinal metastasis). Among the 14 clinically "good risk" patients, percent survival was 80% and 55% for patients with and without LOH, respectively. Among the 7 "poor risk" patients, percent survival was 0% and 40% for patients with and without LOH, respectively. We conclude that in our study population, LOH on 17p was not a significant predictor of survival even among patients with good clinical risk factors.

51. Glial Differentiation Predicts Poor Clinical Outcome in Central Nervous System Primitive Neuroectodermal Tumors

Leslie N. Sutton, MD, Anna J. Janss, MD, PhD, Anthony Yachnis, MD, Jeffrey H. Silber, MD, PhD, John O. Trojanowski, MD, PhD, Virginia M. Y. Lee, PhD, Giorgio Perilongo, MD, Lucy B. Rorke, MD, Peter C. Phillips, MD (Philadelphia, PA)

Background: Primitive neuroectodermal tumors (PNETs) of the central nervous system, including medulloblastomas, are the most common malignant brain tumors of childhood. These tumors often express proteins characteristic of glial differentiation (glial fibrillary acidic protein, GFAP), neuronal differentiation (neurofilament proteins, NFPs), and/or photoreceptor differentiation (retinal-S antigen).

Methods: To identify biological factors of prognostic significance in PNET-medulloblastoma, tumor specimens of 86 patients were studied with a panel of monoclonal antibodies utilizing immunohistochemistry and a microwave antigen enhancement technique. Tumor expression of glial, neuronal, or photoreceptor antigens was compared to relapse-free survival.

Results: Multivariate analysis of PNET-medulloblastoma immunohistochemistry and clinical variables indicated that GFAP expression confered a 6.7-fold greater risk of relapse than tumors withour GFAP or NFPs. Increased risk of relapse was directly related to the amount of GFAP expression. Tumors which exhibited clumps or sheets of GFAP-staining cells were associated with a 3.0-fold increased risk of relapse compared to tumors not expressing GFAP, irrespective of immunohistochemical evidence of other differentiation, while those tumors with scattered GFAP-staining cells had no significant increased risk of relapse. Expression of retinal-S antigen was associated with a 2.3-fold increased risk of relapse but was not statistically significant after correction for high GFAP expression (P = 0.06).

Conclusion: Expression of GFAP identified by immunohistochemistry and microwave antigen enhancement predict poor outcome in children with PNETs. The present findings indicate that expression of GFAP in PNET-medulloblastoma has prognostic power comparable to the most significant clinical factors.

52. Medulloblastoma with Brain Stem Involvement: The Impact of Gross Total Resection on Outcome

R. A. Sanford, A. Gajjar, S. Einhaus, R. Heideman, A. Walter, Y. Li, J. Langston, C. Greenwald, M. Muhlbauer, J. Boyett, L. E. Kun (Memphis, TN)

Brain stem involvement (BSI) is generally considered a negative prognostic factor in medulloblastoma (MB). The impact of gross total resection (GTR) in patients with this feature has not been directly addressed. Between 1984 and 1994, 100 patients with newly diagnosed MB were staged according to a modified Chang system; 40 of these were found to have BSI (T₃₁, 36; T₄ 4). Brain stem involvement included the presence of gross tumor invasion, adherent tumor plaque of any size or "candy coating" noted at surgery. Gross total resection (defined strictly as no residual disease at surgery or on MRI within 24-72 hours) was achieved in 13 of the 40 patients; the remaining 27 had a subtotal resection (STR). The proportion of patients with metastatic disease at presentation did not differ significantly between the 2 groups. All patients have completed therapy (follow up 0.5-8 years; median 3.5 years). All patients received radiation therapy (RT), and 34 were selected for pre-irradiation chemotherapy based on age <4 or measurable residual disease. Progression free survival (PFS) at 4 years is 53% for the GTR group and 63% for the STR group. An analysis stratified by prior treatment (RT only; scheduled chemotherapy plus planned RT, RT following progression on chemotherapy) did not identify an impact of GTR on PFS in comparison to the group with residual tumor postsurgery (p>0.80). These data suggest that the prognostic impact of BSI of any degree may override any influence of degree of resection.

53. Surveillance Scanning in Medulloblastoma

Dennis Shaw, Russ Geyer, Karen Lindsley, Jerrold Milstein, Mitchel S. Berger (Seattle, WA)

The usefulness of postoperative surveillance MRI/CT scanning in the management of children with medulloblastorna (MB) has been recently challenged. We reviewed the 10-year experience (1984-1993) of children with MB treated at our institution to evaluate the role of surveillance scanning in the detection of and outcome following tumor recurrence.

Fifty-nine children with MB were treated. Surveillance scanning with CT or MRI occurred every 3 months for 2 years; then every 6 months for 3 years. Nineteen children have had tumor recurrence; 17 of whom had adequate follow-up for evaluation. The median time to tumor progression was 13 months (3-90 months). In 11 patients progression was discovered on routine surveillance scans, while 6 patients were symptomatic at recurrence.

| | Symptomatic Recurrence | Asymptomatic Recurrence |
|--------------------------|--------------------------------------|-------------------------------|
| | n = 6 | n = 11 |
| Time to First Recurrence | Median: 38.5 mo >24 mo: n = 4 | Median: 12 mo >24 mo: n =0 |
| Treatment of Recurrence | n = 2 | n = 9 |
| Survival Post-Recurrence | Median: 4.5 mo >9 mo: n = 1 (17%) | Median: 6 mo >9 mo: n = 4 |
| (36%) | ` , | |

Conclusion: The majority of patients were asymptomatic at tumor recurrence, which was discovered on scheduled surveillance scanning. While the cure of patients with recurrent MB remains unusual, the early detection of small recurrences may allow for the use of new treatment techniques, such as radiosurgery or permanent radiation implants, and provides a reasonable setting in which to investigate the use of aggressive salvage chemotherapy protocols. In addition, more long-term survivors may be anticipated when treatment is initiated for children with asymptomatic (36%) versus symptomatic (17%) MB recurrences.

54. Value of Postoperative Surveillance Scans in the Management of Children with Some of the Common Brain Tumors

Paul Steinbok, MBBS, BSc, FRCSC, Stephen Hentschel, D. Douglas Cochrane, MD, FRCSC, John R. W. Kestle, BSc, MD, MSc, FRCSC (Vancouver, BC, Canada).

The rationale for surveillance CT or MR scans in pediatric brain tumors is that early detection or recurrence may result in better outcome. The purpose of this study was to investigate, for a variety of common pediatric brain tumors managed at a tertiary care pediatric hospital, the value of surveillance cranial scans.

A retrospective chart review was performed of children with astrocytoma of the cerebral hemisphere, cerebellum, optic chiasm/hypothalamus or thalamus, cerebellar or supratentorial high-grade glioma, supratentorial ganglioglioma, posterior fossa or supratentorial PNET, or posterior fossa ependymoma. Data were analyzed to determine the frequency with which recurrences were identified on a surveillance scan, and how the type of scan at which recurrence was identified related to outcome.

In 159 children, 17 of 44 recurrences were diagnosed by surveillance scanning. The percentage of recurrence identified by surveillance scanning was 64% for ependymoma, 50% for supratentorial PNET, 43% for optic/hypothalamic astrocytoma, and less than 30% for other tumors. The rate of diagnosis of recurrence per surveillance scan varied from 0% to 11% for different tumor types. Only for ependymomas did there appear to be an improved outcome when recurrence was identified prior to symptoms.

Our results suggest that surveillance scanning is not required for cerebellar astrocytoma or supratentorial ganglioglioma, but probably is worthwhile in posterior fossa ependymoma and optic/hypothalamic astrocytoma, and possible for medulloblastoma. Surveillance protocols should be individualized for each type of tumor, based on data about the patterns of recurrence, both with respect to time and sites of recurrence.

55. Better Dead than Dull? Historical and Ethical Issues in the Treatment of Medulloblastoma

Jennifer C. Kernan, MD, Joseph H. Piatt, MD (Portland, OR)

Despite several investigative missteps, the therapy of medulloblastoma has so progressed that the issue of treatment morbidity now rivals that of tumor mortality. Cranial irradiation is both the most significant advance and the most serious source of morbidity The adverse cognitive sequellae of radiation have been incompletely defined. An exhaustive literature search revealed 26 papers which included an assessment of cognitive outcome following treatment for medulloblastoma. These studies were compared against each other and with 11 studies examining the cognitive sequellae of prophylactic cranial irradiation for acute lymphocytic leukemia. The goals were to determine: 1) what percentage of survivors are cognitively impaired; 2) how severe is the loss; 3) what are the characteristic deficits, including the time course; 4) does age correlate with outcome; 5) to what extent is radiation responsible; 6) does radiation dose relate to the degree of impairment. Comparisons between studies are hampered by 1) nonuniformity in assessing cognitive outcome; 2) variation in and incomplete reporting of treatment regimens; 3) lack of prospective controlled studies. We conclude that the majority of survivors are impaired: most have borderline IQs and require special education. Impairment progresses over several years before stabilizing; the youngest patients are worst afflicted. Comparison with children treated for other tumors exculpates surgery or chemotherapy and implicates cranial irradiation, but the question of a dose-outcome relationship remains unanswered. Current clinical research, in substituting chemotherapy for radiation, raises an ethical issue in balancing incompletely defined cognitive morbidity with the possibility of increased mortality. Failure to approach a concensus on this balance may threaten progress in treatment.

56. Outcome and Cost Analysis of Recurrent PNET

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

The 40% to 50% of primitive neuroectodermal tumors (PNET) of the posterior fossa which recur carry a dismal prognosis. We reviewed 27 cases of recurrent PNETs treated over the past five years. Our series included 18 males and 9 females. The age at presentation ranged from 1 month to 15 years, with an average of 5½ years. Twenty of the 27 patients initially met the "poor risk" criteria as defined by the Childrens Cancer Study Group. Twenty-two patients received both chemotherapy and radiation therapy, 3 received chemotherapy only, and an additional 2 had no further treatment. Recurrence was discovered within 3 years for all patients, and as early as 1 month (mean of 7 months) of the initial diagnosis. Nineteen of these patients are dead, having expired a mean of 7 months after their recurrence. Two of the dead, however, survived for 5 years after noting tumor recurrence. Two of the 8 survivors have had a complete response; the other 6 have had partial responses, with an average follow-up of 3 years.

Nineteen of the patients underwent neuropsychological testing. Three of these patients progressed to severe intellectual impairment or developmental delay within the first 6 months of their illness. Eight of the children had low average or borderline scores. Eight patients, two who had no radiation therapy, had average intelligence scores. However, three of these progressed to borderline status at the end stages of their illness.

The economic cost of this disease is large. These patients had an average of 11 in-patient hospitalizations per year for chemotherapy and various complications of their disease. The average hospital cost over the course of the disease per patient was \$355,760 (ranging from \$27,800 to \$810,550). These figures do not include the cost of operative procedures or physician fees. Almost half of this amount was spent on imaging studies, whereas the remainder was divided equally between drugs, laboratory tests, and room charges.

In sum, recurrent PNET is a devastating disease. The vast majority of patients suffer significant neuropsychological deficits and spend a great deal of their short survival time in the hospital, at significant monetary cost.

Scientific Posters

1. Anaplastic Astrocytomas of the Spinal Cord in Children

Tania Shiminski-Maher, MS, CNRN, Cheryl Muszynski, MD, Diana Freed, BA, Douglas Miller, MD, David Zagzag, MD, Fred Epstein, MD (New York, NY)

Pediatric spinal cord tumors are relatively uncommon. comprising only 4% of all central nervous system tumors in childhood. The majority of these tumors are astrocytomas. From 1980-1994, 200 children with intramedullary cord tumors underwent radical tumor resection at New York University Medical Center. Twenty-six of these 200 children (13%) had pathologically confirmed anaplastic astrocytomas. Of this group, 50% died within 2 postoperative years, many despite aggressive adjunctive treatment. In contrast, the other 50% are alive, some having received no adjunctive therapy. This presents a dilemma to the clinician in terms of the postsurgical treatment recommended. It may be possible that some of these tumors while histologically anaplastic have a more indolent course and can simply be observed while others are very aggressive and like glioblastomas do not respond to any treatment. Central pathology review, MIBI staining, adjunctive treatment, and clinical follow-up of these 26 children will be presented with a focus on correlating survival with the above mentioned factors.

Role of Oxygen Free Radicals and Adenosine in Leukocyte-Endothelial Adherence in the Newborn Pig Brain During Postasphyxic Reperfusion

Tae Sung Park, MD, Ernesto R. Gonzales, BSN, Raymond G. Maceren, BS, Aarti R. Shah, MS, Jeffrey M. Gidday, PhD (St. Louis, MO)

Recent evidence suggests that leukocytes contribute to cerebral ischemic injury. We employed customized videomicroscopy and image analysis to investigate acute changes in the adherence of rhodamine 6G-labelled leukocytes to cerebral venules in isoflurane-anesthetized newborn piglets outfitted with closed cranial windows. We demonstrated previously that, following 9 minutes of asphyxia, a progressive increase in leukocyte-endothelial adherence occurs concomitant with significant increases in cortical xanthine and uric acid concentrations (as measured by microdialysis), indicative of superoxide radical formation secondary to the activity of xanthine oxidase. In the present study, we proposed to test the hypothesis that oxygen free radical scavengers would decrease leukocyte-endothelial adherence during reperfusion following asphyxia. In addition, we assessed the effects of adenosine on this inflammatory response to asphyxia. In one animal group, superoxide dismutase (SOD [60 U/ml]) and catalase (CAT [1200]) U/ml]) were superfused through the window (50 µl/min) starting 30 minutes prior to asphyxia; the polyethylene glycol-conjugates of these enzymes were concomitantly administered intravenously at 10,000 U/kg. Windows from a second group of animals were pretreated with a superfusion of the A, adenosine receptor agonist CGS 21680 (5 µM). Shown below are the number of leukocytes adherent to cerebral venules within the 0.55 mm² videofield, above that measured during baseline conditions, at 1 and 2 hours of reperfusion after asphyxia.

| ANIMAL GROUPS | 1 h Reperfusion | 2 h Reperfusion |
|----------------------------|-------------------|-----------------------|
| Non-asphyxia (n=4) | -1 ± 1 | -1 ± 2 |
| Asphyxia (n=6) | 29±5* | 56±8* |
| Asphyxia + SOD/CAT (n=5) | 11±3*† | $20 \pm 9^{\dagger}$ |
| Asphyxia + CGS 21680 (n=5) | $7\pm5^{\dagger}$ | $-1 \pm 10^{\dagger}$ |

(*p<0.05 vs baseline and nonasphyxic control group; †p<0.05 vs asphyxia).

3. Design and Testing of a New Auto-Adjusting Flow Regulating Device for Shunting of CSF

Newton Paes, MD (São Paulo, Brazil)

Traditional shunts were primarily designed to manage hydrocephalus by regulating intracranial pressure. Their performance characteristics, however, can cause them to underdrain or overdrain CSF. Overdrainage has been linked to clinical complications such as "low pressure" headaches, slit ventricle syndrome, subdural hematomas, and may contribute to ventricular catheter occlusion. Newer shunt designs attempt to improve this performance, but fall short for various reasons. The author presents a new shunt design which utilizes patent pending variable aperture technology which results in the physiologic regulation of CSF flow under both positive and negative pressure conditions. This new design offers encouraging promise for the management of hydrocephalus and the prevention of complications due to overdrainage.

4. Pediatric Posterior Fossa Glioblastoma: MRI Findings

W. Robinson, MD, Y. Numaguchi, MD, M. I. Rothman, MD, G. H. Zoarski, MD, M. Zagardo, MD, D. A. Kristt, MD (Baltimore, MD)

Glioblastoma multiforme presents primarily in the supratentorial compartment in all age groups. Its occurrence is unusual in the posterior fossa in adults and rare in childhood. There are several reports of cerebellum or brainstem occurrence, but few reports on the MRI presentation.

This study analyzed the MR findings of 8 patients with posterior fossa glioblastoma multiforme of the cerebellum and brainstem. Between 1986 and 1993, 236 patients were diagnosed at the University of Maryland Medical Center as having glioblastoma. Eight patients between 2 and 18 years old (mean=7.1) had histologically verified glioblastoma of the posterior fossa. Specimens were obtained from resected tumors in 3 cases and from excisional biopsies in 5 cases.

MRI studies were performed using 1.5T (5 cases) and 1.0T (2 cases). Contrast MR studies (GD-DTPA 0.1 mmol/kg) were performed in all patients. The MRI studies were reviewed and analyzed to determine tumor size, location, shape; signal intensities and contrast enhancement; and the presence of edema, hemorrhage, hydrocephalus, necrosis, and metastases.

Illustrative cases and imaging findings will be presented.

5. Endoscopic Stereotaxic Neurosurgery: Frameless Stereotaxy with an Endoscope

George I. Jallo, MD, I. Rick Abbott, MD (New York, NY)

Stereotactic surgery is not a new field in neurosurgery. As well, endoscopic procedures have been performed since the early 1900s. Several neurosurgeons have combined these two techniques, stereotactic frames and rigid endoscopes, in certain approaches for intraventricular tumors. We describe our experience with a frameless stereotaxic navigational system and the flexible endoscope for a variety of cases. We have removed several intraventricular tumors (n=4) and colloid cysts (n=3). In addition, we have treated loculated hydrocephalus (n=5) and retrieved broken shunt catheters (n=3) with this combined technique.

We use the Electra/ISG viewing wand for guiding our endoscope during the operative procedure. We localize our point in space by passing the wand through the peel-away sheath, and then pass the endoscope through the sheath at the precise distance to perform the operative procedure. In certain cases, we have been able to digitize the 1.2 mm endoscope to the stereotactic navigational system and perform the surgery with real-time multiple perspective image updates.

Although these combined minimally invasive techniques have been highly efficient, it is not free of complications. There have been no deaths but two patients sustained intraventricular hemorrhages necessitating external ventriculostomy placement for 72-96 hours. No patient required an open craniotomy for resection of their tumor, treatment of their hydrocephalus, or removal of the intraventricular hemorrhage.

6. Classification of Hydrocephalus and Outcome of Treatment

Koreaki Mori, Junichi Shimada, Masahiro Kurisaka, Kiyoshi Sato, Kazuyoshi Watanabe (Kochi, Japan)

Purpose: Retrospective analysis in cooperative study of hydrocephalus at institutions of members of the Research Committee on Intractable Hydrocephalus sponsored by the Ministry of Health and Welfare of Japan was performed to determine functional prognosis for all types of hydrocephalus and thus to clarify the outcome.

Methods: In preparation of this study, we have proposed the definition, clinical classification, and diagnostic criteria of hydrocephalus. We have classified non-tumoral hydrocephalus into 8 types based on its etiology and the time of onset. To establish the diagnosis in each type of hydrocephalus, we have set up inclusion and exclusion criteria, as well as supplementary criteria which are useful for its diagnosis.

Results: Analysis of the 1450 cases of hydrocephalus of various etiologies stored in the database obtained from the study for each type of hydrocephalus revealed that following types and conditions were associated with a neurologic deficit: 1) early fetal hydrocephalus, 2) overt neonatal hydrocephalus, 3) hydrocephalus associated with such severe brain malformations as hydranencephaly, holoprosencephaly, and lissencephaly, 4) hydrocephalus associated with severe brain damage, 5) hydrocephalus associated with epilepsy, 6) hydrocephalus shunted late after detection, and 7) hydrocephalus complicated by a shunting operation.

Conclusion: The postnatal functional outcome was significantly poor in fetal hydrocephalus diagnosed in the early gestation. Childhood onset hydrocephalus showed a poorer outcome than adult hydrocephalus.

7. Tyrosine Hydroxylase Immunoreactivity Changes in the Nigrostrial Dopaminergic System in Experimental Hydrocephalus

Shushovan Chakrabortty, Yuzuru Tashiro, James M. Drake, Toshiaki Hattori (Toronto, ON, Canada)

Introduction: Disturbances in motor and cognitive functions occur in hydrocephalus. The structural and/or functional changes in the basal ganglia, especially in the nigrostrial dopaminergic system, may have some role in the pathogenesis of gait and postural disturbances. Dopamine levels have been reported to be decreased in the neostriatum of both in infant and adult hydrocephalic animal models. In the present study, we examined the immunocytochemical changes of the catecholamine synthesizing enzyme, tyrosine hydroxylase (TH), in the nigrostrial dopaminergic system of the adult hydrocephalic rat.

Material and Methods: Hydrocephalus was induced in 16 adult Wistar rats by intracisternal injections of kaolin suspension. One, 2, 3, and 4 weeks after the injections, the animals were killed by transcardial perfusion of glutaraldehyde-formaldehyde solution, and the TH-immunoreactive profile was examined in serial rostro-caudal coronal sections of rat neostriatum by light microscopy using the avidin-biotin-peroxidase complex method, and compared with 4 control rats.

Results: Hydrocephalus varied from mild to severe. The neostriatum was compressed by the enlarged ventricles especially in the severely hydrocephalic animals. The TH-immunoreactivity in the 1 week group was similar to that of control animals. In the 2, 3, and 4 weeks groups, the TH-immunoreactivity was reduced in the neostriatum especially in the severely hydrocephalic animals.

Conclusions: The decreased TH-immunoreactivity is in support of the reduced dopamine content in the neostriatum of the hydrocephalic brain. The decreased TH-immunoreactivity may be caused by the direct compression and degeneration of the nigrostrial dopaminergic axons and by subsequent retrograde changes in the dopaminergic neurones.

8. The Changes of Neuronal and Dendritic Calbindin-D Immunoreactivity in Cerebral Cortex, Striatum, and Hippocampus of the Hydrocephalic Rat

Yuzuru Tashiro, Shushovan Chakrabortty, James M. Drake, Toshiaki Hattori (Toronto, ON, Canada)

Introduction: Calbindin-D (CaBP), one of the neuronal calcium-binding proteins, has a broad distribution in the central nervous system. The purpose of this research was to investigate immunohistochemically the morphological and functional changes of CaBP-immunoreactive (IR) neurons and dendrites in the hydrocephalic brain.

Material and Methods: Sixteen adult Wistar rats, under deep anesthesia, received kaolin injections into the cisterna magna with microscopic confirmation of atlanto-occipital dural puncture. They and four control rats were killed after 1, 2, 3, and 4 weeks survival and studied by CaBP immunohistochemical stain and Nissl stain. The neuronal and dendritic immunoreactivity in the cerebral cortex, striatum, and hippocampus in the hydrocephalic brain were compared to that in the control animals.

Results: Hydrocephalus occured in 90% of kaolin-injected animals. In the cerebral cortex, the early change was the reduction of CaBP-IR dendrites, especially of cortical interneurons. With progressive ventricular dilatation, the number of CaBP-IR neurons in deeper layers decreased, and ultimately there were no cortical CaBP-IR neurons. In the striatum of severe hydrocephalic cases, loss of neuronal and dendritic CaBP-IR progressed from medial to lateral. In the hippocampus, there was loss of CaBP-IR interneurons with preservation of CAI pyramidal neurons. Nissl stain revealed no ischemic changes in the hydrocephalic cortex and striatum, but slight ischemic changes in the hippocampus of severe hydrocephalic cases.

Conclusion: Hydrocephalus results in the progressive and characteristic loss of CaBP-IR neurons and dendrites in the cerebral cortex, striatum, and hippocampus of rats.

9. The Rabbit Model for Infantile Hydrocephalus: Preservation of Barrel Fields in Somatosensory Cortex

Charles E. Olmstead, PhD, Katherine Y. Eng, Monica C. Wehby-Grant, MD, Warwick J. Peacock, MD, Robin S. Fisher, PhD (Los Angeles, CA)

The cortical barrel fields of the mystacial vibrissae represent an analog of the homunculus of human postcentral cortex. We describe here the effects of kaolin induced hydrocephalus on the development of barrel field organization in the rabbit.

Rabbits were made hydrocephalic at 4-6 days of age by an intracisternal injection of 100 μ l of kaolin. To assess early development, littermate control and hydrocephalic pairs were sacrificed at 16, 19, 24, 27, and 30 days of age and 40 μ tangential frozen sections processed for cresyl violet histology and cytochrome oxidase histochemistry. Older (30 to 75 days) normal (N = 11) and hydrocephalic (N = 8) animals were perfused with paraformaldehyde, the brains tangentially sectioned at 60 μ thickness and stained with cresyl violet. Barrel fields (BFs) were drawn at 250x magnification and barrel area and number of cells per barrel were determined.

In the normal developing animals the BFs were easily identified and quantified at the first age studied (16 days). The hydrocephalic BFs, on the other hand, were qualitatively different until 24 to 27 days. In the hydrocephalic animals the fields showed little increase in size up to 40 days of age and then showed steady and significant increase with age. In the older animals studied there was a 25% increase in the diameter of the BFs of the hydrocephalics compared to the normals. The hydrocephalic BFs retained their overall ovoid shape and structure and the enlargement was inversely proportional to the thickness of the cortical mantle.

The development of layer IV BFs is retarded in the young hydrocephalic, but the overall integrity is maintained even in the chronic state.

10. Factors Influencing Outcome in Brain Tumor Surgery in Children Less Than Two Years of Age

Keith S. Blum, DO, Steven J. Schneider, MD, Alan D. Rosenthal, MD (New Hyde Park, NY)

Between 1969-1994, 38 children under the age of 2 with signs and symptoms relating to cerebral neoplasms were studied. Variables analyzed included age at onset, clinical presentation, tumor location, histology, extent of surgical resection, postoperative mortality, adjunctive therapy, survival, and quality of life. Correlation between all variables and outcome were subjected to univariate analysis, Pearson's r or chi-square testing. Survival rate was calculated via the Kaplan-Meier method. The most common tumors were benign astrocytomas, ependymomas, and medulloblastomas. Age at presentation ranged from 0-24 months (average 12 months). Most patients presented with a clinical history of 8 weeks or less with common findings including macrocephaly, full fontanel, neurodevelopmental delay, and change in behavior. Supratentorial tumors (63%) were more prevalent than infratentorial lesions (37%). All patients underwent surgical excision with histological confirmation. There were 22 benign tumors and 16 malignant tumors. Surgical mortality was 7.89%. Follow-up ranged from 2 months to 25 years. Outcome was assessed according to a modified Bloom Clinical Evaluation Scale. Seventeen patients had a good outcome while 18 patients had a poor outcome and 15 have expired. Younger patients (less than 1 year) with poorer outcomes and shorter 5-year survival periods (36%) had predominantly supratentorial malignant tumors while the older patients (greater than 1 year) with better outcomes and longer 5-year survival rates (69%) more frequently had benign infratentorial lesions. All influencing factors will be presented in detail.

11. Head Registration Techniques in the Pediatric Age Group

M. S. Alp, J. I. Ausman, M. Dujovny, F. T. Charbel, M. Misra, A. Goldberg (Chicago, IL)

Image-guided surgery is the latest combination of microneurosurgery and computer technology. Stereotactic microsurgery and high-precision definition of vital anatomical structures become available with new stereotactic planning systems and microscopes. Use of these sophisticated systems requires the registration of the patient's head in a precise manner. In this study, we used a Zeiss MKM stereotactic microscope system to operate on five pediatric patients.

Image-guided surgery techniques require the registration of the patient's head, so two-dimensional images can be used to create a three-dimensional stereotactic cube. In order to create the three-dimensional images, the head should be registered. The most common and best-known registration method is the use of stereotactic frames. Since the development of frameless techniques, there are currently other registration methods also used for the same purpose.

In our institution, three different registration techniques have been utilized. These are the Zamorano-Dujovny stereotactic frame, skull fiducials, and adhesive skin markers. Although higher precision values are achieved by more stable methods such as the stereotactic frame, other registration techniques have more flexibility than the frame. Our measurements showed that average error values with the frame are ~0.6 mm, with skull fiducials ~0.9 mm, and with skin markers ~1.1 mm. Despite the fact that skin markers have less accuracy compared with other techniques, it has certain advantages like noninvasiveness and ease of use. Skin markers are more accurate in the pediatric age group than in adults, due to children's high skin tensile strength and the tonus.

12. Chiari Crisis: Early Intervention and Long-Term Survival

R. F. Keating, MD, J. T. Goodrich, MD, PhD (Bronx, NY)

Long considered a deadly manifestation of Chiari II malformation in the infant, aggressive, early surgical intervention of this condition in the past decade has dramatically changed the overall prognosis for these patients. These patients may present with acute onset of stridor, hoarseness, swallowing difficulties, respiratory embarrassment, ophistotonus as well as autonomic dysfunction. Unless recognized and treated in an expeditious fashion, the general prognosis has historically been grim.

During the past 8 years we have treated 7 infants (4 males, 3 females) who presented with acute onset of brainstem compression secondary to Chiari malformation. Evaluation at this time failed to demonstrate any evidence of shunt malfunction. Their ages ranged from 2 weeks to 28 weeks with an average of 10 weeks. All infants continued to deteriorate despite supportive measures and subsequently underwent posterior fossa/cervical decompression following radiological confirmation of their disease. Time of neurological recovery varied according to the severity and duration of symptoms. Nevertheless, all patients with the exception of one, did well over the long term. We believe that prompt diagnosis and surgical decompression undertaken in the infant with Chiari crisis offers these individuals the greatest chance at normalizing brainstem function and avoiding an otherwise fatal deterioration.

13. Non-anastomotic Bypass Surgery for Childhood Moyamoya Disease Using Dural Pedicle Insertion Over the Brain Surface Combined with EGMS

Yasuko Hara, Reizo Shirane, Takashi Yoshimoto (Sendai, Japan)

A new combined surgery of nonanastomotic bypass procedures using dura mater, galea, and deep surface of temporal muscle is proposed for childhood moyamoya disease. Although the direct bypass provides immediate blood supply, it contains some complications caused by technical difficulties with smaller arteries and others. The direct bypass may not always be the best treatment without rapid progression and severe symptom. As the course of this disorder is different and unexpectant in each child, we preserve STA for another surgical treatment which may be required in the future. We cut the dura peduncularly into several pieces and insert them into subdural space outside of the cranial window toward the ischemic area. The dural flaps with branches of meningeal arteries can produce collaterals in extensive area around the cranial window, even in the ACA perfused area by the insertion to the interhemispheric fissure. The operative field should be decided and modified in consideration of PET scan which reveal the area of low hemodynamic reserve. To promote the collateral formation, we tear arachnoid membrane along cerebral sulci. The thinly layered deep surface of the temporal muscle and the galea are used for dural closure. The skin flap with abundant blood supply from remaining temporal muscle and STA can prevent alopecia or necrosis of the scalp. This procedure has been performed 36 times in 16 children and 4 young adults with movamova disease. In all cases, collaterals were well established on follow-up angiography and their clinical symptoms were improved.

14. Ventricular Peritoneal Shunt Malfunction Following Operative Correction of Scoliosis: Report of Three Cases

Michael Egnor, MD, Wesley Carrion, MD, Susan Haralabatos, MD, Michael Wingate, BA (Stony Brook, NY)

Two of the most common causes of hydrocephalus in children are spina bifida and intraventricular hemorrhage of prematurity, both of which are known to be associated with spinal deformity in later childhood. The occurrence of shunt malfunction following mechanical injury or stress to the hardware has been well documented. Newer techniques in the treatment of a neuromuscular scoliosis, including anterior release with segmental fixation, have resulted in more powerful correction of these large spinal deformities. A new potential cause of shunt malfunction is the aggressive correction of scoliotic deformity.

We report three patients with neuromuscular curves averaging 100 degrees who were subsequently recognized to have perioperative shunt malfunction. All three children were young adolescents and had had long-term shunts. Two of the children had spina bifida and a third had cerebral palsy. All children underwent anterior release of their scoliosis with posterior segmental instrumentation with unit rods and subliminal wires. All had significant correction of their scoliosis. Two children had shunt malfunctions within a month of their surgery and one child had intraoperative recognition and externalization of the shunt.

Older children undergoing repair of neuromuscular scoliosis are often preadolescents or adolescents who have had the same indwelling shunt systems originally implanted in early infancy. The shunt may be brittle and calcified and the peritoneal catheter may be short. The correction of scoliosis often results in an almost instantaneous growth of a few inches. Because of the potential difficulty in recognizing shunt malfunction in the perioperative period, consideration should be given to elective revision of the peritoneal catheter in children at risk.

15. Seatbelt Injuries to the Pediatric Spine: A Report of 11 Cases

Michael Egnor, MD, Wesley Carrion, MD, Susan Haralabatos, MD, Ira Chernoff, MD, Thomas Smith, MD (Stony Brook, NY)

Many states have laws requiring restraints for children in cars. While this has undoubtedly saved many lives, we report 11 children who suffered spinal injuries while restrained during accidents.

Six children suffered cervical spinal injuries, all of which were to the occipital-C1-C2 complex. These injuries were most common in the younger children. Three of the children were rendered quadriplegic and 2 children suffered less severe spinal cord injuries. One child was intact and 1 quadriplegic child died. The injuries were managed with surgical stabilization and halo immobilization.

Four children suffered thoracolumbar injuries. One child suffered lumbar plexus avulsion and 3 were intact. They were managed with surgical stabilization and/or TLSO immobilization.

One child suffered both cervical and thoracolumbar injury, with a pattern of neurological injury consisting of Bells cruciate palsy.

Even properly restrained children can suffer spinal injuries during motor vehicle accidents. Associated visceral injuries are common, and frequently these children are the only severely injured occupants of the car. Recognition of these injury patterns is important in the neurosurgical evaluation of children with vehicular trauma.

16. Quantitative Analysis of Cerebrospinal Fluid Spaces in Children with Occipital Plagiocephaly

Paul D. Sawin, MD, Michael G. Muhonen, MD, Arnold H. Menezes, MD (Iowa City, IA)

Introduction: The etiology of occipital plagiocephaly or "functional lambdoid craniosynostosis" is not fully understood. We have observed that many infants with this condition have benign communicating hydrocephalus. This investigation was undertaken to quantify CSF-space caliber in children with occipital plagiocephaly, and to compare these measurements to those derived from normal age-matched controls in an attempt to further elucidate the pathogenesis of this condition.

Methods: Thirty-one infants with unilateral functional lambdoid craniosynostosis, mean age 6.0 months, were studied. Infants with multiple cranial suture abnormalities, impaired neurological function, developmental delay, and associated craniofacial anomalies were excluded. Twenty normal infants were studied for comparison. The volumes of the Sylvian fissures, suprasellar and perimesencephalic cisterns, and anterior subarachnoid spaces were calculated from CT images. Ventricular size was also assessed.

Results: Benign communicating hydrocephalus was observed in 93.5% of those with occipital plagiocephaly. The FOC was significantly greater in the case group (71.4% ile vs. 50.8% ile , p = 0.0002, ANOVA). Sylvian fissure volume was significantly larger in the case group (5.8 ml vs 0.75 ml in controls, p<0.0001). The volume of the contralateral Sylvian fissure was greater than that ipsilateral to the side of plagiocephaly (7.1 ml vs. 4.5 ml, p=0.001). Anterior subarachnoid space volume was greater (27.5 ml vs. 0.6 ml in controls, p<0.0001). Both the suprasellar and perimesencephalic cisterns were also of greater caliber (p=0.007 and p<0.0001, respectively). No significant difference in ventricular size was noted between groups.

Conclusion: The extraventricular CSF spaces in neurologically unimpaired infants with functional lambdoid craniosynostosis are significantly larger than those of age- and sex-matched controls. Enlarged subarachnoid spaces may increase the compliance and malleability of the calvarium and sutures, predisposing to positional deformity. Benign communicating hydrocephalus of infancy may be a fundamental etiologic factor in functional lambdoid craniosynostosis.

17. Use of MRI in the Evaluation of the Cervical Spine in the Multiply-Injured Young Child

R. A. Armonda, R. J. Weil, J. Dormans, J. Hunter, A. C. Duhaime (Philadelphia, PA)

Evaluation of the cervical spine in a multiply-injured young child is a difficult task, especially in the face of an associated head injury and decreased level of consciousness. In our institution, all children whose cervical spines cannot be cleared using plain films, C1-2 CT scan, and clinical exam undergo a screening MRI scan of the region. We report eight infants and young children who were found to have abnormalities on MRI using this screening protocol.

Patients ranged in age from 8 months to 5 years (mean 3 years). All but two children had no clinical evidence of spinal cord injury. Seven injuries were diagnosed in the acute phase and three were found in a delayed fashion because of persistent symptoms (abnormal neck posture and unwillingness to walk). Injuries included ligamentous tears involving the posterior interspinous ligaments, cruciate ligament, and rectorial membrane, spinal cord contusion, cervical subarachnoid hemorrhage, synchondrosis fractures, and instability with neural compression. Ligamentous instability was confirmed by flexion-extension plain films in four of the eight patients, but was limited by poor patient cooperation and inability to assess the extent of neural compression.

Management involved prolonged immobilization in four patients and surgery in four; treatment was clearly influenced by the MRI findings. We conclude that a screening protocol for the cervical spine invloving MRI in young children provides useful clinical information which can influence treatment. The natural history of injuries so detected and the cost-effectiveness of this protocol requires further study.

18. Stereotactic Depth Electrodes for Localizing Seizure Foci in Children

P. David Adelson, MD, A. Leland Albright, MD, Peter C. Gerszten, MD, Patricia K. Crumrine, MD, John K. Vries, MD (Pittsburgh, PA)

Complex partial seizures, though more common in adults, occur in 25% of children with epilepsy. In children with medically intractable seizures, surgical extirpation of the seizure focus is best achieved if it can be precisely localized. Noninvasive evaluation may not localize the epileptogenic zone sufficiently, and invasive monitoring is therefore indicated.

At the Children's Hospital of Pittsburgh, 29 children had stereotactic depth electrodes (DE) placed during 31 procedures for chronic EEG monitoring over a 12-year period. The average age at implantation was 13 years (range 3-18) and the average length of monitoring was 24 days (range 6-63). Twenty-three patients had a diagnosis of complex partial seizures while the others had generalized tonic-clonic (5) and atonic seizures (1). DE were inserted to lateralize a temporal focus or to differentiate between an inferofrontal or parieto-occipital and temporal focus.

Surface electrodes were used for suspected cortical seizure foci or close proximity of functional areas to the seizure focus. Nineteen patients eventually underwent a resection based on the DE findings and 5 others underwent an anterior callosotomy. One patient underwent a callosotomy, was re-evaluated, and then had a frontal resection. Six patients had bilateral foci, were excluded from a surgical option, and are attempting further medical treatment. There were 5 complications, all infections. Four of the patients developed meningitis and the other a cerebritis. All were treated successfully with antibiotics alone. The risk of infection did not correlate with duration of DE monitoring.

In children, DE monitoring is helpful in precisely localizing medial temporal seizure foci not identified by scalp EEGs. It appears to be a safe modality that is well tolerated.

19. The Role of Neuroendoscopy in the Treatment of Pineal Region Tumors

S. Robinson, A. R. Cohen (Cleveland, OH)

Improvements in neuroimaging and microsurgical techniques have allowed safer, more complete resections of pineal tumors. In spite of this, the optimal

treatment for individual pineal lesions, as well as the often-associated obstructive hydrocephalus, remains controversial.

The authors report an alternative surgical strategy for managing patients with pineal region tumors and symptomatic noncommunicating hydrocephalus. Prior to definitive surgical extirpation of the neoplasm, patients presenting with hydrocephalus and increased intracranial pressure undergo emergent endoscopic third ventriculostomy through a coronal burr hole. This permits rapid control of the symptomatic hydrocephalus without the need for ventricular drainage or shunting. During the same procedure, the posterior third ventricular neoplasm can be biopsied under direct vision, and samples of cerebrospinal fluid taken for tumor markers. Formal tumor removal is conducted electively through an infratentorial supracerebellar exposure.

This technique was used successfully to treat two children with hydrocephalus and papilledema secondary to large pineal region neoplasms. In each case, headache resolved and papilledema improved after third ventriculostomy. The pathologic diagnosis was known prior to the primary tumor resection. One patient had an epidermoid, the other a mixed germ cell tumor. Both patients subsequently underwent uncomplicated elective total tumor resection.

This management plan allows rapid, safe, and effective treatment of the obstructive hydrocephalus that occurs with pineal tumors, and eliminates the need for external ventricular drainage or shunting. For patients in whom total tumor resection is not possible, third ventriculostomy provides control of hydrocephalus and allows for safe tissue diagnosis with minimal invasiveness. In some cases, for example low-grade tectal gliomas, third ventriculostomy may be all that is necessary in terms of treatment. Neuroendoscopy contributes to the safe, minimally invasive management of patients with neoplasms of the pineal region and symptomatic obstructive hydrocephalus.

20. Treatment Results for 149 Medulloblastoma Patients from One Institution

Y. Khafaga, A. E. Kandil, A. Jamshed, M. Hassounah, E. DeVol, A. J. Gray (Riyadh, Saudi Arabia)

One hundred forty-nine patients from 1975-1991 with histologically confirmed medulloblastoma were accepted for treatment with curative intent. There were 6 postoperative deaths and 10 patients planned for radiotherapy treatment failed to complete the prescribed course. One hundred thirtythree patients completed a course of radiotherapy after surgery. Adjuvant chemotherapy was not used routinely (6 patients only). Tumors were staged retrospectively according to the Chang staging system. There were no T1 patients. 32 patients had T2 tumors, 76 were T3, and 29 were T4. The T stage could not be allocated in 12 patients. Ninety-nine patients required a shunting procedure either pre- or postoperatively. Forty-six patients had complete resection of tumor, 91 had incomplete resection, and 6 patients had biopsy only. The extent of resection could not be determined in 6 patients. The median radiation dose for the whole brain was 34 Gy; spine, 32.5 Gy; and posterior fossa, 52.8 Gy. Actuarial survival for the whole group of 149 patients was 53% at 5 years and 38% at 10 years. On univariate analysis, patients with T2 tumors did significantly better as compared to patients with T3 and T4 tumors. Survival of patients who had clinical and radiological complete resection of tumor at surgery was significantly better than patients with incomplete tumor removal. The presence of a VP shunt had a significantly negative impact on survival.

On multivariate analysis, the only significant prognostic factor was the presence of a VP shunt in patients with T2 tumors.

T stage, VP shunt, and extent of surgery were important prognostic factors. In this study, radiation doses of more than 50 Gy to the posterior fossa and 30 Gy to the craniospinal axis resulted in improved survival.

21. Brain Stem Gliomas — Possibilities and Limits of Surgical Treatment

Siegfried Vogel, MD, Christiane May, MD (Berlin, Germany)

A series of 167 patients with brain stem gliomas diagnosed and surgically treated in between 1981 and 1990 has been reviewed. The entire group consists of 33 chiasmatic gliomas, 62 thalamic gliomas, and 72 intrinsic gliomas of the mesencephalon, pons, and medulla oblongata. They had been diagnosed either by computerized tomography or magnetic resonance imaging. Especially depending on morphology in neuroimaging, but also on the duration of preoperative symptoms, on the clinical condition of the patient but also on the intraoperative histology, only decompression, biopsy, partial or in many cases radical removal had been performed. Histology was obtained in 166 cases. Sixty-six percent were low-grade astrocytomas. In a follow-up period of 2 to 12 years, 59% of the entire group are alive. Mortality of patients with malignant gliomas was 73% but only 23% of those patients with benign gliomas. In conclusion primary, radical excision of benign gliomas of the brain stem is recommended because surgery may be accomplished with low mortality and morbidity. But we also conclude that in malignant gliomas of the brain stem there is only an indication for decompression, biopsy, or, at most, an extended biopsy.

The American Association of Neurological Surgeons

Section on Pediatric Neurological Surgery

1995 Membership Roster

Abbasy, Munir H., MD 80 Congress St. Springfield, MA 01104 Active Member Aronin, Patricia A., MD 521 Dubuar St. Northville, MI 48167 Active Member Benzel, Edward C., MD Univ. of New Mexico/Neuro. 2211 Lomas Blvd., N.E. Albuquerque, NM 87131 Active Member

Albright, A. Leland, MD Children's Hosp. Pittsburgh 3705 5th Ave./Neuro. Pittsburgh, PA 15213 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, Mitchel S., MD Univ. of Washington Med. Ctr. 1959 N.E. Pacific St., RI-20 Seattle, WA 98195 Active Member

Altenau, Lance, MD 501 Washington Suite #700 San Diego, CA 92103 Active Member Bailey, Walter L., MD Doctors Professional Bldg. 280 N. Smith Ave., Suite #234 St. Paul, MN 55102 Active Member Berger, Thomas S., MD Mayfield Neurological Inst. 506 Oak St. Cincinnati, OH 45219 Active Member

Amacher, A. Loren, MD Geisinger Clinic Dept. of Neuro. Danville, PA 17822 Active Member Balis, Gene A., MD 3000 E. Fletcher Ave. Suite #340 Tampa, FL 33613-4645 Active Member Black, Peter M., MD PhD Children's Hosp./Neuro. 300 Longwood Ave. Boston, MA 02115 Active Member

Amador, Luis V., MD 1440 Veteran Ave., #336 Los Angeles, CA 90024 Lifetime Member Barrer, Steven J., MD 1584 Old York Rd. Abington, PA 19001 Active Member Boggs, John Scott, MD 1820 Barrs St. Suite #104 Jacksonville, FL 32204 Active Member

Anderson, Jim D., MD Redwood City Offices 1150 Veterans Blvd. Redwood City, CA 94063 Active Member Bartkowski, Henry M., MD Neurological Assoc. 931 Chatham Ln. Columbus, OH 43221 Active Member Boop, Frederick A., MD Arkansas Children's Hosp. 800 Marshall St./Neuro. Little Rock, AR 72202 Active Member

Andrews, Brian T., MD 2100 Webster St. Suite #521 San Francisco, CA 94115 Active Member Beatty, Robert M., MD 8919 Parallel Pkwy. Suite #455 Kansas City, KS 66112 Active Member Bressler, Bruce C., MD 720 S. Van Buren Green Bay, WI 54301 Active Member

Arkins, Thomas J., MD Neurosurgical Associates 60 Temple St. New Haven, CT 06510 Active Member Bell, William O., MD Carolina Neuro. Assoc., PA 2810 Maplewood Ave. Winston-Salem, NC 27103 Active Member Brown, Jeffrey Alan, MD Med. Coll. of Ohio Dept. of Neuro. Toledo, OH 43699-0008 Active Member Bruce, Derek A., MD 1935 Motor St.

Dallas, TX 75235-7794 Active Member Cogen, Philip Harry, MD PhD

Univ. of Chicago/Neuro. 5841 S. Maryland, MC-4051 Chicago, IL 60637 Dorsen, Michael, MD 2905 San Gabriel, #310 Austin, TX 78705 Active Member

Buchheit, William A., MD

1015 Chestnut St. Suite #1400 Philadelphia, PA 19107 Active Member Cohen, Alan R., MD

Active Member

Case Western Reserve Univ. 11100 Euclid Ave./Neuro. Cleveland, OH 44106 Active Member Drake, James M., MD

Hosp. for Sick Children 555 University Ave., #1504-D Toronto, ON M5G 1X8 Canada Active Member

Cahan, Leslie D., MD

1505 N. Edgemont St. Neuro./Rm. #4141 Los Angeles, CA 90027 Active Member Coulon, Richard A., Jr. MD

Ochsner Clinic/Neuro. 1514 Jefferson Hwy. New Orleans, LA 70121 Active Member Duhaime, Ann-Christine, MD

Child. Hosp. of Philadelphia 34th & Civic Ctr. Blvd./Neuro. Philadelphia, PA 19104 Active Member

Canady, Alexa Irene, MD

Children's Hosp. of Michigan 3901 Beaubien Detroit, MI 48201 Active Member Crone, Kerry R., MD

Children's Hosp. Med. Ctr. 3333 Burnet Ave. Cincinnati, OH 45229-3039 Active Member Duncan, Charles Cecil, MD

Yale Univ. Sch. of Med./Neuro. P.O. Box 208039 New Haven, CT 06520-0839 Active Member

Carmel, Peter W., MD

New Jersey Med. Sch./Neuro. 90 Bergen St., Suite #7300 Newark, NJ 07103-2499 Active Member Dagi, T. Forcht, MD

993 F. Johnson Ferry Rd. Suite #100 Atlanta, GA 30342 Active Member Duncan, John A., III MD

2 Dudley St. Suite #530 Providence, RI 02905 Active Member

Chapman, Paul H., MD

Massachusetts Gen. Hosp. Fruit St. Boston, MA 02114 Active Member Davidson, Robin I., MD

Univ. Massachusetts Med. Ctr. 55 Lake Ave., N./Neuro. Worcester, MA 01655 Active Member Dunn, Mary E., MD 280 N. Smith Ave., #234 St. Paul, MN 55102 Active Member

Cheek, William R., MD

3009 Robinhood Houston, TX 77005 Lifetime Member Davis, Dudley H., MD Mavo Clinic/Neuro.

200 First St., S.W.
Rochester, MN 55905
Active Member

Edwards, Michael S. B., MD UCSF/Div. of Ped. Neuro. 533 Parnassus Ave.. U-126

533 Parnassus Ave., U-126 San Francisco, CA 94143 Active Member

Choux, Maurice, MD

Hopital 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 Eisenberg, Howard M., MD

Div. of Neuro. 22 S. Greene St., Rm. S12D-10 Baltimore, MD 21201 Active Member Epstein, Mel H., MD 2 Dudley St. Suite #505

Providence, RI 02905 Active Member

Erasmus, Mark D., MD

522 Lomas Blvd., N.E. Albuquerque, NM 87102 Active Member

Faillace, Walter J., MD

Univ. Med. Ctr. 653 W. Eighth St. Jacksonville, FL 32209 Active Member

Fell, David A., MD

Neurological Surg. Inc. 6767 S. Yale Tulsa, OK 74136-3303 Active Member

Fischer, Edwin G., MD

110 Francis St Suite #3B Boston, MA 02215 Active Member

Flannery, Ann Marie, MD

Med. Coll. of Georgia 1120 15th St./Neuro. Augusta, GA 30912-4010 Active Member

Foltz, Eldon L., MD UCI Med. Ctr./Neuro.

P.O. Box 14091 Orange, CA 92613-4091 Active Member

Freed, Michael H., MD

825 Washington St. Suite #100 Norwood, MA 02062 Active Member

French, Kathleen B., MD

3020 Hamaker Ct. Suite B104 Fairfax, VA 22031 Active Member

Fried, Arno H., MD

New York Med. Coll./Neuro. Munger Pavilion Valhalla, NY 10595 Active Member

Gahm, Norman H., MD

100 Retreat Ave. Suite #705 Hartford, CT 06106 Active Member

Galicich, Joseph H., MD

P.O. Box 276 Alpine, NJ 07620 Active Member

Gamache, Francis W., Jr. MD

Aitken Neurosci Inst 523 E. 72nd St. New York, NY 10021 Active Member

George, Richard E., Jr. MD

3506 21st St., #400 Lubbock, TX 79410-1200 Active Member

Godersky, John C., MD

2841 De Barr Rd. Suite #34 Anchorage, AK 99508 Active Member

Goodrich, James T., MD PhD

Dept. of Neuro./Montefiore 111 E. 210th St. Bronx, NY 10467 Active Member

Goumnerova, Liliana C., MD

Children's Hosp.-Bader 3 300 Longwood Ave. Boston, MA 02115 Active Member

Greene, Clarence S., Jr, MD

101 The City Dr., S., #313 Orange, CA 92668-3201 Active Member

Guido, Laurance J., MD

174 Hubbell Ln. Fairfield, CT 06430 Active Member

Gutierrez, Francisco A., MD

707 N. Fairbanks Ct. Suite #911 Chicago, IL 60611 Active Member

Haase, Jens, MD

Aalborg Hosp./Neuro. Aalborg, 9100 Denmark Active Member

Hahn, Joseph F., MD

Cleveland Clinic 9500 Euclid Ave., S80 Cleveland, OH 44195 Active Member

Hahn, Yoon Sun, MD

Christ Hosp, & Med. Ctr. 4440 W. 95th St., Suite #477S Oak Lawn, IL 60453 Active Member

Haines, Stephen J., MD

Box 96 Mayo 420 Delaware St., S.E. Minneapolis, MN 55455 Active Member

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

Hammargren, Lonnie L., MD 3196 S. Maryland Pkwy. Las Vegas, NV 89109 Active Member

Hammock, Mary Kathryn, MD 8650 Sudley Rd. Suite #200 Manassas, VA 22110 Active Member

Handler, Michael H., MD 1010 E. 19th St. Suite #605, Tammen Hall Denver, CO 80218 Active Member

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

Harwood-Nash, Derek C., MD Hosp. for Sick Children Neuro. Radiologist Toronto, ON M5G 1X8 Canada Associate Member

Hawkins, John C., III MD 2545 Riverside Ave. Jacksonville, FL 32204 Active Member

Heafner, Michael D., MD 1010 Edgehill Rd., N. Charlotte, NC 28207 Active Member Heffez, Dan S., MD 2515 N. Clark St. Suite #800 Chicago, IL 60614 Active Member

Hellbusch, Leslie C., MD 8905 Douglas Ct. Omaha, NE 68114 Active Member

Hemmy, David C., MD 20611 Watertown Rd. Suite J Waukesha, WI 53186 Active Member

Hendee, Robert W., Jr. MD 4710 Lookout Mtn. Cove Austin, TX 78731 Active Member

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

Hoffman, Harold J., MD Hosp. for Sick Children 555 University Ave., #1504 Toronto, ON M5G 1X8 Canada Active Member

Holbrook, Thomas J., Jr. MD 3330 Medical Park Rd. Suite #310 Columbia, SC 29203 Active Member

Hollenberg, Robert D., MD McMaster Univ. Med. Ctr. Dept. of Surg./Rm. #4U4 Hamilton, ON L8N 3Z5 Canada Active Member Hudgins, Roger, MD 5455 Meridan Mark Rd. Suite #540 Atlanta, GA 30342 Active Member

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

James, Hector E., MD 7930 Frost St. Suite #304 San Diego, CA 92123 Active Member

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

Johnson, Dennis L., MD Milton Hershey Med. Ctr. P.O. Box 850/Div. of Neuro. Hershey, PA 17033 Active Member

Johnson, John K., MD Greenville Neuro. Group 27 Memorial Medical Dr. Greenville, SC 29605 Active Member

Johnson, Martin, MD 2800 N. Vancouver Suite #106 Portland, OR 97227 Active Member

Johnson, Mary M., MD 5555 Peachtree Dunwoody Suite #337 Atlanta, GA 30342 Active Member Jones, Robert F. C., MD 21 Norfolk St. Paddington, NSW 2021 Australia

International Member

Joseph, Allen S., MD 7777 Hennessy Blvd.

Suite #10000 Baton Rouge, LA 70808

Active Member

Kalsbeck, John E., MD

Riley Children's Hosp. 702 Barnhill Dr. Indianapolis, IN 46202-5200

Active Member

Kanev, Paul M., MD

256 S. Van Pelt St. Philadelphia, PA 19103-4813 Active Member

Kasoff, Samuel S., MD New York Med. Coll./Neuro. Munger Pavillion Rm. #329

Valhalla, NY 10595 Active Member

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

Active Member

Active Member

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

Kestle, John R. W., MD B.C.'s Children Hosp. 4480 Oak St./Dept. of Neuro. Vancouver, BC V6H 3V4 Canada Active Member

Klein, David M., MD 690 Fearrington Post Pittsboro, NC 27312 Active Member

Knierim, David S., MD Director Pediatric Neuro. Loma Linda Univ Childrens Hosp Loma Linda, CA 92354

Active Member

Kosnik, Edward J., MD

Chatham Village Prof. Bldg. 931 Chatham Ln. Columbus, OH 43221 Active Member

Kril, Merv. MD 158 Oakdale St.

Redwood City, CA 94062 Active Member

Laurent, John P., MD Clinical Care Ctr., MC 3-3435

6621 Fannin, Suite #950 Houston, TX 77030 Active Member

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

Active Member

Lipow, Kenneth I., MD 267 Grant St. Bridgeport, CT 06610 Active Member

Loeser, John D., MD UWMC-Roosevelt 4245 Roosevelt Way, N.E. Seattle, WA 98105 Active Member

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

Longo-Cordero, Rafael, MD Calle Rochester 911 Univ Gardens Rio Piedras, PR 00927 Active Member

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

Luerssen, Thomas G., MD James Whitcomb Riley Hosp. One Children's Square Indianapolis, IN 46202

Magid, Gail A., MD 1661 Soquel Dr., F Santa Cruz, CA 95065 Active Member

Active Member

Magram, Gary, MD 4106 Kennett Pike Greenville, DE 19807 Active Member

Manwaring, Kim Herbert, MD Phoenix Children's Hosp. 909 E. Brill St. Phoenix, AZ 85006 Active Member

Mapstone, Timothy B., MD Div. of Neuro./CHOA 1600 7th Ave., S., Ste. ACC400 Birmingham, AL 35233 Active Member

Marlin, Arthur E., MD

Methodist Plaza 4499 Medical Dr., Suite #397 San Antonio, TX 78229 Active Member

Mawk, John R., MD

2230 N.W. Pettygrove St. Portland, OR 97210-2659 Active Member

McAllister, James P., Il PhD

Cleveland Clinic Foundation 9500 Euclid Ave./Neuro. Cleveland, OH 44195 Associate Member

McCallum, Jack E., MD

800 8th Ave. Suite #220 Fort Worth, TX 76104-2619 Active Member

McComb, J. Gordon, MD

1300 N. Vermont Ave. Suite #906 Los Angeles, CA 90027 Active Member

McLanahan, C. Scott, MD

Charlotte Neuro. Assoc. 1010 Edgehill Rd., N. Charlotte, NC 28207 Active Member

McLaurin, Robert L., MD

250 William Howard Taft Rd. Suite #210 Cincinnati, OH 45219-2664 Active Member

McLone, David Gordon, MD PhD

Children's Mem. Hosp. 2300 Children's Plaza, Box 28 Chicago, IL 60614 Active Member

Meacham, William F., MD

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

Mealey, John, Jr. MD

Indiana Univ. Med. Ctr. 545 Barnhill, Emerson #139 Indianapolis, IN 46202 Lifetime Member

Menezes, Arnold H., MD

Univ. of Iowa Hosp. Dept. of Neuro. Iowa City, IA 52242 Active Member

Meyer, Glenn A., MD

Med. Coll. of Wisconsin 9200 W. Wisconsin Ave. Milwaukee, WI 53226 Active Member

Michelsen, W. Jost, MD

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

Milhorat, Thomas H., MD

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

Miller, Clinton F., MD

Coastal New Hampshire Neuro. 330 Borthwick Ave., Suite #108 Portsmouth, NH 03801 Active Member

Moiel, Richard H., MD

3656 Ella Lee Ln. Houston, TX 77027-4105 Lifetime Member

Montes, Jose L., MD

Montreal Children's Hosp. 2300 Tupper St./Rm. C-811 Montreal, PQ H3H 1P3 Canada Active Member

Montoya, German, MD

1801 Cook Ave. Orlando, FL 32806 Active Member

Morrison, Glenn, MD

Miami Children's Hosp. 3200 S.W. 60 Ct., Suite #301 Miami, FL 33156 Active Member

Moss, S. David, MD

Phoenix Children's Hosp. 909 E. Brill St. Phoenix, AZ 85006 Active Member

Moyes, Peter D., MD

2210 W. 40th
Apt. #303
Vancouver, BC V6M 1W6
Canada
Active Member

Muakkassa, Kamel F., MD

130 W. Exchange St. Akron, OH 44302 Active Member

Muhlbauer, Michael S., MD

930 Madison Ave. Suite #600 Memphis, TN 38103 Active Member

Murtagh, Frederick, MD

Univ. of Pennsylvania Hosp. 3400 Spruce St. Philadelphia, PA 19104 Lifetime Member Myles, Terence, MD 1820 Richmond Rd., S.W. Calgary, AB T2T 5C7 Canada Active Member

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

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 Active Member

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

Nijensohn, Daniel E., MD 340 Capitol Ave. Bridgeport, CT 06606 Active Member

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

O'Brien, Mark Stephen, MD 1900 Century Blvd. Suite #4 Atlanta, GA 30345 Active Member Oakes, W. Jerry, MD Children's Hosp. of Alabama Suite #400 Birmingham, AL 35233 Active Member

Page, Larry Keith, MD 13845 S.W. 73rd Ct. Miami, FL₂33158 Active Member

Pang, Dachling, MD Univ. of California, Davis 2516 Stockton Blvd. Sacramento, CA 95817 Active Member

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

Park, Tae Sung, MD St. Louis Children's Hosp. 1 Children's PI., Rm. #1546 St. Louis, MO 63110 Active Member

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

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

Piatt, Joseph H., MD 3181 S.W. Sam Jackson Park Rd. Div. of Neuro. (L-472) Portland, OR 97201-3098 Active Member Pittman, Hal Watson, MD Barrow Neurological Inst. 2910 N. 3rd Ave. Phoenix, AZ 85013 Active Member

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

Pudenz, Robert H., MD 574 Garfield Ave. South Pasadena, CA 91030 Active Member

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

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

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

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

Reynolds, Arden F., Jr. MD Magan Med. Clinic 420 W. Rowland St. Covina, CA 91723 Active Member Roberts, Theodore S., MD Univ. of Washington/Neuro. CH-50, P.O. Box 5371 Seattle, WA 98195

Active Member

Robinson, Walker L., MD Univ. of Maryland Hosp. 22 S. Greene St./Neuro.

Baltimore, MD 21201 Active Member

Rosenblum, Bruce R., MD 29 Route 34 N. Suite #103 Colts Neck, NJ 07722 Active Member

Rosenthal, Alan, MD Long Island Neuro. Assoc. 410 Lakeville Rd. New Hyde Park, NY 11042 Active Member

Rothman, Allen S., MD 1160 Fifth Ave. Suite #106 New York, NY 10029 Active Member

Ruzicka, Petr O., MD Children's Hosp. of New Jersey 15 S. 9th St. Newark, NJ 07107 Active Member

Sanford, Robert A., MD Semmes-Murphey Clinic 930 Madison Ave., Suite #600 Memphis, TN 38103 Active Member

Sato, Osamu, MD Dept. of Neuro. Bo-Sei Dai Isehara Kanagawa, 259-11 Japan International Member Saunders, Richard, MD
Dartmouth-Hitchcock Med. Ctr.
1 Medical Ctr. Dr./Neuro.
Lebanon, NH 03756
Active Member

Scarff, Timothy B., MD 33 S.W. Upper Riverdale Rd. Suite #25 Riverdale, GA 30274 Active Member

Schiff, Steven J., MD Children's National Med. Ctr. 111 Michigan Ave., N.W./Neuro. Washington, DC 20010 Active Member

Schut, Luis, MD Children's Hosp. Philadelphia 34th and Civic Ctr. Blvd. Philadelphia, PA 19104 Active Member

Scott, R. Michael, MD Boston Neuro. Foundation 300 Longwood Ave., Bader 319 Boston, MA 02115 Active Member

Seljeskog, Edward L., MD 2805 Fifth St., S. Suite #110 Rapid City, SD 57701-7306 Active Member

Selker, Robert G., MD Western Pennsylvania Hosp. 4800 Friendship Ave./Neuro. Pittsburgh, PA 15224 Active Member

Shallat, Ronald F., MD 3000 Colby St. Suite #101 Berkeley, CA 94705 Active Member Shapiro, Kenneth N., MD 1935 Motor St. Dallas, TX 75235-7794 Active Member

Shillito, John, Jr. MD 595D Fearrington Post Pittsboro, NC 27312 Active Member

Simmons, James C., MD 190 Grove Park Rd. Memphis, TN 38117 Lifetime Member

Sklar, Frederick H., MD 1935 Motor St. Dallas, TX 75235-7794 Active Member

Smith, Harold P., MD 300 20th Ave., N., #506 Nashville, TN 37203-2115 Active Member

St. Louis, Phillip G., MD 1801 Cook Ave. Orlando, FL 32806-2913 Active Member

Stein, Sherman Charles, MD Dept. of Surg. 3 Cooper Plaza, Suite #411 Camden, NJ 08103 Active Member

Steinbok, Paul, MD B.C. Children's Hosp. I C 4480 Oak St. Vancouver, BC V6H 3V4 Canada Active Member Storrs, Bruce B., MD Med. Univ. of South Carolina 171 Ashley Ave., 428-CSB Charleston, SC 29425-2272 Active Member

Stringer, Douglas L., MD 2011 Harrison Ave. Panama City, FL 32405 Active Member

Stringer, Merle Preston, MD 2011 Harrison Ave. Panama City, FL 32405 Active Member

Sukoff, Michael H., MD 17602 17th St., #102-118 Tustin, CA 92680-1961 Lifetime Member

Susen, Anthony F., MD RR 3 Box 140-J Burgess, VA 22432-9801 Lifetime Member

Sutton, Leslie N., MD Child. Hosp. of Philadelphia 34th & Civic Center Blvd. Philadelphia, PA 19104 Active Member

Taekman, Michael S., MD 3000 Colby Suite #101 Berkeley, CA 94705 Active Member

Tew, John M., Jr. MD Mayfield Neuro. Inst. 506 Oak St. Cincinnati, OH 45219 Active Member Tomita, Tadanori, MD Children's Mem. Hosp. 2300 Children's Plaza Chicago, IL 60614 Active Member

Tulipan, Noel, MD 8533 McCrory Ln. Nashville, TN 37221 Active Member

Turner, Michael S., MD 1801 N. Senate Blvd. Suite #535 Indianapolis, IN 46202 Active Member

Uscinski, Ronald H., MD 3301 Woodburn Rd., #209 Annandale, VA 22003 Active Member

Venes, Joan L., MD 27517 Via Montoya San Juan Capistrano, CA 92675 Active Member

Ventureyra, Enrique C., MD 401 Smyth Rd. Ottawa, ON K1H 8L1 Canada Active Member

Vries, John Kenric, MD Univ. of Pittsburgh 217 Victoria Bldg. Pittsburgh, PA 15261 Active Member

Wald, Steven L., MD 10 Grove Ln. Shelburne, VT 05482 Active Member Waldman, John B., MD Albany Med. Coll. Div. of Neuro., A-61 Albany, NY 12208 Active Member

Walker, Marion L., MD Primary Children's Med. Ctr. 100 N. Medical Dr. Salt Lake City, UT 84113-1100 Active Member

Walsh, John Willson, MD 6411 Fannin, Box 61 Houston, TX 77030 Active Member

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

Ward, John D., MD Virginia Commonwealth Univ. Neuro., MCV Station, Box 631 Richmond, VA 23298 Active Member

Weiss, Martin H., MD LAC/USC Med. Ctr., Box 786 1200 N. State St. Los Angeles, CA 90033 Active Member

Wernick, Shelley, MD 2350 W. Villard Ave. Room #101 Milwaukee, WI 53209 Active Member

White, Robert Joseph, MD PhD MetroHealth Med. Ctr. 2500 MetroHealth Dr. Cleveland, OH 44109-1998 Active Member Willman, Philip J. A., MD 3006 S. Alameda St. Corpus Christi, TX 78404 Active Member Woodward, Meredith V., MD 7130 N. Sharon Ave., #100 Fresno, CA 93720 Active Member Zampella, Edward J., MD 10 Parrott Mill Rd. P.O. Box 808 Chatham, NJ 07928 Active Member

Winfield, Jeffrey A., MD PhD Hill Med. Ctr. 1000 E. Genesse St., Ste. #202 Syracuse, NY 13210 Active Member Yamada, Shokei, MD Loma Linda Univ. Sch. of Med. Rm. #2539/Neuro. Loma Linda, CA 92350 Active Member Zanetti, Paul Henry, MD 3006 S. Alameda St. Corpus Christi, TX 78404 Active Member

Winston, Ken R., MD 1950 Ogden, B467 Denver, CO 80218 Active Member Zakalik, Karol, MD Wm. Beaumont Hosp. 3535 W. 13 Mile Rd., Suite 504 Royal Oak, MI 48073 Active Member **Zavala, L. Manuel, MD** 800 Third St. Marysville, CA 95901 Active Member

