The American Association of Neurological Surgeons

1998 Annual Meeting

Pennsylvania Convention Center
Philadelphia, Pennsylvania

April 25–30, 1998

(by registration only)
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Edward R. Laws, Jr., MD, FACS, of Charlottesville, Virginia, is the 65th President of The American Association of Neurological Surgeons.

An active member of the AANS since 1975, Dr. Laws has held such leadership positions as Treasurer (1992-95) and member of the Board of Directors (1989 to 1995). He currently serves as the Neurosurgery Regent to the American College of Surgeons Board of Regents and as a member of the Executive Council of the AANS Research Foundation.

Dr. Laws currently serves as Professor of Neurological Surgery and Medicine at the University of Virginia. He is widely known for his expertise in pituitary surgery, and has published extensively on pediatric surgery, brain tumor surgery, and epilepsy surgery.

Dr. Laws earned his bachelor’s degree in American Civilization from Princeton University. He received his medical degree from Johns Hopkins University School of Medicine, where he also completed his surgical internship and residency.

A prolific author and researcher, Dr. Laws has written more than 200 articles for peer-reviewed journals, seven books, 89 book chapters, and 145 abstracts and editorials. He also served as Editor of the scientific journal, Neurosurgery (1987-92) and currently is a member of the Editorial Board of the Journal of Neurosurgery. A dedicated educator as well, Dr. Laws has lectured and completed visiting professorships at medical schools and clinics all over the world.

In addition to his volunteer activities with the AANS, Dr. Laws has taken on active leadership roles in several other professional societies, including serving as President of the Congress of Neurological Surgeons (1984) and Vice President of The World Federation of Neurosurgical Societies (present). He was a founding member of the American Pituitary Association, the Brain Surgery Society, the International Society of Pituitary Surgeons, and the Pituitary Society. Dr. Laws also holds memberships in the American Brain Tumor Association, the American College of Surgeons, the American Medical Association, and the American Epilepsy Society.

Dr. Laws and his wife, Margaret, are the parents of four daughters.
Each year at the Annual Meeting of The American Association of Neurological Surgeons (AANS), the outgoing President is presented with a special memento of his year in office (the “Cushing Cigarette Box”) representing more than a half century of neurosurgical tradition.

The sterling silver Cushing Cigarette Box was originally presented to Harvey Cushing, M.D., by the surgical staff at Peter Bent Brigham Hospital on April 14, 1931, commemorating Dr. Cushing’s 2,000th verified intracranial tumor operation. Beginning a cherished tradition in 1959, Dr. Cushing’s nephew, E.H. Cushing, M.D., presented the Cigarette Box to the AANS President and asked that it be passed on to each succeeding President as a symbol of the Association’s highest office.

Reflecting the Association’s increased concern for the preservation of this special artifact, the Cigarette Box was entrusted to the Yale Medical School Library for safe-keeping in 1961. An exact replica was made and the tradition was continued. Each year, the name of the AANS President was engraved on this replica and it was passed down, from President to President.

In 1985, it was decided the replica (displaying the names of every President from Bronson S. Ray to Byron C. Pevehouse) should no longer be circulated as it also had become historically significant. Consequently, each year thereafter, the Association has presented a faithful replica of the Cushing Cigarette Box to the outgoing AANS President for his service to the Association.

In July of 1990, the original Cushing Cigarette Box was returned to the Association by Yale University. The original and the E.H. Cushing replica (containing the names of the successive AANS Presidents who possessed it during their terms in offices) have been placed on permanent display at the AANS National Office in Park Ridge, Illinois.

AANS PRESIDENTS

1932-33 William P. Van Wagenen, MD
1933-34 John F. Fulton, MD
1934-35 R. Glen Spurling, MD
1935-36 Merrill C. Sosman, MD
1936-37 Kenneth G. McKenzie, MD
1937-38 Temple Fay, MD
1938-39 Louise Eisenhardt, MD
1939-40 R. Eustace Semmes, MD
1940-41 Cornelius G. Dyke, MD
1941-42 Tracy J. Putnam, MD
1942-43 Eric Oldberg, MD
1943-44 Edgar F. Fincher, MD
1944-46 Franc D. Ingraham, MD
1946-47 Frank R. Teachener, MD
1947-48 Cobb Pilcher, MD
1948-49 Winchell McK. Craig, MD
1949-50 Frank Turnbull, MD
1950-51 W. Edward Chamberlain, MD
1951-52 Paul C. Bucy, MD
1952-53 William J. German, MD
1953-54 Edgar A. Kahn, MD
1954-55 Harry Wilkins, MD
1955-56 Frederic Schreiber, MD
1956-57 Leo M. Davidoff, MD
1957-58 Howard A. Brown, MD
1958-59 Bronson S. Ray, MD
1959-60 James L. Poppen, MD
1960-61 J. Grafton Love, MD
1961-62 Leonard T. Furlow, MD
1962-63 David L. Reeves, MD
1963-64 Barnes Woodhall, MD
1964-65 Frank H. Mayfield, MD
1965-66 Francis Murphey, MD
1966-67 Eben Alexander, Jr., MD
1967-68 Henry G. Schwartz, MD
1968-69 Donald D. Matson, MD
1969-70 A. Earl Walker, MD
1970-71 Collin S. MacCarty, MD
1971-72 Guy L. Odom, MD
1972-73 William F. Meacham, MD
1973-74 Lyle A. French, MD
1974-75 Richard C. Schneider, MD
1975-76 Richard L. DeSausssure, MD
1976-77 Lester A. Mount, MD
1977-78 Charles G. Drake, MD
1978-79 Donald F. Dohn, MD
1979-80 W. Eugene Stern, MD
1980-81 Robert B. King, MD
1981-82 W. Kemp Clark, MD
1982-83 Frank R. Wrenn, MD
1983-84 Byron C. Pevehouse, MD
1984-85 Sidney Goldring, MD
1985-86 Russel H. Patterson, Jr., MD
1986-87 Robert G. Ojemann, Jr., MD
1987-88 Henry D. Garretson, MD
1988-89 George T. Tindall, MD
1989-90 Albert L. Rotroh, Jr., MD
1990-91 David L. Kelly, Jr., MD
1991-92 James T. Robertson, MD
1992-93 Merwyn Bagan, MD
1993-94 Julian T. Hoff, MD
1994-95 Edward L. Seljeskog, MD
1995-96 Sidney Tolchin, MD
1996-97 J. Charles Rich, MD
1997-98 Edward R. Laws, Jr., MD
ERIC WIESCHAUS, PhD
1998 CUSHING ORATOR

Eric Wieschaus, PhD, co-winner of the 1995 Noble Prize in Physiology or Medicine, is the 1998 Cushing Orator. Dr. Wieschaus will speak on “What Fly Genes Can Tell Us About How Human Embryos Develop.”

Currently, Dr. Wieschaus is the Squibb Professor of Molecular Biology at Princeton University and is studying the development of Drosophila in all its manifest implications. He earned his bachelor’s degree from the University of Notre Dame and did his Yale University doctoral study with Walter Gehring at the Biozentrum in Basel, Switzerland, before moving on to complete his post-doctoral work at the University of Zurich. His first independent research position was at the European Molecular Biology Laboratory in Heidelberg.

While in Heidelberg, Dr. Wieschaus and his colleague, Christiane Nusslein-Volhard, set out to identify and define, using mutant screens, the zygotic genes essential to the development of the Drosophila embryo. The two scientists interbred the flies and fed the flies mutagens in order to create lines of flies that had the same mutation. Ultimately, they established some 40,000 lines, in which some 18,000 genes harbored mutations that affected a fly’s survival.

Analyzing the mutations and noting mutation combinations, Wieschaus and Nusslein-Volhard were able to piece together the types of cellular decisions an embryo makes and discovered that only 139 of the 20,000 fly genes were important in determining body structure and function. They later learned that virtually all the genes they had identified also exist in humans. Wieschaus and Nusslein-Volhard earned the 1995 Nobel Prize for Physiology or Medicine for this work.

In his presentation, Dr. Wieschaus will discuss how these findings have influenced our view of development in vertebrates and humans. A decade ago, no one anticipated the degree to which human and fly genes are similar and in many cases it has been possible to identify human genes that, based on their DNA sequence, are direct homologues of genes that control specific steps in fly development. Mutations in many such human genes cause birth defects ranging from deafness to craniofacial abnormalities. Other genes identified in flies cause cell proliferation and cancer in humans when they are mutated or mis-expressed in adults. The similarity between human and fly genes and the potential to analyze gene function in fly embryos, provides a new approach to defining the relationship between these genes and their clinical manifestations.

The American Association of Neurological Surgeons is pleased to welcome Eric Wieschaus to the 1998 Annual Meeting. Dr. Wieschaus’ participation in the ranks of the esteemed Cushing Orators continues an ongoing tradition of excellence.
For more than 30 years, The American Association of Neurological Surgeons has sponsored the Annual Cushing Oration (named for Harvey Cushing, MD, universally recognized as the Father of Modern Neurosurgery). The Cushing Oration is traditionally presented by a contemporary philosopher who has clearly demonstrated that he or she has something of lasting importance and interest to impart to the neurosurgical community.

1965 Louise Eisenhardt, MD
1966 Philip Handler, MD
1967 William H. Stewart, MD
1968 R. Buckminster Fuller, PhD
1969 John S. Millis, PhD
1970 Edwin L. Crosby, MD
1971 Wilder Penfield, MD
1972 Robert Q. Marston, MD
1973 Wernher von Braun, PhD
1974 Malcolm Moos, PhD
1975 Paul W. McCracken, PhD
1976 Robert O. Egeberg, MD
1977 Eli Glinzberg, PhD
1978 C. Rollins Hranter, MD
1979 The Hon. Paul Rogers
1980 The Hon. Kingman Brewster
1981 Julius Axelrod, PhD
1982 Mortimer J. Adler, PhD
1983 Edmund D. Pellegrino, MD
1984 Robert M. Rosenthal, PhD
1985 Raymond E. Arvidson, PhD
1986 The Hon. Richard D. Lamm
1987 H. Ross Perot
1988 The Hon. Brian Dickson
1989 Theodore Cooper, MD, PhD
1990 President James Earl Carter, Jr.
1991 Yevgeny Yevtushenko
1992 Susan Eisenhower and Ronald Z. Sagdeev, PhD
1994 Beverly Sills
1995 General Colin L. Powell
1996 William F. Buckley, Jr.
1997 William J. Bennett, PhD
1998 Eric Wieschaus, PhD
Albert L. Rhoton, Jr., MD, of the University of Florida, is the 1998 recipient of the Cushing Medal, the highest honor granted by The American Association of Neurological Surgeons (AANS). He is being recognized for his many years of outstanding leadership and dedication to the field of neurological surgery.

Born in Parvin, Kentucky, Dr. Rhoton received his bachelor’s degree from The Ohio State University and earned his medical degree from Washington University in St. Louis, graduating with the highest academic standing in his class in 1959. He served his internship at Columbia-Presbyterian Medical Center in New York City, and completed his residency in neurological surgery at Washington University in St. Louis. Dr. Rhoton became board certified in 1969.

A consummate physician and educator, Dr. Rhoton has spent more than three decades teaching a younger generation about the practice of neurosurgery. He began his teaching career as an Instructor in neurological surgery at the University of Minnesota, Mayo Foundation (1966–1969). Following a three year appointment as Assistant Professor at Mayo, Dr. Rhoton accepted the position of Professor of Surgery at the University of Florida College of Medicine (1972–1978). In 1978, he became the R.D. Keene Family Professor of Neurological Surgery, a position he holds till today.

In addition to his academic appointments, Dr. Rhoton also serves as Chairman of the Department of Neurological Surgery at the University of Florida Health Center (since 1980).

With a natural flair for leadership, Dr. Rhoton has been elected President of numerous professional organizations, including the Congress of Neurological Surgeons (CNS) (1978), Florida Neurosurgical Society (1978), the AANS (1989), the Society of Neurological Surgeons (1993), North American Skull Base Society (1993), International Society for Neurosurgical Technology and Instrument Inventors (1995), and the International Interdisciplinary Congress on Craniofacial and Skull Base Surgery (1996–1998). In addition, he has served as Chairman of the AANS/CNS Joint Section on Cerebrovascular Surgery (1980), Chairman of the Medical Advisory Board of the Acoustic Neuroma Association (1991–present) and Delegate to the World Federation of Neurosurgical Societies (1986–present). Dr. Rhoton also has served on the Boards of Directors or Executive Committees of the CNS (1972–1979); Society of Neurological Surgeons (1976–1980); Cerebrovascular Section (1976–1982); American College of Surgeons, Board of Governors (1978–1984); AANS (1983–1986, 1987); American Board of Neurological Surgery (1985–1991); Acoustic Neuroma Associa-

Dr. Rhoton has published extensively throughout his career, authoring more than 200 scientific papers, as well as a book on microsurgical anatomy. He has also served on the editorial boards of such research journals as Neurosurgery (1977–1983), Journal of Microsurgery (1979–present), American Journal of Otology (1978–present), Surgical Neurology (1982–1987), and Neurological Research (1986–present).

In recognition of his many professional accomplishments, Dr. Rhoton has received dozens of awards throughout his career, including the American Heart Association, Florida Affiliate, Bronze Service Award; Alumni Achievement Award from the Washington University School of Medicine; the Distinguished Faculty Award from the University of Florida; and the Mayor’s Key to the City of Osaka, Japan. He also has served as the Honored Guest or been elected to Honorary Membership in neurosurgical societies in Asia, Africa, Europe and North and South America.

Dr. Rhoton and his wife, Joyce, have four children, all pursuing medical careers.

THE CUSHING MEDAL

The Harvey Cushing Medal, the highest honor The American Association of Neurological Surgeons can bestow upon a member, was established at the recommendation of President Lester Mount, MD, in 1976. Presented annually since 1977, the award recognizes an AANS member for distinguished service to the field of neurological surgery. The recipient is selected by the Board of Directors upon the recommendation of the Awards Committee.

CUSHING MEDALISTS

1977  Frank H. Mayfield, MD
1978  William H. Sweet, MD
1979  Henry G. Schwartz, MD
1980  Paul C. Bucy, MD
1981  Bronson S. Ray, MD
1982  W. James Gardner, MD
1983  Guy L. Odom, MD
1984  Eben Alexander, Jr. MD
1985  Francis Murphey, MD
1986  Lyle French, MD
1987  William F. Meacham, MD
1988  Charles G. Drake, MD
1989  Lester A. Mount, MD
1990  Robert B. King, MD
1991  William F. Collins, MD
1992  W. Eugene Stern, MD
1993  Sidney Goldring, MD
1994  Byron C. Pevehouse, MD
1995  Richard DeSaussure, MD
1996  Shelley N. Chou, MD, PhD
1997  Robert G. Ojemann, MD
1998  Albert L. Rhoton, Jr., MD
The American Association of Neurological Surgeons (AANS) is pleased to present Ronald D.G. McKay, PhD, with the 1998 Decade of the Brain Medal in recognition of his pioneering work in stem cell biology. His work has importance beyond the intrinsic academic interest in the properties of stem cells and strongly influences strategies for cell and gene based therapies for CNS disease.

Dr. McKay is the Chief of the Laboratory of Molecular Biology in the Basic Neuroscience Program of the National Institute of Neurological Disorders and Stroke. He has held this position since April 1993, when he moved from Massachusetts Institute of Technology, where he served as the Edward J. Poitras Associate Professor of Human Biology and Experimental Medicine in the Department of Brain and Cognitive Sciences (1984–1993). He also worked at Oxford University and at Cold Spring Harbor Laboratory in Cold Harbor Spring Harbor, New York. He received his doctorate for work in nucleic acid chemistry with Dr. Ed Southern at the University of Edinburgh.

In the first of his contributions to neurobiology, Dr. McKay showed that the nervous system was composed of very many molecularly distinct neuronal types. This fact has now been supported by additional studies but, at the time, these experiments had a strong impact because they were the first to use a powerful molecular technique to demonstrate the extraordinary biochemical complexity of the nervous system. His recent work has focused on the stem cells of the central nervous system.

In 1988, Dr. McKay provided the first clear proof that neuronal precursors could be identified. These studies have generated a series of important insights into the properties of stem cells in the developing and adult nervous system. They challenge the textbook view that the differences between brain regions are irreversibly imprinted on cells at the time of gastrulation. Rather, the results of his work add striking support to the conclusion that extracellular signals play a major role directing cell differentiation through the entire period of CNS development.

As a result of his research, Dr. McKay has been granted two patents, one for a Method for the Manipulation of Cell types in Eukaryotes and Nestin Expression as an Indicator of Neurepithelial Tumors.

Widely published, Dr. McKay is the author or co-author of 92 research articles. He also has served on the editorial boards of such major journals as

The work of his research group is widely recognized and Dr. McKay is a sought after speaker and conference organizer. He has spoken at scientific meetings throughout the United States, Europe, and Japan. He also serves as an advisor to the National Research Agency of Japan on Neuroscience at the Tsukuba Research Institute.

DECADE OF THE BRAIN MEDAL

President George Bush signed a resolution on July 17, 1990, proclaiming the decade beginning January 1, 1990, as the Decade of the Brain in order to enhance public awareness of the benefits to be derived from brain research. To commemorate this event, The American Association of Neurological Surgeons and the Congress of Neurological Surgeons (CNS), together, established an annual award designed to honor a distinguished neuroscientist for his or her contributions to brain research. The scientist is given a commemorative medal imprinted with the Decade of the Brain logo and invited to deliver a presentation at the Annual Meeting of either the AANS or CNS on a topic of his or her choice. The award presentation occurs in alternating years between the two organizations. In 1998, the AANS has the honor of awarding the Decade of the Brain Medal.

DECADE OF THE BRAIN MEDALISTS

1991 — Albert J. Aguayo, MD (CNS Annual Meeting)
1992 — Marcus E. Raichle, MD (AANS Annual Meeting)
1993 — Fred H. Gage, PhD (CNS Annual Meeting)
1994 — Eugene M. Johnson, Jr., PhD (AANS Annual Meeting)
1995 — Zach W. Hall, PhD (CNS Annual Meeting)
1996 — Dennis D. M. O’Leary, PhD (AANS Annual Meeting)
1997 — Congressman John Edward Porter (CNS Annual Meeting)
1998 — Ronald D. G. McKay, PhD (AANS Annual Meeting)
In recognition of his many years of dedication to neurosurgical science, medicine and the global community, Lee Finney, MD, of Great Falls, Montana, has been named the recipient of the 1998 Humanitarian Award of The American Association of Neurological Surgeons. Dr. Finney is being recognized for his extensive volunteer work overseas, providing badly needed neurosurgical care to patients in Honduras and assisting the development of the country’s first neurosurgical residency program.

Dr. Finney has been in private practice since 1978 in Montana. He earned his undergraduate degree from the University of Kansas and his medical degree from the University of Missouri. He did his neurosurgical residency at the University of Utah and became board certified in 1981. He has been an active member of the AANS since 1984 and the Congress of Neurological Surgeons since 1980.

In 1986, after discussing a case with a plastic surgeon colleague who had traveled to Honduras to perform cleft palate surgery, Dr. Finney made his first trip to that Central American country to shunt a young patient who had no way of obtaining this necessary surgery in his home country. For the next seven years, Dr. Finney traveled annually to the village of La Ceiba to perform various tumor, spine and other neurological surgeries and, in the process, became the community’s main source of neurosurgical care.

During a 5-month volunteer trip to Honduras in 1993, Dr. Finney was introduced to Herman Corletto, MD, a senior neurosurgeon at the Hospital Escuela in Tegucigalpa, the capital of Honduras. Hospital Escuela was in the process of developing the country’s first neurosurgical residency program and Dr. Corletto asked Dr. Finney to mentor the program. Since then, Dr. Finney has played an instrumental role in developing the Honduras neurosurgical program, making frequent pilgrimages to the hospital to teach both neurosurgeons and residents such techniques as transsphenoidal surgery and microvascular decompressions.

Dr. Finney has also been instrumental in convincing several other neurosurgeons including Marion Walker, MD, Roberto Heros, MD, Dan Kelly, MD, and Ron Wilson, MD, to make trips to Honduras to teach and perform neurological surgery.

Most recently, Dr. Finney played an essential role in securing transportation and medical care for craniopagus twins born in Honduras and then sur-
gically-separated by Dr. Walker in Salt Lake City, Utah. Impressed with Dr. Finney's efforts, the Foundation for International Education in Neurological Surgery and the International Committee of the Congress of Neurological Surgeons, have made a three-year commitment to further assist Honduras in developing its neurosurgical residency program.

Besides being a dedicated teacher, Dr. Finney has also spent endless hours securing badly-needed medical supplies and equipment for Hospital Escuela. To help increase the donation of supplies, Dr. Finney started Help Honduras, a not-for-profit organization. The goal of Help Honduras is to secure medical equipment and supplies, either new or those that are considered out-date in the United States, for the Honduran teaching hospital. He has also donated numerous books and scientific journals.

Dr. Finney is further hoping to increase the exchange of American neurosurgeons traveling to Honduras and the number of residents from Honduras coming to the United States to attend scientific meetings and expand their knowledge base.

In addition to his contributions to medicine and neurosurgery, Dr. Finney has also played an active role in the social arena of Honduras, Central America's poorest country. He and his wife are the prime supporters of the SOS Orphanage located outside of La Ceiba and consistently bring supplies, food and clothing to the orphanage. The Finney’s are godparents to three children in the orphanage and have found godparents for countless other children living there. This year, the Finneys will support a teacher who will make daily visits to the orphanage to teach English to the staff and children. As a result of his travels, Dr. Finney and his wife also have adopted three children - a 5-year old from Honduras and 4-year old twins from Paraguay.

Because of this sustained personal effort, and his recruitment of organized neurosurgical support, the future for Honduran neurosurgery and neurosurgical training there is exceedingly bright. In recognition of these accomplishments and his outstanding service to needy patients in Central America, the AANS is proud to honor Dr. Lee Finney with the 1998 Humanitarian Award.

HUMANITARIAN AWARD

At the recommendation of President Robert Ojemann, the Board of Directors established the Humanitarian Award in January, 1987. The award, presented annually in most years since, recognizes AANS members for activities outside the art and science of neurosurgery which bring great benefit to mankind. The recipient is selected by the Board of Directors on the recommendation of the Selection Committee. The Humanitarian Award distinguishes those members who have given time or talents selflessly to a charitable or public activity, and whose actions have brought honor to the specialty.

HUMANITARIAN AWARD RECIPIENTS

1987 — Courtland H. Davis, Jr. MD
1988 — Gaston Acosta-Rua, MD
1989 — Hugo V. Rizzoli, MD
1990 — A. Roy Tyrer, Jr., MD
1991 — George B. Udvarhelyi, MD
1992 — William H. Mosberg, Jr., MD
1993 — Manual Velasco-Suarez, MD
1994 — E. Fletcher Eyster, MD
1995 — Melvin L. Cheatham, MD
1997 — Robert J. White, MD
1998 — Lee Finney, MD
MARK J. KUBALA, MD
1998 DISTINGUISHED SERVICE AWARD

In honor of his many years of service to organized neurosurgery, the Officers of The American Association of Neurological Surgeons (AANS) are pleased to honor Mark J. Kubala, MD, with the 1998 Distinguished Service Award.

After receiving his bachelor’s degree in 1955 from the University of Texas, Dr. Kubala went on to earn his medical degree (with honors) from the University of Texas Medical Branch of Galveston in 1958. He served his internship at Herman Hospital and residency at the Baylor University College of Medicine. In 1966, following two years of military service at Williford Hall Hospital, Lackland Air Force Base, Texas, he entered private practice in Beaumont, Texas. He became board certified in 1968.

Dr. Kubala has been an Active member of the AANS since 1970, holding a variety of leadership positions, including service as a member of the Board of Directors (1990–1992), member of the Nominating Committee (1984–1986), and member of the Neurosurgical Guidelines and Outcomes Committee (1990–present).

In addition to his service with the AANS, Dr. Kubala has also been active in the Council for State Neurosurgical Societies (CSNS), serving as a delegate from Texas and as a member of the CSNS Medico-legal Committee (1986–present). He also has served in the leadership of the Congress of Neurological Surgeons, as a member of the Executive Committee (1982–1985) and of the Nominating Committee (1981–1982, 1985–1986). A dedicated spokesperson for neurosurgery, Dr. Kubala was a member of the Joint AANS/CNS Public Relations Committee (1984–1986) and the AANS Public Relations Committee (1988–1997). He has also served as President of the Texas Association of Neurological Surgeons (1977–1985) and, more recently, as President of the Texas Medical Association (1995–1996).

As a practicing neurosurgeon, Dr. Kubala demonstrated a special commitment to patients in the areas of trauma and injury prevention by offering his expertise to several voluntary organizations serving the public in this area. Foremost was his involvement with the THINK FIRST Foundation, neurosurgery’s National Head and Spinal Cord Injury Prevention Program. He served as the Foundation’s Secretary (1990–1992) and as the sponsoring physician for the Beaumont, Texas Chapter (1989–present). He also served as Neurosurgical Editorial Advisor for the National Safety Council (1985–1986) and as President of the Texas Head and Spinal Cord Injury Prevention Foundation (1989–present).
Dr. Kubala has also been an outspoken advocate for neurosurgery in the socioeconomic area, serving as Chairman of the Manpower Subcommittee of the Joint Socioeconomic Committee (JSEC) (1980–1981), Co-Chairman of JSEC (1982–1985), as Neurosurgical Representative to the Council of Medical Specialty Societies Task Force on Physician Manpower (1982–1983), and as Alternate Neurosurgical Delegate to the AMA House of Delegates (1994).

In recognition of his many years of dedicated service to the AANS, the neurosurgical community, and to patients, the Officers of the Association are pleased to honor Mark J. Kubala, MD, with the 1998 Distinguished Service Award.

RECIPIENTS OF THE DISTINGUISHED SERVICE AWARD

Roy W. Black — 1993
William A. Buchheit, MD — 1994
Charles Edwin Bracket, MD — 1995
Robert E. Florin, MD — 1996
Ralph and Ala Isham — 1996
Ernest W. Mack, MD — 1997
Mark J. Kubala, MD — 1998
Kamal Thapar, MD, is the recipient of the 1998 Van Wagenen Fellowship. Dr. Thapar is currently a neurosurgical resident in the Division of Neurosurgery at the University of Toronto. He was recently awarded a PhD in molecular biology with a minor in statistics and research design from the University of Toronto, completed under the direction of Kalman Kovacs, MD, PhD, DSc and Edward R. Laws, Jr., MD. Dr. Thapar earned his bachelor’s degree from Saint Mary’s University in Halifax, Nova Scotia and his medical degree from the University of Calgary School of Medicine.

Dr. Thapar’s research has focused on the molecular mechanisms underlying pituitary tumorigenesis, structuring his PhD thesis around the molecular events underlying neoplastic progression in acromegaly-associated pituitary tumors. Dr. Thapar will use the Van Wagenen Fellowship help him prepare for an academic career as a pituitary neurosurgeon. He will under the co-supervision of Professor Rudolf Fahlbusch and Dr. Michael Buchfelder at the University of Erlangen-Nurnberg, Germany. This clinic is a major European referral center for pituitary tumors and for various non-pituitary sellar and skull base lesions, and includes a well-established pituitary research laboratory.

Dr. Thapar will spend six months in clinical training developing a broad repertoire of operative approaches; five months in research investigating how alterations in the expression patterns of GHRH and SRIF mRNA’s, their protein products, and their receptors within surgically-removed somatotroph adenomas affect the signal transduction cascades in the neoplastic somatotroph in vitro; and one month observing other European pituitary surgery/research centers.

During his clinical training in Germany, Dr. Thapar’s goals are to practice several operative approaches including standard and modified transsphenoidal approaches, contemporary skull base approaches, and endoscopic approaches to the sella. He also hopes to expand his knowledge of intraoperative MRI as an adjunct to pituitary resection and pre-surgical pharmacologic therapies. Dr. Thapar’s aims for the research section of his fellowship include:

1) to determine in vitro whether somatotroph tumors expressing GHRH and SRIF mRNA transcripts preferentially use one or another of the classical adenylate cyclase protein kinase A or the alternate protein kinase C-phospholipid-dependent signal transduction cascades;
2) to determine the relationship between tumoral GHRH and SRIF mRNAs, protein and receptor expression in the presence and absence of activating mutations of the gsp oncogene and their respective responsiveness to GHRH, SRIF and their agonists and antagonists in tissue culture;
to determine the effects of pre-surgical therapy of the SRIF analog, octreotide, on the expression of basal GHRH mRNA, protein and receptor levels in somatotroph adenomas; and

4) to acquire experience with heterologous tissue transplantation and begin work on the development of an immunodeficient xenograft mouse model of human somatotroph adenomas.

Already a well established clinical investigator, Dr. Thapar also been awarded five research grants in support of his projects from such diverse organizations as NASA and the Canadian Space Agency, Physicians Services Incorporated Foundation, and St. Michael's Hospital Research Foundation. In recognition of his work, he has received more than 17 academic or research awards including: the World Federation of Neurosurgical Societies Young Neurosurgeons Research Award (1997); the Royal College of Physicians and Surgeons of Canada National Resident Research Award (1997); Gallie Bateman Surgical Research Prize (1996); Thomas P. Morely Neurosurgical Research Price (1996); Canadian Society for Clinical Investigation and Medical Research council of Canada Research Award (1995); Stella Klotz Medal in Cellular and Molecular Pathology (1995) the AANS/CNS Joint Section on Tumors Pruess Resident Research Award (1994); the William Horsey Neurosurgical Research Prize (1994); and the Medical Research Council of Canada Research Fellowship (1994–1997).

Dr. Thapar has written extensively about his work, including 12 research articles as well as 23 book chapters. He is also a reviewer for six scientific journals and is editing a 25-chapter, multi-authored reference text dealing with basic science, clinical and therapeutic aspects of pituitary tumors.

THE WILLIAM P. VAN WAGENEN FELLOWSHIP

The William P. Van Wagenen Fellowship was established in 1966 by Mrs. Abigail R. Van Wagenen as a memorial to her late husband, who was a Charter member and first President of the Harvey Cushing Society, now known as The American Association of Neurological Surgeons. Advised by Dr. Cushing, Dr. Van Wagenen spent an interval following his residency in Europe broadening his knowledge of neurosurgery and related basic sciences.

The Fellowship is awarded annually and carries a $20,000 stipend for living and travel to a country outside of North America for a period of at least six months. The first Fellowship was awarded in 1968 and the program has been administered by the AANS since its inception.

Through Mrs. Van Wagenen’s generosity and continued support, along with recent contributions from past Fellowship recipients and members of the Van Wagenen Committee, the stipend has increased throughout the past 19 years. However, the basic philosophy and criteria for the award remain unchanged: To provide for travel to a foreign area in observation, study, or research for scientific enrichment, in preparation for an academic career in neurological surgery or its allied sciences.

VAN WAGENEN FELLOWS

1968 Richard M. Bergland, MD
1969 John M. Tew, Jr., MD
1970 M. Peter Heilbrun, MD
1971 Ira C. Denton, Jr., MD
1972 Robert A. Ratcheson, MD
1973 Joan Venes, MD
1974 W. Michael Vise, MD
1975 Lawrence H. Pitts, MD
1976 Patrick J. Kelly, MD
1977 William F. Chandler, MD
1978 Jay D. Law, MD
1979 George W. Tyson, MD
1980 L. Dade Lunsford, MD
1981 Stephen J. Haines, MD
1982 Larry V. Carson, MD
1983 Lawrence F. Borges, MD
1984 Edmund H. Frank, MD
1985 Marc Robert Mayberg, MD
1986 Edward G. Hames II, MD
1987 Emily D. Friedman, MD
1988 Mark W. Jones, MD
1989 David W. Newell, MD
1990 Walter A. Hall, MD
1991 Ian F. Pollack, MD
1992 Mary Louise Hlavin, MD
1993 Mary E. Linskey, MD
1994 Ivar Mendez, MD
1995 Timothy Charles Ryken, MD
1996 Howard L. Weiner, MD
1997 Zelma Kiss, MD
1998 Kamal Thapar, MD
RESEARCH FOUNDATION

The first Research Foundation grants were awarded in 1983, a year ahead of schedule due to the overwhelming financial support of the neurosurgical community and the medical industry. In the 15 years since the first grant was awarded, the Research Foundation has received 391 applications and has funded what it has deemed as the best 50 proposals representing 74 years of neuroscience research.

It is the clinician investigators trained at the interface who will bridge the gap between clinical neurosurgery and molecular neuroscience. Without this avenue of financial assistance, these young researchers find it difficult to obtain funding to start their research careers.

The following awardees are nearing the completion of their funding by the Research Foundation. You should anticipate seeing more detailed descriptions of their work presented at upcoming meetings and in scientific journals. Thank you for your part in launching their research careers.

FRANK FEIGENBAUM, MD
1995 RESEARCH FELLOW

Parent Organization: Georgetown University

Sponsor and Chairman: Robert L. Martuza, MD

Transcriptional Targeting of Recombinant HSV for Treatment of Nestin Producing Brain Tumors

This project involved the development of a recombinant Herpes Simplex virus (SV) whose cytotoxicity is limited to intentionally targeted tumor cell types. An HSV immediate-early (IE) gene essential for viral replication was first placed under the control of regulatory elements from the nestin gene, a gene shown to be upregulated in tumors of neuroectodermal origin. The ability of this construct to drive IE gene expression was then demonstrated in vitro. The construct was then introduced into the HSV-1 genome using homologous recombination. The tumor cell killing efficacy and safety of the recombinant virus are now being examined.
ADAM MAMELAK, MD  
1997 YOUNG CLINICIAN INVESTIGATOR  

Parent Organization: California Institute of Technology  
Chairman: William L. Caton, MD  
Sponsors: Drs. Scott E. Fraser and Erin M. Schuman  

Injury Induced Neuronal Reorganization in the Hippocampus  

We have been studying the process of neuronal reorganization that occurs following seizure-induced injury to the hippocampus, a process known as Mossy Fiber Sprouting (MFS). MFS may be an important model of epileptogenesis. We are using time-lapse two photon laser scanning microscopy to study the dynamic aspects of this reorganization process in an in vitro hippocampal slice preparation. The time-lapse imaging does demonstrate dynamic aspects of neuronal reorganization following injury. Early results are encouraging and suggest that the interaction between injury, cell death, cell birth, and synaptic reorganization is a highly dynamic process which can be altered by neuroprotective agents such as Na-Sal. Completion of experiments over the next 6-8 months will be needed to confirm these initial observations.

E. SANDER CONNOLLY, MD  
1997 YOUNG CLINICIAN INVESTIGATOR  

Parent Organization: Columbia University  
Chairman: Robert A. Soloman, MD  
Sponsors: David J. Pinsky, MD  

Leukocyte Adhesion Receptors and Thrombosis in the Pathogenesis of Evolving Stroke
THE AMERICAN ASSOCIATION OF NEUROLOGICAL SURGEONS

1997–1998

OFFICERS AND EXECUTIVE COMMITTEE

Edward R. Laws, Jr. .......................................................... President
Russell L. Travis .......................................................... President-Elect
William Shucart ......................................................... Vice President
Stanley Pelofsky .......................................................... Secretary
Stewart B. Dunsker ....................................................... Treasurer
J. Charles Rich ............................................................ Past President

BOARD OF DIRECTORS

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<thead>
<tr>
<th>Name</th>
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<tbody>
<tr>
<td>Edward R. Laws, Jr., President</td>
<td>1999</td>
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<tr>
<td>Russell L. Travis, President-Elect</td>
<td>2000</td>
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<td>William Shucart, Vice President</td>
<td>1998</td>
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<td>Stanley Pelofsky, Secretary</td>
<td>2000</td>
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<tr>
<td>Stewart B. Dunsker, Treasurer</td>
<td>1998</td>
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<tr>
<td>J. Charles Rich, Past President</td>
<td>1998</td>
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<tr>
<td>Arthur L. Day, Director-at-Large</td>
<td>1999</td>
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<tr>
<td>Roberto C. Heros, Director-at-Large</td>
<td>1998</td>
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<tr>
<td>John A. Kusske, Southwest Region</td>
<td>1999</td>
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<td>Robert B. Page, Northeast Region</td>
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<tr>
<td>A. John Popp, Director-at-Large</td>
<td>1998</td>
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<td>P. Robert Schwetschenau, Northwest Region</td>
<td>1999</td>
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<td>R. Michael Scott, Director-at-Large</td>
<td>1998</td>
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<tr>
<td>William E. Mayher III, Southeast Region</td>
<td>2000</td>
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<tr>
<td>Robert A. Ratcheson, Director-at-Large</td>
<td>2000</td>
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<tr>
<td>David F. Jimenez, Liaison from Young Neurosurgeons Committee</td>
<td>1998</td>
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<tr>
<td>James R. Bean, Liaison from CSNS</td>
<td>1999</td>
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</table>
APPOINTED OFFICERS

HISTORIAN
Byron C. Pevehouse (1994)

EXECUTIVE DIRECTOR
Robert E. Draba (1996)

PARLIAMENTARIAN
Edith Huck (1987)

JOURNAL OF NEUROSURGERY

John A. Jane, Editor
Martin H. Weiss, Associate Editor Neurosurgical Focus (1987)

Editorial Board
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Edward H. Oldfield (1994)
Robert A. Ratcheson (1992)
H. Richard Winn (1987)

AANS BULLETIN
Michael L.J. Apuzzo, Editor (1991)

NEUROSURGERY://ON-CALL®
John J. Oró, Editor (1997)

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Richard D. Bucholz
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Domenic P. Esposito
William A. Friedman, Past Editor
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Joel MacDonald
David McKalip
A. John Popp
Cavett Robert, Jr.
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Sidney Tolchin
Bradford B. Walters
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Stanley Pelofsky, Secretary
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* ex officio without vote

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** Advisory without vote

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William S. Shucart (1997)
*Members, ex officio without vote

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Sean F. Mullan, ad hoc advisor to Chairman (1993)

NOMINATING

(Year indicates expiration of term)


PROFESSIONAL CONDUCT

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Sidney Tolchin (1997)

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Vincent C. Traynelis, Secretary (1997)
Curtis A. Dickman, Treasurer (1997)

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Beverly C. Walters, Chairman (1995), Guidelines
Robert E. Harbaugh, Chairman (1997), Outcomes

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Ralph G. Dacey (1997)
Troy Tippett (1997)
William A. Friedman, President (1996)
H. Hunt Batjer, President-Elect (1997)

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Timothy Harrington, Corresponding Secretary (1995)
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Samuel J. Hassenbusch (1995)
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C. Scott McLanahan (1996)
Christopher S. Kliefoth (1995)
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David W. Roberts (1995)
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Richard M. Toselli (1992)
Clarence B. Watridge (1991)
Craig H. Yorke, Jr. (1994)

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Peter K. Dempsey (1995)
Gregg N. Dyste (1995)
Barton L. Guthrie (1995)
David A. Herz (1996)
Stanley B. Martin (1995)
Paul C. McCormick (1995)
Thomas C. Origitano (1995)
Troy M. Tippett (1996)
Russell L. Travis (1996)
Craig A. Van Der Veer (1995)
Julian K. Wu (1995)
Joseph A. Zabramski (1996)
Greg Zorman (1991)
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Karl Stecher Philip J. A. Willman (1995)

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Bruce L. Kihlstrom Donald J. Prolo (1994)
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Edie E. Zusman, Chairman (1997)
Adam I. Lewis, Vice Chairman (1997) and AANS Representative to the House of Delegates
Jose L. Rodriguez (1994)
Richard M. Toselli (1997)
Andrew G. Chennelle (1997) CNS Representative to AMA House of Delegates

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- **G. Bloomgarden, CT**
- **B. Bose, DE**
- **S. Burstein, NY**
- **M. Epstein, RI**
- **J. Gastaldo, PA**
- **D. Gaylon, NY**
- **G. Harsh, MA**
- **R. Hodosh, NJ**
- **I. Kasoff, NJ**
- **E. Kornel, NY**
- **E. Leibowitz**
- **B. Northrup, PA**
- **H. Oestreicher, NY**
- **J. Phillips, PA**
- **H. Richter, PA**
- **P. Spurgas, NY**
- **B. Trembly, ME**
- **R. Page, PA, ex officio**

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- **W. B. Blackett, WA**
- **H. Chandler, MT**
- **S. Cushman, WI**
- **D. Danoff, MN**
- **F. Diaz, MI**
- **K. Follett, IA**
- **W. Ganz, MN**
- **C. Getch, IL**
- **J. Godersky, AK**
- **J. Goodman, IN**
- **J. Hubbard, OR**
- **C. Kam, HI**
- **D. Kelman, WI**
- **C. Koski, ND**
- **B. Le Compte, IL**
- **J. Oakley, WA**
- **S. Ondra, IL**
- **M.B. Pritz, IN**
- **J. Rock, MI**
- **D. Szymanski, MI**
- **R. Taniguchi, HI**
- **C. Zimmerman, ID**
- **P. Schwetschenau, OH, ex officio**

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- **F. Boop, AR, Vice Chairman**
- **B. Allen, VA**
- **J. Bean, KY**
- **S. Bloomfield, WV**
- **D. Cahill, FL**
- **C. Clark, TN**
- **R. Cohen, MD**
- **R. Davis, MD**
- **G. Dennis, DC**
- **G. Hunt, VA**
- **P.R. Jacob, FL**
- **D. Jimenez, MO**
- **G.N. Kadis, GA**
- **B. Kihlstrom, NC**
- **A. Lewis, MS**
- **A. Marchosky, MO**
- **H. Mercado, PR**
- **J. Metcalf, TN**
- **W.L. Pritchard, NC**
- **T. Rigsby, AL**
- **P.W. Tally, FL**
- **M. Tyler, SC**
- **W. Mayher III, GA, ex officio**

### SOUTHWEST QUADRANT

- **J. McVicker, CO, Chairman**
- **T. Bertucci, LA**
- **A. Capanna, NV**
- **J. Cone, TX**
- **J. Dunn, AZ**
- **T. Harrington, AZ**
- **T. Hoyt, CA**
- **R. Jackson, TX**
- **T. Kenefick, CA**
- **J. Levy, CA**
- **E. Marchand, NM**
- **S. Pelofsky, OK**
- **D. Prolo, CA**
- **J. Rich, CA**
- **R. Smith, CA**
- **R.D. Smith, LA**
- **K. Stecher, CO**
- **F. Todd, TX**
- **F. Wagner, CA**
- **R. Weiner, TX**
- **P. Willman, TX**
- **E. Zusman, CA**
- **J. Kusske, CA, ex officio**
Council of State Neurosurgical Societies Representatives

Alabama  Thomas Rigsby (Alt: Thomas Staner)
Alaska  John Godersky
Arizona  Jack Dunn, Timothy Harrington (Alts: Hillel Baldwin, Stephen Ritland, Thomas Scully)
Arkansas  Frederick Boop (Alt: Charles Teo)
California  Thomas Hoyt, Thomas Kenefick, John Kusske, ex officio, Jay Levy, Donald Prolo, J. Ronald Rich, Randall Smith, Franklin Wagner, Edie Zusman (Alts: Philipp Lippe, Jose Rodriguez, Moris Senegor)
Colorado  John McVicker, Karl Stecher (Alt: D. James Scelats)
Connecticut  Ramon Batson, Gary Bloomgarden (Alt: Stanley Pugsley)
Delaware  Bikash Bose (Alt: Magdy Boulos)
District of Columbia  Gary Dennis (Alts: Frederic Schwartz, Ronald Uscinski)
Florida  David Cahill, Patrick Jacob, Philip Tally, (Alts: Jacques Farkas, Dean Loehse, Ignacio Magana)
Georgia  Domenic Esposito, Gerald Kadis, William Mayher III, ex officio (Alt: Michael Hartman)
Hawaii  Calvin C.M. Kam, Raymond Taniguchi
Idaho  NO ACTIVE NEUROSURGICAL SOCIETY
Illinois  Christopher Getch, Benjamin Le Compte III, Stephen Ondra (Alt: Charles Kennedy, Donald Pearson)
Indiana  Julius Goodman, Michael Pritz (Alts: Jeffrey Crecelius, Thomas Leipzig)
Iowa  SEE MIDWEST NEUROSURGICAL SOCIETY
Kansas  SEE MIDWEST NEUROSURGICAL SOCIETY
Kentucky  James Bean (Alt: George Raque)
Louisiana  Thomas Bertuccini, Roger Smith (Alt: David Cavanaugh)
Maine  Bruce Trembley
Maryland  Ronald Cohen, Reginald Davis (Alt: Massimo Fianadaca)
Massachusetts  Griffith Harsh, Eugene Liebowitz (Alt: Joseph Arena)
Michigan  Fernando Diaz, Jack Rock, Dennis Szymanski (Alts: Alexa Canady, Reynaldo Castillo)
Midwest Neurosurgical Society  Kenneth Follett, Lyal Leibrock (Iowa, Kansas, Nebraska, South Dakota)
Minnesota  David Danoff, William Ganz (Alt: Gregg Dyste)
Mississippi  Adam Lewis (Alt: Jimmy Miller)
Missouri  David Jimenez, Alexander Marchosky (Alts: Geoffrey Blatt, John Oro)
Montana  Howard Chandler
Nebraska  SEE MIDWEST NEUROSURGICAL SOCIETY
Nevada  Albert Capanna (Alt: Debra Nelson)
New Hampshire  Theodore Jacobs (Alt: Robert Harbaugh)
New Jersey  Richard Hodosh, Ira Kasoff (Alts: Henry Liss, Tariq Siddiqi)
New Mexico  Erich Marchand (Alt: Venkat Narayan)
North Carolina  Bruce Kihlstrom, William Pritchard (Alts: Douglas Jones, Victoria Neave)
North Dakota  Charles Koski
Ohio  Gene Barnett, Jeffrey Brown, P. Robert Schwetschenau, ex officio
Oklahoma  Stanley Pelofsky (Alt: Robert Remondino)
Oregon  Mark Belza, Jerry Hubbard (Alt: Maurice Collada)
Pennsylvania  John Gastaldo, Bruce Northrup, Robert Page, ex officio John Phillips, Howard Richter
Puerto Rico  Hiram Mercado (Alt: Jaime Inserni)
Rhode Island  Mel Epstein (Alt: Samuel Greenblatt)
South Carolina  Michael Tyler (Alt: Byron Bailey)
South Dakota  SEE MIDWEST NEUROSURGICAL SOCIETY
Tennessee  Craig Clark, James Metcalf
Texas  Jeffrey Cone, Richard Jackson, Frederick Todd, Richard Weiner, Philip Willman (Alts: Lee Ansell, Bruce Ehni, Clark Watts)
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<tr>
<th>State</th>
<th>Active Neurosurgical Society</th>
<th>Alternates</th>
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<tr>
<td>Utah</td>
<td>NO ACTIVE NEUROSURGICAL SOCIETY</td>
<td>(Alt: Laverne Erickson)</td>
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<td>Vermont</td>
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<td>NO ACTIVE NEUROSURGICAL SOCIETY</td>
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<tr>
<td>Virginia</td>
<td>Benjamin Allen, George Hurt</td>
<td>(Alts: John Feldenzer, Jackson Salvant)</td>
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<tr>
<td>Washington</td>
<td>W. Ben Blackett, John Oakley</td>
<td>(Alt: A. Basil Harris)</td>
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<td>West Virginia</td>
<td>Steve Bloomfield</td>
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<td>Wisconsin</td>
<td>S. Marshall Cushman, Donald Kelman</td>
<td>(Alt: Mohammed Rafiullah)</td>
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<tr>
<td>Wyoming</td>
<td>NO ACTIVE NEUROSURGICAL SOCIETY</td>
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NEUROSURGICAL REPRESENTATIVES AND LIAISONS TO OTHER ORGANIZATIONS

ACCREDITATION COUNCIL FOR GRADUATE MEDICAL EDUCATION

Edward R. Laws, Jr., Liaison

Residency Review Committee for Neurological Surgery

Robert G. Ojemann, Chairman (AMA 1997)
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Howard M. Eisenberg (ACS 1999)
George A. Ojemann (ABNS 1999)
John C. Van Gilder (ABNS 2001)
Edward R. Laws, Jr. (AMA 1998)

AMERICAN ACADEMY OF ORTHOPAEDIC SURGEONS

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Charles L. Branch, Liaison (1993)
Volker K.H. Sonntag, Liaison (1993)

Council on Spine Societies (COSS)

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Richard G. Fessler, Representative (1997)
Arnold H. Menezes, Representative (1997)
Stephen M. Papadopoulos, Alternate (1998)

AMERICAN ACADEMY OF PAIN MEDICINE

Kim J. Burchiel, Liaison (1995)

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(Year indicates expiration of term)

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Burton M. Onofrio, Vice Chairman (AAcad 1997)
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Stewart B. Dunsker, Treasurer (AANS 1999)

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M. Peter Heilbrun (AMA 2000) Charles J. Hodge, Jr. (SNS 2002)
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Issam A. Awad (AANS 1997) W. Ben Blackett (AANS 1997)
Ralph G. Dacey, Jr. Arthur L. Day
Karin M. Muraszko Raj K. Narayan (Mem. at Large 1998)
(Young Neurosurgeons Rep. AANS 1997) Robert P. Page

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Glenn Morrison (SoNS 1998)
Bryce K. Weir (SNS 1998)
H. Richard Winn (AANS 1998)

AMERICAN MEDICAL ASSOCIATION

House of Delegates

Mark J. Kubala, Delegate (1994)

Section Council on Neurosurgery

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Howard A. Richter, Pennsylvania, Delegate
Joseph S. Sadowski, Connecticut, Delegate
Peter R. Wilson, American Academy of Pain Medicine

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Philipp M. Lippe, Representative (1984)

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Donald C. Wright, Liaison (1993)

AMERICAN TORT REFORM ASSOCIATION

P. Robert Schwetschenau, Liaison (1997)
W. Ben Blackett, Liaison (1990)
ASSOCIATION OF AMERICAN MEDICAL COLLEGES, COUNCIL OF ACADEMIC SOCIETIES

Donlin M. Long, Representative (1991)
Mel H. Epstein, Representative (1994)

EPILEPSY ORGANIZATIONS

Dennis Spencer, Representative (1997)

NATIONAL ASSOCIATION FOR BIOMEDICAL RESEARCH

Phanor L. Perot, Jr., Liaison (1988)

NATIONAL ASSOCIATION FOR BRAIN RESEARCH

Robert L. Martuza, Representative (1994)

NATIONAL INSTITUTES OF HEALTH

Arthritis & Musculoskeletal Institute

Arnold H. Menezes, Liaison (1996)

National Cancer Institute

Robert L. Martuza, Liaison (1992)

National Institute of Neurological Disorders and Stroke (NINDS)

Julian T. Hoff, Liaison (1997)

Rehabilitation Council

Guy L. Clifton, Liaison (1991)

NORTH AMERICAN SPINE SOCIETY

Volker K.H. Sonntag, Liaison (1996)

VETERANS ADMINISTRATION

H. Richard Winn, Liaison Chairman (1993)
Robert E. Maxwell (1994)
Paul B. Nelson (1993)
Robert A. Ratcheson (1993)

WORLD FEDERATION OF NEUROSURGICAL SOCIETIES

Madjid Samii, President (1997-2001)
Edward R. Laws, Jr., First Vice President (1997-2001)
Maurice Choux, Secretary (1997-2001)
Richard Perrin, Assistant Secretary (1997-2001)
S. Kobayashi, Treasurer (1997-2001)
Iftikhar Ali Raja, Assistant Treasurer (1997-2001)
Albert L. Rhoton, Jr., AANS Delegate and Liaison (1990-1997)
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Sidney Goldring
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Robert L. Grubb, Jr.
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Albert L. Rhoton, Jr.
Edward R. Laws, Jr., AANS President
Russell L. Travis, AANS President-Elect
Stewart B. Dunske, AANS Treasurer
Gail L. Rosseau, Liaison from Young Neurosurgeons Committee (1998)

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Darrell D. Bigner
James Ferrendelli
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THINK FIRST FOUNDATION
Formerly The National Head & Spinal Cord Injury Prevention Program
(Founded jointly by the American Association of Neurological Surgeons and Congress of Neurological Surgeons)

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Albert R. Buscaione, Secretary
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Michael J. Caron, Vice Chairman

Liaison from AANS

Mitchel S. Berger
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✔ Reduced Professional Development Course Fees
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✔ Special Journal of Neurosurgery subscription rate
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Candidate—for residents who are enrolled in a neurosurgery residency training program approved by ABNS, RCS of Canada, or MCNS.

Associate—for those who are not neurosurgeons but have shown distinction in related medical disciplines. Eligible individuals include certified neuroscience nurses (CNRN, CNOR, CCRN) and physician assistants (PA-C). Associate members are nominated for membership by three voting members of the AANS.

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Honorary—for those who are recognized internationally for their outstanding education, research, or clinical contributions to neurologic science. The Honorary member must be proposed by voting members in good standing and approved by the Board of Directors and the voting membership.

For more information and/or an application for membership, please contact the AANS National Office at:

22 South Washington Street
Park Ridge, Illinois 60068-4287
Phone: (847) 692-9500; Fax: (847) 692-6770
e-mail: info@aans.org

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### AANS Membership by Category

<table>
<thead>
<tr>
<th>Category</th>
<th>Count</th>
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<tr>
<td>Active</td>
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<tr>
<td>Lifetime Active</td>
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<tr>
<td>Lifetime (Inactive)</td>
<td>692</td>
</tr>
</tbody>
</table>

**Total AANS Membership** ....... 5,263

*Figures as of February, 1998*
NEWLY ELECTED MEMBERS
(January 1, 1997, through December 31, 1997)

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Mexico, D.F., Mexico

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Oaxaca, Mexico

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Kettering, OH

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Springfield, MO

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Denver, CO

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Modesto, CA

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Puebla, Pue., Mexico

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Monterrey, N.L., Mexico

Gustavo Chagoya-Galaz, MD  
Monclova, Coah., Mexico

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Hermosillo, Sonora, Mexico

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Portland, OR

Caetano Porto Coimbra, MD  
Dallas, TX

John J. Collins, MD  
Honolulu, HI

Blanca Azucena Contreras, MD  
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Hammond, LA

Robert C. Dauser, MD  
Houston, TX

Jose R. De La Cruz, MD  
Mexico, D.F., Mexico

Philip C. Deaton, MD  
Greensboro, NC

Mario Del Valle, MD  
Torreon, Coah., Mexico

Ramiro Del Valle Robles, MD  
Mexico, D.F., Mexico

Victor Manuel Diaz-Simental, MD  
Culiacan, Sin., Mexico

Oliver N.R. Dold, MD  
Decatur, IL

Manuel Dominguez Cahero, MD  
Guadalajara, Jal., Mexico

John D. Ebeling, MD  
Topeka, KS
<table>
<thead>
<tr>
<th>Name</th>
<th>City, State</th>
</tr>
</thead>
<tbody>
<tr>
<td>Michael R. Egnor, MD</td>
<td>Stony Brook, NY</td>
</tr>
<tr>
<td>Carlos L. Encinas-Olea, MD</td>
<td>Nogales, Son., Mexico</td>
</tr>
<tr>
<td>Ricardo Flores Escamilla, MD</td>
<td>Mexicali, Mexico</td>
</tr>
<tr>
<td>Ruben Eduardo Flores Reyes, MD</td>
<td>Monclova, Coah., Mexico</td>
</tr>
<tr>
<td>David T. Floyd, MD</td>
<td>Aiken, SC</td>
</tr>
<tr>
<td>Ruben Franco Davalos, MD</td>
<td>Leon, Gto., Mexico</td>
</tr>
<tr>
<td>William F. Ganz, MD</td>
<td>Rapid City, SD</td>
</tr>
<tr>
<td>Cesar Arcadio Garcia De Llano, MD</td>
<td>Guadalajara, Jal., Mexico</td>
</tr>
<tr>
<td>Victoriano Garcia Gonzalez, MD</td>
<td>Guadalajara, Jal., Mexico</td>
</tr>
<tr>
<td>Abraham Garcia Nieva, MD</td>
<td>Puebla, Pue., Mexico</td>
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<tr>
<td>Joel Eduardo Garcia Pacheco, MD</td>
<td>Los Mochis, Sin., Mexico</td>
</tr>
<tr>
<td>Luis Garcia-Munoz, MD</td>
<td>Naucalpan, Mexico</td>
</tr>
<tr>
<td>Gilberto Gardea Loera, MD</td>
<td>Delicias, Chih., Mexico</td>
</tr>
<tr>
<td>Sarah J. Gaskill, MD</td>
<td>San Antonio, TX</td>
</tr>
<tr>
<td>Marc S. Goldman, MD</td>
<td>Columbus, GA</td>
</tr>
<tr>
<td>Gary B. Goplen, MD</td>
<td>Saskatoon, SK, Canada</td>
</tr>
<tr>
<td>Charles R. Gordon, MD</td>
<td>Tyler, TX</td>
</tr>
<tr>
<td>Paul L. Gorsuch, Jr., MD</td>
<td>Great Falls, MT</td>
</tr>
<tr>
<td>Jose Efren I. Grijalva Otero, MD</td>
<td>Mexico, D.F., Mexico</td>
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<tr>
<td>Armando G. Guerrero, MD</td>
<td>Mexico, D.F., Mexico</td>
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<tr>
<td>Francisco J. Guerrero Jazo, MD</td>
<td>Mexico, D.F., Mexico</td>
</tr>
<tr>
<td>Jose F. Gutierrez, MD</td>
<td>Mexico, D.F., Mexico</td>
</tr>
<tr>
<td>Mario A. Gutierrez, MD</td>
<td>Roswell, NM</td>
</tr>
<tr>
<td>Carl Barnes Heilman, MD</td>
<td>Boston, MA</td>
</tr>
<tr>
<td>Susan R. Hemley, MD</td>
<td>Bethlehem, PA</td>
</tr>
<tr>
<td>James M. Herman, MD</td>
<td>Ventura, CA</td>
</tr>
<tr>
<td>Tenoch Herrada-Pineda, MD</td>
<td>Mexico, D.F., Mexico</td>
</tr>
<tr>
<td>M. Fernando Herreman, MD</td>
<td>Mexico City, Mexico</td>
</tr>
<tr>
<td>Matthew A. Howard, III, MD</td>
<td>Iowa City, IA</td>
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<tr>
<td>John Nai-Keung Hsiang, MD, PhD</td>
<td>Seattle, WA</td>
</tr>
<tr>
<td>Steven M. James, MD</td>
<td>Beechgrove, IN</td>
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<tr>
<td>Stephen D. Johnson, MD</td>
<td>Denver, CO</td>
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<td>Patrick Alton Juneau, III, MD</td>
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<td>Marcus F. Keep, MD</td>
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<td>Michel Kliot, MD</td>
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<tr>
<td>Stefan J. Konasiewicz, MD</td>
<td>Duluth, MN</td>
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<tr>
<td>David E. Kosmoski, MD</td>
<td>Austin, TX</td>
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<tr>
<td>Diana L. Abson Kraemer, MD</td>
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<td>Gary Edward Kraus, MD</td>
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<td>Jorge Kuri Bujaidar, MD</td>
<td>Puebla, Pue., Mexico</td>
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<tr>
<td>Gregory B. Lanford, MD</td>
<td>Nashville, TN</td>
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<tr>
<td>Sander W. Leivy, MD</td>
<td>Roanoke, VA</td>
</tr>
<tr>
<td>Victor Manuel Leon-Meza, MD</td>
<td>Leon, Gto., Mexico</td>
</tr>
<tr>
<td>Allan D. Levi, MD, PhD</td>
<td>Miami, FL</td>
</tr>
<tr>
<td>Name</td>
<td>City, State/St., Country</td>
</tr>
<tr>
<td>----------------------------------------------------------------------</td>
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</tr>
<tr>
<td>George H. Lien, MD</td>
<td>Murfreesboro, TN</td>
</tr>
<tr>
<td>Mark E. Linskey, MD</td>
<td>San Diego, CA</td>
</tr>
<tr>
<td>Shih Sing Liu, MD</td>
<td>Tucson, AZ</td>
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<tr>
<td>Aloyysis V. Llaguno, MD</td>
<td>Loomis, CA</td>
</tr>
<tr>
<td>Marie L. Long, MD</td>
<td>Decatur, IL</td>
</tr>
<tr>
<td>Blas Ezequiel Lopez Felix, MD</td>
<td>Mexico, D.F., Mexico</td>
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<tr>
<td>Manuel Lopez-Portillo, MD</td>
<td>Morelos, Mexico</td>
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<tr>
<td>Eduardo Magallon, MD</td>
<td>Mexico, D.F., Mexico</td>
</tr>
<tr>
<td>Kasargod B. Mallya, MD</td>
<td>Port Jefferson Stat., NY</td>
</tr>
<tr>
<td>Alfonso Marhx-Bracho, MD</td>
<td>Mexico, D.F., Mexico</td>
</tr>
<tr>
<td>Jesus Martinez-Garza, MD</td>
<td>Monterrey, N.L., Mexico</td>
</tr>
<tr>
<td>Ivar M. Mendoza, MD</td>
<td>Halifax, NS, Canada</td>
</tr>
<tr>
<td>Albert Louis Meric, III, MD</td>
<td>Knoxville, TN</td>
</tr>
<tr>
<td>Charles J. Miller, MD</td>
<td>Apo, AE, New York</td>
</tr>
<tr>
<td>Juan Miquelajauregui, MD</td>
<td>Mexico, D.F., Mexico</td>
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<tr>
<td>Ross R. Moquin, MD</td>
<td>Silver Spring, MD</td>
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<tr>
<td>William J. Morris, MD</td>
<td>Tacoma, WA</td>
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<tr>
<td>Juan Arturo Mucino Mercado, MD</td>
<td>Leon, Gto., Mexico</td>
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<tr>
<td>Jose Manuel Munoz Tagle, MD</td>
<td>Mexico, D.F., Mexico</td>
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<tr>
<td>Steven E. Murk, MD</td>
<td>Colorado Springs, CO</td>
</tr>
<tr>
<td>Ali J. Naini, MD</td>
<td>Woodland Hills, CA</td>
</tr>
<tr>
<td>Edgar Nathal, MD</td>
<td>Mexico, D.F., Mexico</td>
</tr>
</tbody>
</table>

ACTIVE MEMBERS (Cont.)
<table>
<thead>
<tr>
<th>Name</th>
<th>City, State/Country</th>
</tr>
</thead>
<tbody>
<tr>
<td>Candelario Rivas Olvera, MD</td>
<td>Pachuca, Hgo., Mexico</td>
</tr>
<tr>
<td>Daniel Payne Robertson, MD</td>
<td>Fort Myers, FL</td>
</tr>
<tr>
<td>Roberto Rodriguez Della, MD</td>
<td>San Luis Potosi, SLP, Mexico</td>
</tr>
<tr>
<td>Alfredo Roman Messina, MD</td>
<td>Mazatlan, Sin., Mexico</td>
</tr>
<tr>
<td>Victor Manuel Romero Vazquez, MD</td>
<td>Monterrey, N.L., Mexico</td>
</tr>
<tr>
<td>Ian B. Ross, MD</td>
<td>Winnipeg, MB, Canada</td>
</tr>
<tr>
<td>David P. Sachs, MD</td>
<td>Boca Raton, FL</td>
</tr>
<tr>
<td>Octavio A. Salazar, MD</td>
<td>Mexico, D.F., Mexico</td>
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<tr>
<td>Salvador Salazar Gama, MD</td>
<td>Aguascalientes, Ags., Mexico</td>
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<tr>
<td>Jose M. Sanchez Cabrera, MD</td>
<td>Mexico, D.F., Mexico</td>
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<tr>
<td>Carl J. Sartorius, MD</td>
<td>Indianapolis, IN</td>
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<tr>
<td>Scott M. Schlesinger, MD</td>
<td>Little Rock, AR</td>
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<td>Jose A. Segovia, MD</td>
<td>Irapuato, Gto., Mexico</td>
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<td>Alberto Segovia Philip, MD</td>
<td>Puebla, Pue., Mexico</td>
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<tr>
<td>R. James Seymour, MD</td>
<td>Bay Shore, NY</td>
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<tr>
<td>Morris M. Soriano, MD</td>
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<td>Timothy E. Stepp, MD</td>
<td>Shawnee Mission, KS</td>
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<td>Leslie E. Stern, MD</td>
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<td>Maximino Tellez-Gutierrez, MD</td>
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<tr>
<td>Harvey G. Thomas, MD</td>
<td>Phoenix, AZ</td>
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<tr>
<td>Jaime G. Torrespore, MD</td>
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<tr>
<td>Ian A. Trueba-Reyes, MD</td>
<td>Toluca, Edo., Mexico</td>
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<tr>
<td>David D. Udehn, MD</td>
<td>Moline, IL</td>
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<tr>
<td>Eduardo Vazquez-Celis, MD</td>
<td>Mexico City, D.F., Mexico</td>
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<td>Leon, Gto., Mexico</td>
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<td>Jose M. Viveros Utrera, MD</td>
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<tr>
<td>Edward Von der Schmidt, III, MD</td>
<td>Princeton, NJ</td>
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<tr>
<td>Thomas M. Wascher, MD</td>
<td>Appleton, WI</td>
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<tr>
<td>Harry C. Weiser, MD</td>
<td>Evans, GA</td>
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<td>Crystl D. Willison, MD</td>
<td>Cincinnati, OH</td>
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<tr>
<td>Charles J. Wright, MD</td>
<td>Rockford, IL</td>
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<td>Jacob N. Young, MD</td>
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<td>Juan M. Zamarripa, MD</td>
<td>Irapuato, Gto., Mexico</td>
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<td>Carlos Miguel Zamorano, MD</td>
<td>Mexico City, D.F., Mexico</td>
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<tr>
<td>Andrew Stephen Selby, MD</td>
<td>Maywood, IL</td>
</tr>
</tbody>
</table>
ACTIVE (PROVISIONAL) MEMBERS

J. Greg Adderholt, Jr., MD
Muscle Shoals, AL

David E. Adler, MD
Hartsdale, NY

Joseph Aferzon, MD
New Britain, CT

Edward W. Akeyson, MD, PhD
New Haven, CT

Todd D. Alexander, MD
Rockford, IL

James M. Alvis, MD
Norman, OK

Sherry L. Apple, MD
Shreveport, LA

Anthony L. Asher, MD
Charlotte, NC

Edward F. Aulisi, MD
Washington, DC

David W. Barnett, MD
Dallas, TX

Konrad N.M. Barth, MD
Scarborough, ME

Donald L. Behrman, MD
Columbus, OH

Deborah L. Benzil, MD
Croton, NY

William B. Betts, MD
Austin, TX

Ajay K. Bindal, MD
Houston, TX

William E. Bingaman, Jr., MD
Cleveland, OH

Randolph C. Bishop, MD
Savannah, GA

David R. Blatt, MD
Nashville, TN

Scott B. Boyd, MD
West Columbia, SC

Gregory A. Brandenberg, MD, PhD
Omaha, NE

John Brayton, MD
Elburn, IL

Jason Adler Brodkey, MD
Ann Arbor, MI

Adam P. Brown, MD
Wilmington, NC

Norman Neil Brown, MD, PhD
Champaign, IL

J. Travis Burt, MD
Bristol, TN

John R. Caruso, MD
Hagerstown, MD

James P. Chandler, MD
Chicago, IL

Thomas C. Chen, MD
La Canada, CA

Theresa M. Cheng, MD
Eau Claire, WI

Mike W. Chou, MD
Brooklyn, NY

Mark A. Cobb, MD
Newburgh, IN

David William Cockerill, MD
El Paso, TX

Douglas S. Cohen, MD
New York, NY

David L. Cooper, MD
Syracuse, NY

Mark D. D’Alise, MD
Lubbock, TX

John Diaz Day, MD
Burlington, MA

Maurice J. Day, Jr., MD
Huntington, WV

Richard A.A. Day, MD
Missoula, MT

Carlo M. De Luna, MD
Edison, NJ

Rajiv D. Desai, MD
Lewiston, ME

Curtis E. Doberstein, MD
East Greenwich, RI

Stephen E. Doran, MD
Omaha, NE

Joshua L. Dowling, MD
Webster Groves, MO

Randall G. Drye, MD
Lexington, SC
ACTIVE (PROVISIONAL) MEMBERS (Cont.)

Scott C. Dulebohn, MD
Omaha, NE

Scott T. Dull, MD
Toledo, OH

Eric H. Elowitz, MD
New York, NY

David Y. Eng, MD, PhD
Syracuse, NY

Matthew J. Eppley, MD
Newark, DE

James R. Fick, MD
Augusta, GA

Aaron G. Filler, MD
Los Angeles, CA

Richard M. Foltz, MD
Fort Lauderdale, FL

Damiere T. Fossett, MD
Baltimore, MD

Paul C. Francel, MD, PhD
Piedmont, OK

Alleyne B. Fraser, MD
Middletown, NY

Michael D. Fromke, MD
Hattiesburg, MS

Mark A. Gardon, MD
Waco, TX

David S. Geckle, MD
Richmond, VA

Jeffrey S. Gerdes, MD
St. Cloud, MN

Christopher C. Getch, MD
Chicago, IL

Robert J. Gewirtz, MD
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Salem, OR

Joseph T. King, Jr., MD
Cleveland, OH

Daniel L. Kitchens, MD
St. Louis, MO

Robert S. Knego, MD
Tyler, TX
<table>
<thead>
<tr>
<th>Name</th>
<th>City, State</th>
</tr>
</thead>
<tbody>
<tr>
<td>Timothy C. Kriss, MD</td>
<td>Lexington, KY</td>
</tr>
<tr>
<td>Robert Lacin, MD</td>
<td>Goldsboro, NC</td>
</tr>
<tr>
<td>Barry J. Landau, MD</td>
<td>Grand Blanc, MI</td>
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<tr>
<td>Carl Laurysen, MD</td>
<td>St. Louis, MO</td>
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<td>Jodie K. Levitt, MD</td>
<td>Elmira, NY</td>
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<tr>
<td>Andrew Stewart Levy, MD</td>
<td>Denver, CO</td>
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<tr>
<td>Adam I. Lewis, MD</td>
<td>Jackson, MS</td>
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<tr>
<td>Leon K. Liem, MD</td>
<td>Honolulu, HI</td>
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<td>Michael J. Link, MD</td>
<td>Jacksonville, FL</td>
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<tr>
<td>Joel D. MacDonald, MD</td>
<td>Tucson, AZ</td>
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<td>Chrriss A. Mack, MD</td>
<td>Missoula, MT</td>
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<td>Jacek M. Malik, MD, PhD</td>
<td>Little Rock, AR</td>
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<td>David G. Malone, MD</td>
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<td>George T. Mandybur, MD</td>
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<td>Miguel Angel Melgar, MD, PhD</td>
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<td>B. Theo Mellon, PhD, MD</td>
<td>Carbondale, IL</td>
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<td>Peter Daniel Miller, MD, PhD</td>
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<td>N. Nicole Moayeri, MD</td>
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<td>Vittorio M. Morreale, MD</td>
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<td>Sean O’Malley, MD</td>
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<td>Kimberly A. Page, MD</td>
<td>Redding, CA</td>
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<td>Peyman Pakzaban, MD</td>
<td>Houston, TX</td>
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<td>Kee B. Park, MD</td>
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<td>Stig Peitersen, MD</td>
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<td>John G. Piper, MD</td>
<td>Bettendorf, IA</td>
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<td>Bruce E. Pollock, MD</td>
<td>Rochester, MN</td>
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<td>Kimball N. Pratt, MD</td>
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<td>Mark C. Preul, MD</td>
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<td>Paul K. Ratzker, MD</td>
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<td>Abraham Rayhaun, MD</td>
<td>Long Beach, CA</td>
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<td>Mark P. Redding, MD</td>
<td>Concord, NC</td>
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<tr>
<td>Rafael Rodriguez Mercado, MD</td>
<td>San Juan, PR</td>
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<tr>
<td>Gerald Edward Rodts, Jr., MD</td>
<td>Atlanta, GA</td>
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<td>Patrick A. Roth, MD</td>
<td>Hackensack, NJ</td>
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<td>Gregory J. Rubino, MD</td>
<td>Los Angeles, CA</td>
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<td>Michael J. Rutigliano, MD</td>
<td>Greensburg, PA</td>
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<td>Timothy C. Ryken, MD</td>
<td>Syracuse, NY</td>
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<td>Robert A. Sabo, MD</td>
<td>Somers Point, NJ</td>
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<tr>
<td>Oren Sagher, MD</td>
<td>Ann Arbor, MI</td>
</tr>
</tbody>
</table>
ACTIVE (PROVISIONAL) MEMBERS (Cont.)

Kamram Sahракar, MD
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Mark K. Thompson, MD
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Jeffrey S. Weinberg, MD
New York, NY
<table>
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<tr>
<th>Name</th>
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<tr>
<td>David M. Weitman, MD</td>
<td>Washington, DC</td>
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<tr>
<td>Timothy M. Wiebe, MD</td>
<td>Jackson, MS</td>
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<td>Brian H. Wieder, MD</td>
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<td>Taylor, MI</td>
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<td>G. Andrew Wilson, MD</td>
<td>Wauwatosa, WI</td>
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<td>Dennis D. Winters, MD</td>
<td>Prairie Village, KS</td>
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<td>Miami Beach, FL</td>
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<td>Neill M. Wright, MD</td>
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<td>Kennedy Yalamanchili, MD</td>
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<td>Bakhtier Yamin, MD</td>
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<td>Joseph S. Yazdi, MD</td>
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<td>Peter J. Yeh, MD</td>
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<td>Michael S. Yoon, MD</td>
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<tr>
<td>Julie E. York, MD</td>
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<tr>
<td>Andrew S. Youkilis, MD</td>
<td>Ann Arbor, MI</td>
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<tr>
<td>Gregory J. Zipfel, MD</td>
<td>Gainesville, FL</td>
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### Thursday, April 23, 1998

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<tr>
<th>Event</th>
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<tr>
<td>AANS Long Range Planning Committee &amp; Breakfast</td>
<td>7:00 AM – 11:00 AM</td>
<td>Philadelphia Marriott Room 411/412</td>
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<tr>
<td>AANS Finance and Investment Committee</td>
<td>11:00 AM – 2:30 PM</td>
<td>Philadelphia Marriott Room 411/412</td>
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<tr>
<td>Council of State Neurosurgical Societies Office</td>
<td>12:00 NOON – 5:00 PM</td>
<td>Philadelphia Marriott Registration II</td>
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<tr>
<td>AANS Executive Committee</td>
<td>2:30 PM – 5:30 PM</td>
<td>Philadelphia Marriott Room 411/412</td>
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### Friday, April 24, 1998

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<tr>
<td>AANS Board of Directors Breakfast</td>
<td>7:00 AM – 7:30 AM</td>
<td>Philadelphia Marriott Salon G</td>
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<tr>
<td>Council of State Neurosurgical Societies Office</td>
<td>7:00 AM – 5:00 PM</td>
<td>Philadelphia Marriott Registration II</td>
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<tr>
<td>Council of State Neurosurgical Societies</td>
<td>7:00 AM – 5:00 PM</td>
<td>Philadelphia Marriott Room 309/310</td>
</tr>
<tr>
<td>AANS Board of Directors Meeting</td>
<td>7:30 AM – 11:30 PM</td>
<td>Philadelphia Marriott Salon E</td>
</tr>
<tr>
<td>Council of State Neurosurgical Societies Executive Committee</td>
<td>10:00 AM – 12:00 NOON</td>
<td>Philadelphia Marriott Room 309/310</td>
</tr>
<tr>
<td>Council of State Neurosurgical Societies Luncheon</td>
<td>12:00 PM – 1:00 PM</td>
<td>Philadelphia Marriott Salon H</td>
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<tr>
<td>THINK FIRST Office</td>
<td>12:00 NOON – 5:00 PM</td>
<td>Philadelphia Marriott Room 404</td>
</tr>
<tr>
<td>Congress of Neurological Surgeons Executive Committee Meeting and Lunch</td>
<td>12:00 PM – 5:30 PM</td>
<td>Philadelphia Marriott Room 401/402</td>
</tr>
</tbody>
</table>
Council of State Neurosurgical Societies
Medical Practices Committee Meeting
Philadelphia Marriott
Room 302

Council of State Neurosurgical Societies
Work Force Committee Meeting
Philadelphia Marriott
Room 303

Council of State Neurosurgical Societies
Reimbursement Committee Meeting
Philadelphia Marriott
Room 304

Council of State Neurosurgical Societies
Health Systems Committee Meeting
Philadelphia Marriott
Room 305

Council of State Neurosurgical Societies
Medical/Legal Committee Meeting
Philadelphia Marriott
Room 306

Council of State Neurosurgical Societies
Plenary Session
Philadelphia Marriott
Salon G

Council of State Neurosurgical Societies
Reference Hearings
Philadelphia Marriott
Salon G

Council of State Neurosurgical Societies
Beverage Break
Philadelphia Marriott
Salon H Foyer

Council of State Neurosurgical Societies
Reference Committee
Philadelphia Marriott
Room 501

Council of State Neurosurgical Societies
Northwest Quadrant Caucus
Philadelphia Marriott
Room 303

Council of State Neurosurgical Societies
Northeast Quadrant Caucus
Philadelphia Marriott
Room 304

Council of State Neurosurgical Societies
Southwest Quadrant Caucus
Philadelphia Marriott
Room 305

Council of State Neurosurgical Societies
Southeast Quadrant Caucus
Philadelphia Marriott
Room 306
<table>
<thead>
<tr>
<th>Event</th>
<th>Time</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Young Physicians Committee</td>
<td>7:00 PM – 8:00 PM</td>
<td>Philadelphia Marriott Room 308</td>
</tr>
<tr>
<td>Council of State Neurosurgical Societies</td>
<td>7:00 AM – 5:00 PM</td>
<td>Philadelphia Marriott Room 304</td>
</tr>
<tr>
<td>AANS Annual Meeting Office</td>
<td>7:00 AM – 5:00 PM</td>
<td>Room 304</td>
</tr>
<tr>
<td>Speaker Ready Room</td>
<td>7:00 AM – 5:00 PM</td>
<td>Room 201B</td>
</tr>
<tr>
<td>Marshall’s Headquarters</td>
<td>7:00 AM – 5:00 PM</td>
<td>Room 107A</td>
</tr>
<tr>
<td>Think First Office</td>
<td>7:00 AM – 5:00 PM</td>
<td>Philadelphia Marriott Room 404</td>
</tr>
<tr>
<td>Council of State Neurosurgical Societies Office</td>
<td>7:00 AM – 5:00 PM</td>
<td>Philadelphia Marriott Registration II</td>
</tr>
<tr>
<td>Council of State Neurosurgical Societies Conference Room</td>
<td>7:00 AM – 5:00 PM</td>
<td>Philadelphia Marriott Room 309/310</td>
</tr>
<tr>
<td>AANS Registration</td>
<td>7:00 AM – 5:30 PM</td>
<td>Registration Bridge</td>
</tr>
<tr>
<td>Council of State Neurosurgical Societies/ American Association of</td>
<td>7:30 AM – 9:00 AM</td>
<td>Philadelphia Marriott Room 302</td>
</tr>
<tr>
<td>Neurological Surgeons Caucus</td>
<td></td>
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<tr>
<td>Council of State Neurosurgical Societies/ Congress of Neurological</td>
<td>7:30 AM – 9:00 AM</td>
<td>Philadelphia Marriott Room 307</td>
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<tr>
<td>Surgeons Caucus</td>
<td></td>
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</tr>
<tr>
<td>Council of State Neurosurgical Societies Northwest Quadrant Caucus</td>
<td>7:30 AM – 9:00 AM</td>
<td>Philadelphia Marriott Room 303</td>
</tr>
<tr>
<td>Council of State Neurosurgical Societies Northeast Quadrant Caucus</td>
<td>7:30 AM – 9:00 AM</td>
<td>Philadelphia Marriott Room 304</td>
</tr>
<tr>
<td>Council of State Neurosurgical Societies Southwest Quadrant Caucus</td>
<td>7:30 AM – 9:00 AM</td>
<td>Philadelphia Marriott Room 305</td>
</tr>
<tr>
<td>Council of State Neurosurgical Societies Southeast Quadrant Caucus</td>
<td>7:30 AM – 9:00 AM</td>
<td>Philadelphia Marriott Room 306</td>
</tr>
<tr>
<td>Time</td>
<td>Activity</td>
<td>Room</td>
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<tr>
<td>8:00 AM – 12:00 NOON</td>
<td><strong>Practical Clinic 001:</strong> Spinal Biomechanics</td>
<td>Room 114</td>
</tr>
<tr>
<td>8:00 AM – 5:00 PM</td>
<td><strong>Practical Clinic 002:</strong> Microsurgical Anatomy of the Ventricles, Deep Cisterns, and Cranial Nerves</td>
<td>Room 103B/C</td>
</tr>
<tr>
<td>8:00 AM – 5:00 PM</td>
<td><strong>Practical Clinic 003:</strong> Surgical Anatomy of the Thoracic and Lumbar Spine</td>
<td>Room 108A/B</td>
</tr>
<tr>
<td>8:00 AM – 5:00 PM</td>
<td><strong>Practical Clinic 040:</strong> Second Pallidotomy Accord</td>
<td></td>
</tr>
<tr>
<td>9:00 AM – 12:00 NOON</td>
<td><strong>Council of State Neurosurgical Societies Plenary Session</strong></td>
<td>Philadelphia Marriott</td>
</tr>
<tr>
<td>9:00 AM – 10:30 AM</td>
<td><strong>Think First Finance Committee</strong></td>
<td>Room 406</td>
</tr>
<tr>
<td>9:00 AM – 2:00 PM</td>
<td><strong>Journal of Neurosurgery Editorial Board</strong></td>
<td>Philadelphia Marriott</td>
</tr>
<tr>
<td>11:00 AM – 12:30 PM</td>
<td><strong>Think First Resource Development Committee</strong></td>
<td>Room 406</td>
</tr>
<tr>
<td>12:00 NOON – 2:00 PM</td>
<td><strong>Council of State Neurosurgical Societies Luncheon with AANS and CNS</strong></td>
<td>Philadelphia Marriott</td>
</tr>
<tr>
<td>1:00 PM – 2:30 PM</td>
<td><strong>Practical Clinic 034:</strong> An Introductory Tour of the Internet</td>
<td>Room 102A/B</td>
</tr>
<tr>
<td>1:00 PM – 3:00 PM</td>
<td><strong>Think First Program Advisory Committee</strong></td>
<td>Room 406</td>
</tr>
<tr>
<td>1:00 PM – 5:00 PM</td>
<td><strong>Think First Workshop</strong></td>
<td>Philadelphia Marriott</td>
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<tr>
<td>1:00 PM – 5:00 PM</td>
<td><strong>Think First Exhibits</strong></td>
<td>Philadelphia Marriott</td>
</tr>
<tr>
<td>1:00 PM – 5:00 PM</td>
<td><strong>Practical Clinic 004:</strong> Trigeminal Neuralgia</td>
<td>Room 204C</td>
</tr>
<tr>
<td>1:00 PM – 5:00 PM</td>
<td><strong>Practical Clinic 005:</strong> Cranio-Cervical Junction: Approaches and Treatment</td>
<td>Room 203A/B</td>
</tr>
</tbody>
</table>
Practical Clinic 006: 1:00 PM – 5:00 PM
Cervical Spine Instability: Instrumentation and Other Methods of Management
Room 111A/B

Practical Clinic 007 1:00 PM – 5:00 PM
Interactive Image Guided Neurosurgery
Room 113B/C

Practical Clinic 008: 1:00 PM – 5:00 PM
Ablative Neurosurgery for Pain
Room 105A/B

Practical Clinic 009: 1:00 PM – 5:00 PM
Building a Booming Practice Using Lumbar Stenosis: The Science and Marketing Program (SMART)
Room 114

Practical Clinic 031: 1:00 PM – 5:00 PM
Microsoft® Word for Windows® 95 Fundamentals
Room 110A/B

AANS Outcomes Committee 1:00 PM – 5:00 PM
Philadelphia Marriott
Room 410

AANS Board of Directors Meeting 2:00 PM – 3:30 PM
Philadelphia Marriott
Salon E

Practical Clinic 035: 3:00 PM – 4:30 PM
An Introductory Tour of the Internet
Room 102A/B

AANS/CNS Joint Officers Meeting 3:30 PM – 5:30 PM
Philadelphia Marriott
Conference Suite 3

American Brain Injury Consortium Meeting 5:00 PM – 6:30 PM
Philadelphia Marriott
Room 407/408

THINK FIRST Hospitality Suite 5:30 PM – 7:00 PM
Philadelphia Marriott
Room 411/412

Sunday, April 26, 1998

AANS Annual Meeting Office 7:00 AM – 5:00 PM
Room 304

Marshal’s Headquarters 7:00 AM – 5:00 PM
Room 107A

Speaker Ready Room 7:00 AM – 5:00 PM
Room 201B

Council of State Neurosurgical Societies Office 7:00 AM – 5:00 PM
Philadelphia Marriott
Registration II

Think First Office 7:00 AM – 5:00 PM
Philadelphia Marriott
Room 404
AANS Registration 7:00 AM – 5:30 PM
Registration Bridge

Practical Clinic 036: 8:00 AM – 9:30 AM
An Introductory Tour of the Internet
Room 102A/B

Practical Clinic 010: 8:00 AM – 12:00 NOON
Neuroendoscopy: Indications, Techniques and Instrumentation
Room 202A/B

Practical Clinic 011: 8:00 AM – 12:00 NOON
Palidotomy: Stereotactic Treatments on Movement Disorders
Room 112A/B

Practical Clinic 012: 8:00 AM – 12:00 NOON
Stereotactic Spine Surgery: Techniques and Applications
Room 113A

Practical Clinic 013: 8:00 AM – 12:00 NOON
Lumbar Inter-Body Fusion and Interbody Techniques
Room 108A

Practical Clinic 014: 8:00 AM – 12:00 NOON
Thoracic and Lumbar Stabilization Techniques
Room 111A/B

Practical Clinic 015: 8:00 AM – 12:00 NOON
Lateral Approaches to Tumors and Aneurysms: Application of the Transcondylar, Far Lateral and Extreme Approaches
Room 103B/C

Practical Clinic 016: 8:00 AM – 12:00 NOON
Practical and Technical Aspects of Transsphenoidal and Transoral Surgery
Room 104A/B

Practical Clinic 017: 8:00 AM – 12:00 noon
Surgical Anatomy for Residents
Room 114

Practical Clinic 018: 8:00 AM – 12:00 NOON
Carotid Endarterectomy: An Interactive Video Clinic
Room 103A

Practical Clinic 032: 8:00 AM – 12:00 NOON
Microsoft® Excel for Windows 95® Fundamentals
Room 110A/B

Practical Clinic 019: 8:00 AM – 5:00 PM
Critical Care for the Neurosurgeon
Room 204B

Practical Clinic 020: 8:00 AM – 5:00 PM
Temporal Bone Anatomy
Room 201C

Practical Clinic 021: 8:00 AM – 5:00 PM
Surgical Exposure, Decompression and Stabilization of the Cervical Spine
Room 109A/B

American Brain Injury Consortium 9:00 AM – 11:00 AM
Philadelphia Marriott
Salon D
AANS Summit Meeting
Philadelphia Marriott
Room 305/306
9:00 AM – 12:30 PM

AANS Publications Committee
Philadelphia Marriott
Salon C
9:00 AM – 3:00 PM

Think First Workshop
Philadelphia Marriott
Salon H
9:00 AM – 5:00 PM

Think First Exhibits
Philadelphia Marriott
Salon I/J
9:00 AM – 5:00 PM

Practical Clinic 037:
An Introductory Tour of the Internet
Room 102A/B
10:00 AM – 11:30 AM

Assessment of Quality Committee
Philadelphia Marriott
Room 304
12:00 NOON – 2:00 PM

Think First Executive Committee
Philadelphia Marriott
Room 406
12:00 PM – 5:00 PM

Research Foundation Corporate Advisory Luncheon
Philadelphia Marriott
Room 302/303
12:30 PM – 2:00 PM

Practical Clinic 038:
An Introductory Tour of the Internet
Room 102A/B
1:00 PM – 2:30 PM

Press Room
Room 302
1:00 PM – 5:00 PM

Practical Clinic 022:
Brain Mapping and Epilepsy Surgery
Room 107B
1:00 PM – 5:00 PM

Practical Clinic 023:
Anterior and Anterior Lateral Approaches to Tumors and Aneurysms
Room 103B/C
1:00 PM – 5:00 PM

Practical Clinic 024
Surgical Techniques in Intracranial Aneurysms
Room 203A/B
1:00 PM – 5:00 PM

Practical Clinic 025:
Stereotactic Surgery: Principles, Techniques and Instrumentation
Room 204A
1:00 PM – 5:00 PM

Practical Clinic 026:
Neuroendoscopy: Indications, Techniques and Instrumentation
Room 202A/B
1:00 PM – 5:00 PM

Practical Clinic 027:
Pallidotomy: Stereotactic Treatments on Movement Disorders
Room 112A/B
1:00 PM – 5:00 PM
Practical Clinic 028: Stereotactic Spine Surgery: Techniques and Applications  Room 113A
1:00 PM – 5:00 PM

Practical Clinic 029: Lumbar Inter-Body Fusion and Interbody Techniques  Room 108A
1:00 PM – 5:00 PM

Practical Clinic 030: Thoracic and Lumbar Stabilization Techniques  Room 111A/B
1:00 PM – 5:00 PM

Practical Clinic 033: Microsoft® PowerPoint for Windows 95® Fundamentals  Room 110A/B
1:00 PM – 5:00 PM

Research Foundation Corporate Advisory Committee Philadelphia Marriott  Room 305/306
2:00 PM – 3:00 PM

Guidelines Committee Philadelphia Marriott  Room 304
2:00 PM – 4:00 PM

Practical Clinic 039: An Introductory Tour of the Internet  Room 102A/B
3:00 PM – 4:30 PM

Neurosurgery Editorial Board Philadelphia Marriott  Salon B
3:00 PM – 5:00 PM

Metastasis Guidelines Committee Philadelphia Marriott  Room 308
3:00 PM – 5:00 PM

AANS Public Relations Committee Philadelphia Marriott  Room 303
3:00 PM – 5:00 PM

Research Foundation Executive Council Philadelphia Marriott  Room 305/306
3:00 PM – 5:00 PM

American Brain Injury Consortium Philadelphia Marriott  Salon C/D
3:00 PM – 6:00 PM

Military Neurosurgeons Philadelphia Marriott  Salon A
3:30 PM – 5:30 PM

Tumor Satellite Symposium Planning Committee Philadelphia Marriott  Room 301
5:00 PM – 6:00 PM

AANS/CNS Section on Pain Executive Committee Philadelphia Marriott  Room 302
5:00 PM – 6:30 PM

Opening Reception Grand Hall
6:30 PM – 9:00 PM

Monday, April 27, 1998

AANS Annual Meeting Office 6:00 AM – 5:30 PM

62
Room 304
Speaker Ready Room
Room 201B

6:00 AM – 5:30 PM

6:00 AM – 5:30 PM

6:00 AM – 5:30 PM

6:30 AM – 5:30 PM

6:30 AM – 5:30 PM

6:30 AM – 5:30 PM

6:45 AM – 7:30 AM

7:00 AM – 5:00 PM

7:00 AM – 5:00 PM

7:00 AM – 5:00 PM

7:00 AM – 5:00 PM

8:00 AM – 12:00 NOON

8:00 AM – 12:00 NOON

9:00 AM – 3:00 AM

9:00 AM – 4:30 PM

9:45 AM – 11:35 AM

11:40 AM – 12:15 PM

12:15 PM – 1:00 PM

1:00 PM – 2:30 PM

1:00 PM – 2:45 PM

1:00 PM – 2:45 PM

Room 201B
Marshals Headquarters
Room 107A
AANS Registration
Registration Bridge
Breakfast for Seminar Participants
Grand Hall
Council of State Neurosurgical Societies Office
Philadelphia Marriott
Registration II
Think First Office
Philadelphia Marriott
Room 404
Breakfast Seminars (Numbers 101-121)
Various Room–See Monday Program
3-D Video Seminar
Room 204A
Think First Workshop
Philadelphia Marriott
Salon H
Think First Exhibits
Philadelphia Marriott
Salons I/J
Press Room
Room 302
View Exhibits & Posters
Exhibit Halls A/B/C
Plenary Session I
Ballroom A/B
Special Lecture II:
Decade of the Brain Medalist
Ballroom A/B
Presidential Address
Ballroom A/B
American Board of Pediatric Neurological Surgery Executive Committee
Philadelphia Marriott
Room 308
AANS/CNS Section on Neurotrauma and Critical Care Executive Committee
Philadelphia Marriott
Room 305
AANS/CNS Section on Tumors Executive Council
Philadelphia Marriott
Room 303
AANS/CNS Section on Cerebrovascular Surgery Executive Council 1:00 PM – 2:45 PM Philadelphia Marriott Room 310

SMART Committee 1:00 PM – 2:45 PM Philadelphia Marriott Room 501

AANS/CNS Section on Disorders of the Spine and Peripheral Nerves Executive Committee 1:00 PM – 2:45 PM Philadelphia Marriott Salon J

NEUROSURGERY://ON-CALL® Editorial Board 1:00 PM – 2:45 PM Philadelphia Marriott Room 407

*By invitation only.

Past President’s Luncheon 1:00 PM – 3:00 PM
Four Seasons Hotel Logan Room
(Transportation will be provided)

Surgical Neurology Editorial Board 1:15 PM – 2:30 PM Philadelphia Marriott Room 306

Van Wagenen Fellowship Luncheon 1:15 PM – 2:30 PM Philadelphia Marriott Room 302

WFNS Spine Committee 1:30 PM – 2:45 PM Philadelphia Marriott Room 304

Professional Development Committee 1:30 PM – 2:45 PM Philadelphia Marriott Room 309

Scientific Session I 2:45 PM – 5:15 PM Room 113A/B/C

Scientific Session II 2:45 PM – 5:15 PM Room 103A/B/C

Scientific Session III 2:45 PM – 5:15 PM Room 114

Scientific Session IV 2:45 PM – 5:15 PM Room 108A/B

AANS Annual Business Meeting 5:15 PM – 6:15 PM 204B/C

CPT Task Force 6:00 PM – 7:30 PM Philadelphia Marriott Room 301

AASAN Meeting 6:15 PM – 7:00 PM Philadelphia Marriott Room 306

University of Miami Alumni Reception 6:15 PM – 7:30 PM Philadelphia Marriott Room 309

*By invitation only.
University of Oklahoma Alumni Reception
Philadelphia Marriott
Room 407

Massachusetts General Hospital Alumni Reception
Philadelphia Marriott
Room 501

Indiana University Alumni Reception
Philadelphia Marriott
Room 411

Albany Medical College Reception
Philadelphia Marriott
Room 302/303

Einstein/Montefiore Alumni Reception
Philadelphia Marriott
Room 405

Northwestern University Alumni Reception
Philadelphia Marriott
Room 408

Temple University Reception
Philadelphia Marriott
TBD

Neurological Institute of New York Reception
Philadelphia Marriott
Room 401

Mayo Clinic Neurosurgery Alumni Reception
Philadelphia Marriott
Room 402

New York University Medical College Alumni Reception
Philadelphia Marriott
Room 412

Yale Neurosurgery Alumni Reception
Philadelphia Marriott
Room 403

University of Pittsburgh Reception
Philadelphia Marriott
Salon J

Cleveland Clinic Alumni Reception
Philadelphia Marriott
Room 414/415

The Dandy Society
Philadelphia Marriott
Room 309

Young Neurosurgeons Committee
Philadelphia Marriott
Salon D

Duke University Alumni Reception
Philadelphia Marriott
Room 310

Wayne State University Reception
Philadelphia Marriott
Salon B
Stanford Neurosurgery Alumni Reception
Philadelphia Marriott
Salon K

Neurosurgery Reception
Philadelphia Marriott
Salon L

University of Iowa Friends Reception
Philadelphia Marriott
Room 410

Baylor University Neurosurgery Alumni Reception
Philadelphia Marriott
Room 409

University of Michigan Alumni Reception
Vesper Club
223 South Sydenham Street

Tuesday, April 28, 1998

AANS Annual Meeting Office
Room 304
6:00 AM – 5:30 PM

Speaker Ready Room
Room 201B
6:00 AM – 5:30 PM

Marshal’s Headquarters
Room 107A
6:30 AM – 5:30 PM

AANS Registration
Registration Bridge
6:30 AM – 5:30 PM

Breakfast for Seminar Participants
Grand Hall
6:45 AM – 7:30 AM

Think First Office
Philadelphia Marriott
Room 404
7:00 AM – 5:00 PM

Breakfast Seminars (Numbers 201-221)
Various Rooms—See Tuesday Program
7:30 AM – 9:30 AM

3-D Video Seminar
Room 204A
7:30 AM – 9:30 AM

Press Room
Room 302
9:00 AM – 3:00 PM

View Exhibits & Posters
Exhibit Halls A/B/C
9:00 AM – 4:30 PM

Plenary Session 2
Ballroom A/B
9:45 AM – 11:25 AM

The Richard C. Schneider Lecture
Ballroom A/B
11:30 AM – 12:00 NOON

Cushing Oration
Ballroom A/B
12:00 NOON – 1:00 PM

AANS Membership Committee
Philadelphia Marriott
Room 302
1:00 PM – 2:30 PM
Physicians Reimbursement Committee  
Philadelphia Marriott  
Room 413

WFNS Administrative Council Meeting  
Philadelphia Marriott  
Room 307

AANS/CNS Pediatric Section Executive Committee  
Philadelphia Marriott  
Room 303

ASPN Executive Committee  
Philadelphia Marriott  
Room 301

AANS/CNS Section on Neurotrauma and Critical Care Executive Committee  
Philadelphia Marriott  
Room 305

AANS Archives Committee  
Philadelphia Marriott  
Room 309

Congress of Neurological Surgeons Education Committee  
Philadelphia Marriott  
Salon B

NEUROSURGERY://ON-CALL® Public Pages  
Philadelphia Marriott  
Room 409

AANS/CNS Section on Disorders of the Spine and Peripheral Nerves Scientific Planning  
Philadelphia Marriott  
Room 308

*Cushing Oration Luncheon  
Philadelphia Marriott  
Salons C/D

AANS/CNS Section on Functional and Stereotactic Neurosurgery Executive Committee  
Philadelphia Marriott  
Room 304

Professional Development Committee  
Philadelphia Marriott  
Room 401/402

Ethics and Human Values Committee  
Philadelphia Marriott  
Room 306

Special Session:  
AANS/CNS Section on Cerebrovascular Surgery  
Room 114

Special Session:  
AANS Section on History  
Room 108A/B

Special Session:  
AANS/CNS Section on Pediatric Neurosurgery  
Room 103A/B/C

*By invitation only.
Special Session:  
AANS/CNS Section on Stereotactic & Functional Neurosurgery  
Room 113A/B/C  
2:45 PM – 5:30 PM

Thomas Jefferson University Reception  
Philadelphia Marriott  
Room 403  
6:00 PM – 7:30 PM

Women in Neurosurgery  
Philadelphia Marriott  
Salon I  
6:00 PM – 8:00 PM

STASCIS Investigators  
Philadelphia Marriott  
Room 303  
6:00 PM – 9:00 PM

Brigham and Women’s Alumni Reception  
Philadelphia Marriott  
Room 402  
6:30 PM – 8:00 PM

Matson Lecturer Reception  
Philadelphia Marriott  
Rooms 407/408/409  
6:30 PM – 8:00 PM

University of Pennsylvania Reception  
Philadelphia Marriott  
Room 407/408  
6:30 PM – 8:30 PM

New York University Alumni Reception  
Philadelphia Marriott  
Room 405  
7:00 PM – 9:00 PM

President’s Reception  
University of Pennsylvania  
Museum of Archaeology and Anthropology  
Chinese Rotunda  
(Transportation will be provided)  
7:00 PM – 9:00 PM

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**Wednesday, April 29, 1998**

AANS Annual Meeting Office  
Room 304  
6:00 AM – 5:30 PM

Speaker Ready Room  
Room 201B  
6:00 AM – 5:30 PM

Marshal’s Headquarters  
Room 107A  
6:30 AM – 5:30 PM

AANS Registration  
Registration Bridge  
6:30 AM – 5:30 PM

Breakfast for Seminar Participants  
Grand Hall  
6:45 AM – 7:30 AM

Breakfast Seminars (Numbers 301-320)  
Various Rooms—See Wednesday Program  
7:30 AM – 9:30 AM

3-D Video Seminar  
Room 204A  
7:30 AM – 9:30 AM

Press Room  
Room 302  
9:00 AM – 3:00 PM
View Exhibits & Posters
Exhibit Halls A/B/C
9:00 AM – 3:30 PM

Scientific Session V
Room 103A/B/C
9:45 AM – 11:15 AM

Scientific Session VI
Room 108A/B
9:45 AM – 11:15 AM

Scientific Session VII
Room 113A/B/C
9:45 AM – 11:15 AM

Scientific Session VIII
Room 114
9:45 AM – 11:15 AM

Special Lecture III
Ballroom A/B
11:25 AM – 11:45 AM

Special Symposium
Ballroom A/B
11:45 AM – 12:45 AM

Young Neurosurgeons Luncheon Session
Room 204B
12:45 PM – 1:45 PM

AANS Research Foundation Grant Recipient Luncheon
Philadelphia Marriott
Salon I
1:00 PM – 2:45 PM

American Academy of Neurology
Philadelphia Marriott
Room 305
1:00 PM – 2:45 PM

Special Session:
AANS/CNS Section on Pain
Room 108A/B
2:45 PM – 5:30 PM

Special Session:
AANS/CNS Section on Disorders of the Spine and Peripheral Nerves
Room 113A/B/C
2:45 PM – 5:30 PM

Special Session:
AANS/CNS Section on Neurotrauma and Critical Care
Room 103A/B/C
2:45 PM – 5:30 PM

Special Session:
AANS/CNS Section on Tumors
Room 114
2:45 PM – 5:30 PM

International Reception
Philadelphia Marriott
Salons C/D
5:45 PM – 6:30 PM

Celebrate Neurosurgery!
AANS Annual Reception & Dinner
Crystal Tea Room
6:30 PM – 11:00 PM

Thursday, April 30, 1998

AANS Annual Meeting Office
Room 304
6:00 AM – 12:00 NOON

Speaker Ready Room
Room 201B
6:00 AM – 12:00 NOON
Marshal’s Headquarters
Room 107A  6:30 AM – 12:00 NOON

AANS Registration
Registration Bridge  6:30 AM – 12:00 NOON

Breakfast for Seminar Participants
Grand Hall  6:45 AM – 7:30 AM

AANS Adjourned Board of Directors Meeting
204A  8:00 AM – 12:00 NOON

Breakfast Seminars (Numbers 401-415)
Various Rooms—See Thursday Program  7:30 AM – 9:30 AM

Special Course I
Computer Technology  9:45 AM – 12:00 NOON
Room 113A/B/C

Special Course II
Neuroendoscopy  9:45 AM – 12:00 NOON
Room 103A/BC

Special Course III
Surgical Neuro-oncology  9:45 AM – 12:00 NOON
Room 108A/B

AANS Post-Convention Meeting
110 A/B  12:00 NOON – 1:00 PM
Spouse Program

(All tours depart from the Philadelphia Marriott–12th Street entrance)

Saturday, April 25, 1998

Registration and Ticket Sales 7:00 AM – 5:30 PM
Pennsylvania Convention Center
Registration Bridge

No. 501/502–Philadelphia for the Entire Family: An Overview Tour
1:00 PM – 5:00 PM

No. 503–Welcome Armchair Tour of Philadelphia
Philadelphia Marriott
Room 411/412
3:00 PM – 3:45 PM

Sunday, April 26, 1998

Spouse Hospitality Suite 7:00 AM – 4:00 PM
Philadelphia Marriott
JW's

Registration and Ticket Sales 7:00 AM – 5:30 PM
Pennsylvania Convention Center
Registration Bridge

No. 504–Fitness Plus 7:30 AM – 8:30 PM
Philadelphia Marriott
Salon L

No. 505–Welcome Armchair Tour of Philadelphia
Philadelphia Marriott
Room 411/412
8:00 AM – 8:45 AM

No. 506–A Morning of Culture and Education: Gardens and Garden Sculptures
Philadelphia Marriott
Salon A
9:00 AM – 10:30 AM

No. 507–The Barnes Foundation 9:00 AM – 12:00 NOON

No. 508–Your Brandywine Valley Experience: Longwood Gardens and the Brandywine River Museum
9:00 AM – 5:00 PM

No. 509–Walking Tour of Society Hill 1:30 PM – 5:30 PM

No. 510/511–Valley Forge National Historical Park 2:00 PM – 5:00 PM

Opening Reception 6:30 PM – 9:00 PM
Pennsylvania Convention Center
Grand Hall

Monday, April 27, 1998

Registration and Ticket Sales 6:30 AM – 5:30 PM
Pennsylvania Convention Center
Registration Bridge

Spouse Hospitality Suite 7:00 AM – 4:00 PM
Philadelphia Marriott
JW's
No. 512–Yoga
Philadelphia Marriott
Salon L

No. 514–The Avenue of the Arts: A Walking Tour
8:30 AM – 11:00 AM
Philadelphia Marriott
Salon L

No. 515–Conservation Center for Art and Historic Artifacts
8:45 AM – 11:00 AM
Philadelphia Marriott
Salon A

No. 513–A Morning of Culture and Education:
Ceramics
9:00 AM – 10:30 AM
Philadelphia Marriott
Salon A

View Exhibits & Posters
9:00 AM – 4:30 PM
Pennsylvania Convention Center
Exhibit Halls A/B/C

No. 516–Spouse Program and Luncheon:
The History of American Furniture
11:30 AM – 2:00 PM
Crystal Tea Room

Presidential Address
12:15 PM – 1:00 PM
Ballroom A/B

No. 517–Rittenhouse Square
2:00 PM – 5:00 PM

No. 518–The Beauty of Art Nouveau Jewelry
3:00 PM – 4:00 PM
Philadelphia Marriott
Salon A

Tuesday, April 28, 1998

Registration and Ticket Sales
6:30 AM – 5:30 PM
Pennsylvania Convention Center
Registration Bridge

Spouse Hospitality Suite
7:00 AM – 4:00 PM
Philadelphia Marriott
JW's

No. 519–Fitness Plus
7:30 AM – 8:30 AM
Philadelphia Marriott
Salon L

No. 521–Littlest Street: A Walking Tour of Washington Square West
8:30 AM – 11:00 AM

No. 520–A Morning of Culture and Education:
Rugs
9:00 AM – 10:30 AM
Philadelphia Marriott
Salon A

View Exhibits & Posters
9:00 AM – 4:30 PM
Pennsylvania Convention Center
Exhibit Halls A/B/C

No. 522–Winterthur and the Historic Houses of Odessa
9:00 AM – 5:00 PM

No. 523–Your Pennsylvania Dutch Adventure
9:00 AM – 5:00 PM

Cushing Oration
12:00 NOON – 1:00 PM
Ballroom A/B
*Cushing Oration Luncheon
Philadelphia Marriott
Salons C/D

1:00 PM – 2:45 PM

No. 524/525–University of Pennsylvania Museum of Archaeology and Anthropology

1:30 PM – 4:30 PM

President's Reception
Museum of Archaeology and Anthropology
Chinese Rotunda
(Transportation will be provided)

7:00 PM – 9:00 PM

Wednesday, April 29, 1998

Registration and Ticket Sales
Pennsylvania Convention Center
Registration Bridge

6:30 AM – 5:30 PM

Spouse Hospitality Suite
Philadelphia Marriott
JW's

7:00 AM – 4:00 PM

No. 526–Yoga
Philadelphia Marriott
Salon L

7:30 AM – 8:30 AM

No. 527–A Morning of Culture and Education:
American Paintings
Philadelphia Marriott
Salon A

9:00 AM – 10:30 AM

No. 528–Still Another Philadelphia: Pennsylvania Hospital and The Masonic Temple

8:30 AM – 11:00 AM

View Exhibits & Posters
Pennsylvania Convention Center
Exhibit Halls A/B/C

9:00 AM – 3:30 PM

No. 529–The Houses of Fairmount Park and The Philadelphia Museum of Art

9:30 AM – 4:30 PM

No. 530–Outdoor Sculpture: A Walking Tour of Philadelphia’s Public Treasures

1:00 PM – 4:00 PM

Celebrate Neurosurgery!
AANS Annual Reception & Dinner
Crystal Tea Room
(Transportation will be provided)

6:30 PM – 11:00 PM

Thursday, April 30, 1998

Registration and Ticket Sales
Pennsylvania Convention Center
Registration Bridge

6:30 AM – 12:00 NOON

Spouse Hospitality Suite
Philadelphia Marriott
JW's

7:00 AM – 12:00 NOON

*By invitation only.
<table>
<thead>
<tr>
<th>Company</th>
<th>Booth</th>
<th>Address</th>
<th>Phone</th>
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<tbody>
<tr>
<td>Acra-Cut, Inc.</td>
<td>601</td>
<td>989 Main Street, Acton, MA 01720</td>
<td>(978) 263-2210</td>
</tr>
<tr>
<td>AcroMed</td>
<td>401</td>
<td>3303 Carnegie Avenue, Cleveland, OH 44115</td>
<td>(216) 432-6851</td>
</tr>
<tr>
<td>Ad-Tech Medical Instrument Corp.</td>
<td>1240</td>
<td>1901 William Street, Racine, WI 53404</td>
<td>(800) 776-1555</td>
</tr>
<tr>
<td>Advanced Neuromodulation Systems</td>
<td>1734</td>
<td>One Allentown Parkway, Allen, TX 75002</td>
<td>(972) 390-9800</td>
</tr>
<tr>
<td>Advanced Spine Fixation Systems, Inc.</td>
<td>1038</td>
<td>18 Technology Drive, #110, Irvine, CA 92618</td>
<td>(714) 453-1290</td>
</tr>
<tr>
<td>Advantage Electronic Billing</td>
<td>227</td>
<td>23961 Calle Magdalena, #113, Laguna Hills, CA 92653</td>
<td>(714) 206-4614</td>
</tr>
<tr>
<td>Aesculap</td>
<td>901</td>
<td>1000 Gateway Blvd., South San Francisco, CA 94080</td>
<td>(415) 876-7000</td>
</tr>
<tr>
<td>Aloka</td>
<td>1527</td>
<td>10 Fairfield Blvd., Wallingford, CT 06492</td>
<td>(203) 269-5088</td>
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<tr>
<td>American Red Cross</td>
<td>110</td>
<td>8111 Gatehouse Road, Falls Church, VA 22042</td>
<td>(703) 206-7537</td>
</tr>
<tr>
<td>American Surgical Sponges, Div.</td>
<td>808</td>
<td>82 Sanderson Avenue, Lynn, MA 01902</td>
<td>(781) 592-7200</td>
</tr>
<tr>
<td>AnatoMark Systems</td>
<td>229</td>
<td>99 Hayden Avenue, Lexington, MA 02173</td>
<td>(781) 402-3430</td>
</tr>
<tr>
<td>The Anspach Companies</td>
<td>839</td>
<td>4500 Riverside Drive, Palm Beach Gardens, FL 33410</td>
<td>(561) 627-1080</td>
</tr>
</tbody>
</table>
Applied Fiberoptics  
100 East Chestnut Avenue  
Westmont, IL 60559  
(630) 325-5500

Aries Systems Corporation  
200 Sutton Street  
North Andover, MA 01845  
(978) 975-7570

Arrow International  
2400 Bernville Road  
Reading, PA 19605  
(610) 378-0131

ATL Ultrasound  
22100 Bothell-Everett Highway  
Bothell, WA 98021  
(425) 487-7000

Axon Instruments, Inc.  
1101 Chess Drive  
Foster City, CA 94404  
(650) 571-9400

Axon Systems, Inc.  
400-2200 Oser Avenue  
Hauppauge, NY 11788  
(516) 436-5112

B&K Medical Systems, Inc.  
267 Boston Road  
North Billerica, MA 01862  
(800) 876-7226

Bayer Corporation, Pharmaceutical Division  
400 Morgan Lane  
West Haven, CT 06516  
(203) 812-2000

Baylis Medical Company, Inc.  
5253 Boulevard Decarie  
Montreal, Quebec H3W 3C3  
Canada  
(514) 488-9801

BFW  
750 Enterprise Drive  
Lexington, KY 40510  
(606) 231-8411

Biodynamics International, Inc.  
1719 Route 10, Suite 314  
Parsippany, NJ 07054  
(201) 359-8444

Bionx Implants, Inc.  
1777 Sentry Pkwy, W.  
Gwynedd Hall, Suite 400  
Blue Bell, PA 19422  
(215) 643-5000

Bio-Vascular, Inc.  
2575 University Avenue  
St. Paul, MN 55114  
(612) 603-3716
Blackstone Medical, Inc. Booth 343
90 Brookdale Drive
Springfield, MA 01104
(413) 731-8711

BOSS Instruments, Ltd. Booth 1533
1310 Central Court
Hermitage, TN 37076
(615) 885-2231

BrainLAB USA, Inc. Booth 1201
3120 Hansen Way
Palo Alto, CA 94304
(800) 982-7229

The Bremer Group Company Booth 1442
11243-5 St. Johns Industrial Pkwy. S.
Jacksonville, FL 32246
(904) 645-0004

Buxton BioMedical, Inc. Booth 1613
24 Dartmouth Road
Mt. Lakes, NJ 07046
(973) 316-5431

Carl Zeiss, Inc. Booth 827
One Zeiss Drive
Thornwood, NY 10594
(914) 747-1800

Clinical Neuro Systems Booth 218
309 Commerce Drive
Exton, PA 19341
(610) 524-7400

CLOWARD Instruments Booth 509
224 Kaalawai Place
Honolulu, HI 96816
(808) 734-3511

Codman/Johnson & Johnson Booth 101
325 Paramount Drive
Raynham, MA 02767
(508) 828-3246

Colorado Biomedical, Inc. Booth 1600
6851 Highway 73
Evergreen, CO 80439
(303) 674-5447

COMPASS International, Inc. Booth 1427
919 37th Avenue NW
Rochester, MN 55901
(507) 281-2143

Computational Diagnostics, Inc. Booth 1142
5001 Baum Blvd., Suite 426
Pittsburgh, PA 15213
(412) 681-9990

Connell Booth 214
309 Commerce Drive
Exton, PA 19341
(610) 524-7400
<table>
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<td><strong>Cook Incorporated</strong></td>
<td>501</td>
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<tr>
<td>925 South Curry Pike</td>
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<tr>
<td>Bloomington, IN 47402</td>
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<tr>
<td>(812) 339-2235</td>
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<tr>
<td><strong>Cordis/Elektia</strong></td>
<td>327</td>
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<tr>
<td>Newbury Road</td>
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<tr>
<td>Andover, Hampshire SP10 4DR</td>
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<td>England</td>
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<tr>
<td>(44) 1264-345700</td>
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<tr>
<td><strong>Cross Medical Products</strong></td>
<td>1438</td>
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<tr>
<td>5160 Blazer Memorial Parkway</td>
<td></td>
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<tr>
<td>Dublin, OH 43017</td>
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<tr>
<td>(614) 718-0530</td>
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<td><strong>Cyberonics, Inc.</strong></td>
<td>119</td>
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<tr>
<td>16511 Space Center Blvd.</td>
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<tr>
<td>Houston, TX 77058</td>
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<tr>
<td>(281) 228-7200</td>
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<td><strong>Davol, Inc.</strong></td>
<td>1701</td>
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<tr>
<td>100 Sockanossett Crossroad</td>
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<tr>
<td>Cranston, RI 02920</td>
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<tr>
<td>(401) 463-7000</td>
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<tr>
<td><strong>DePuy Motech</strong></td>
<td>636</td>
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<tr>
<td>700 Orthopaedic Drive</td>
<td></td>
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<tr>
<td>Warsaw, IN 46581</td>
<td></td>
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<tr>
<td>(219) 372-7147</td>
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<td><strong>Designs For Vision, Inc.</strong></td>
<td>607</td>
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<tr>
<td>760 Koehler Avenue</td>
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<tr>
<td>Ronkonkoma, NY 11779</td>
<td></td>
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<tr>
<td>(800) 345-4009</td>
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<td><strong>Diversified Diagnostic Products, Inc.</strong></td>
<td>1045</td>
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<tr>
<td>11603 Windfern</td>
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<tr>
<td>Houston, TX 77064</td>
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<tr>
<td>(281) 955-5323</td>
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<td><strong>EBI Medical Systems, Inc.</strong></td>
<td>1400</td>
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<tr>
<td>6 Upper Pond Road</td>
<td></td>
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<tr>
<td>Parsippany, NJ 07054</td>
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<tr>
<td>(800) 526-2579</td>
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<td><strong>Elekta Instruments, Inc.</strong></td>
<td>427</td>
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<tr>
<td>8 Executive Park West</td>
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<tr>
<td>Atlanta, GA 30329</td>
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<tr>
<td>(404) 315-1225</td>
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<td><strong>Elsevier Science</strong></td>
<td>1614</td>
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<tr>
<td>655 Avenue of the Americas</td>
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<tr>
<td>New York, NY 10010</td>
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<tr>
<td>(212) 633-3766</td>
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<tr>
<td><strong>Endius Incorporated</strong></td>
<td>1540</td>
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<tr>
<td>23 West Bacon Street</td>
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<tr>
<td>Plainville, MA 02762</td>
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<tr>
<td>(508) 643-0983</td>
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<tr>
<td><strong>Ethicon, Inc.</strong></td>
<td>101A</td>
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<tr>
<td>Route 22 West</td>
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<tr>
<td>P.O. Box 151</td>
<td></td>
</tr>
<tr>
<td>Somerville, NJ 08876</td>
<td></td>
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<tr>
<td>(908) 218-2517</td>
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</tbody>
</table>
Fehling Surgical Instruments
4981 Falcon Wood Trail
Marietta, GA 30066
(800) 334-5464

FHC, Inc. (Frederick Haer Co.)
9 Main Street
Bowdoinham, ME 04008
(207) 666-8190

Florida Brace Corporation
P.O. Box 1299
Winter Park, FL 32790
(407) 644-2650

Florida Institute for Neurologic Rehabilitation
P.O. Box 1348
Wauchula, FL 33873
(800) 697-5390

Flowtronics, Inc.
10250 North 19th Avenue, #B
Phoenix, AZ 85021
(602) 997-1364

Forefront Publishing
5 River Road, Suite 113
Wilton, CT 06897
(203) 834-0631

Futura Publishing Company
135 Bedford Road
Armonk, NY 10504
(914) 273-1014

GE Medical Systems
P.O. Box 414 (W-439)
Milwaukee, WI 53201
(414) 548-2223

GenSci Regeneration Sciences, Inc.
2250-650 West Georgia Street
Vancouver, BC V6B 4N7
Canada
(604) 683-8330

Gliatech, Inc.
23420 Commerce Park Road
Cleveland, OH 44122
(216) 831-3200

Hall Surgical/Zimmer
11311 Concept Blvd.
Largo, FL 33773
(813) 392-6464

HEALTHSOUTH
One HealthSouth Parkway
Birmingham, AL 35243
(205) 970-3455

IMRIS, Inc.
435 Ellice Avenue
Winnipeg, Manitoba R3B 1Y6
Canada
(204) 984-5196
Innova International, Inc.  
850 S. Greenville Avenue, Suite 114  
Richardson, TX 75081  
(972) 761-0491

Instratek, Inc.  
11210 Steeplecrest, #130  
Houston, TX 77065  
(281) 890-8020

Integrated Surgical Systems, Inc.  
829 West Stadium Lane  
Sacramento, CA 95834  
(916) 646-3487

International Healthcare Devices  
1901 Obispo Avenue  
Long Beach, CA 90804  
(800) 295-2776

Jerome Medical  
305 Harper Drive  
Mooresstown, NJ 08057  
(609) 234-8600

Johnson’s Orthopedic Designs  
1560 Commerce  
Corona, CA 91720  
(909) 278-2031

Karger Publishers  
26 West Avon Road  
P.O. Box 529  
Farmington, CT 06085  
(860) 675-7834

Keeler Instruments  
456 Parkway  
Broomall, PA 19008  
(610) 353-4350

Kilgore International, Inc.  
36 West Pearl Street  
Coldwater, MI 49036  
(517) 279-9123

Kinamed, Inc.  
2192-C Anchor Court  
Newbury Park, CA 91320  
(805) 499-5999

Kirwan Surgical Products, Inc.  
180 Enterprise Drive  
Marshfield, MA 02050  
(781) 834-6600

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

KMedic, Inc.  
190 Veterans Dr.  
Northvale, NJ 07647  
(201) 767-4002
Komet Medical
800 King George Blvd.
Savannah, GA 31419
(912) 925-8525

T. Koros Surgical Instrument Corp.
610 Flinn Avenue
Moorpark, CA 93021
(805) 529-0825

Leibinger
14540 Beltwood Pkwy. E.
Dallas, TX 75244
(972) 392-3636

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

Life Instrument Corporation
14 Wood Road
Braintree, MA 02184
(781) 849-0109

LifeNet
5809 Ward Court
Virginia Beach, VA 23455
(757) 464-4761

Link America, Inc.
321 Palmer Road
Denville, NJ 07834
(973) 328-4333

Linscan Ultrasound
P.O. Box 1217
Rolla, MO 65402
(800) 533-7226

Lippincott-Raven Publishers
227 East Washington Square
Philadelphia, PA 19106
(215) 413-4042

Lone Star Medical Products, Inc.
8733 Knight Road
Houston, TX 77054
(713) 796-0505

Luxtec
326 Clark Street
Worcester, MA 01606
(508) 856-9454

Market Access Partners
Genesee Center One
602 Park Point Drive
Golden, CO 80401
(303) 526-1900

Medical Education & Research Institute
44 S. Cleveland
Memphis, TN 38104
(901) 722-8001

Booth 1503
Booth 1519
Booth 827A
Booth 515
Booth 328
Booth 1728
Booth 1042
Booth 639
Booth 1628
Booth 1733
Booth 114
Booth 133
Booth 1735
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<td>Medirex Systems Corp.</td>
<td>1639</td>
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<tr>
<td>49 Walnut Park, Bldg. #4</td>
<td></td>
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<tr>
<td>Wellesley Hills, MA 02181</td>
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<tr>
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<td>Medtronic, Inc.</td>
<td>1311A</td>
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<tr>
<td>800 53rd Avenue NE</td>
<td></td>
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<tr>
<td>Minneapolis, MN 55421</td>
<td></td>
</tr>
<tr>
<td>(612) 514-5000</td>
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<tr>
<td>Medtronic PS Medical</td>
<td>1311</td>
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<tr>
<td>125 Cremona</td>
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<tr>
<td>Goleta, CA 93117</td>
<td></td>
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<tr>
<td>(805) 968-1546</td>
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<tr>
<td>Midas Rex, L.P.</td>
<td>807</td>
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<tr>
<td>3000 Race Street</td>
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<tr>
<td>Ft. Worth, TX 76111</td>
<td></td>
</tr>
<tr>
<td>(817) 831-2604</td>
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<tr>
<td>MIDCO</td>
<td>1242</td>
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<tr>
<td>5995 Mira Mesa Blvd., Suite B</td>
<td></td>
</tr>
<tr>
<td>San Diego, CA 92121</td>
<td></td>
</tr>
<tr>
<td>(619) 558-5880</td>
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<tr>
<td>Mitaka Kohki Co., Ltd.</td>
<td>1433</td>
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<tr>
<td>5-1-4 Oosawa</td>
<td></td>
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<tr>
<td>Mitaka-shi, Tokyo 181</td>
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<td>Japan</td>
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<tr>
<td>(81) 422-32-1491</td>
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<tr>
<td>Mizuho America</td>
<td>707</td>
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<tr>
<td>123 Brimbal Avenue</td>
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<tr>
<td>Beverly, MA 01915</td>
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<tr>
<td>(978) 921-1718</td>
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<tr>
<td>Moller Microsurgical</td>
<td>1100</td>
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<tr>
<td>7 Industrial Park</td>
<td></td>
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<tr>
<td>Waldwick, NJ 07463</td>
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<td>(201) 251-9592</td>
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<tr>
<td>Mosby/Williams &amp; Wilkins</td>
<td>1608</td>
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<tr>
<td>2043 Woodland Pkwy., Suite 175</td>
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<tr>
<td>Street Louis, MO 63146</td>
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<tr>
<td>(800) 231-2629</td>
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<tr>
<td>MSI Precision</td>
<td>1440</td>
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<tr>
<td>665 Nutt Road</td>
<td></td>
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<tr>
<td>Phoenixville, PA 19460</td>
<td></td>
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<tr>
<td>(800) 322-4674</td>
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<tr>
<td>MTF</td>
<td>1138</td>
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<tr>
<td>125 May Street, Suite 300</td>
<td></td>
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<tr>
<td>Edison, NJ 08837</td>
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<tr>
<td>(732) 661-0202</td>
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<td>Myelotec, Inc.</td>
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<tr>
<td>4000 Northfield Way, Suite 900</td>
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<tr>
<td>Roswell, GA 30076</td>
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<tr>
<td>(770) 664-4656</td>
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<tr>
<td>Nada-Chair</td>
<td>137</td>
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<tr>
<td>2448 Larpenteur Avenue W.</td>
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<tr>
<td>Street Paul, MN 55113</td>
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<tr>
<td>(612) 644-4466</td>
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</table>
Nadia International, Inc.  
Booth 1708  
1807 Slaughter Lane, #200-461  
Austin, TX 78748  
(512) 301-3888

National Biological Labs, Inc.  
Booth 706  
P.O. Box 2496  
Jackson Hole, WY 83001  
(307) 733-0577

National Fibromyalgia Research Assn.  
Booth 1544  
P.O. Box 500  
Salem, OR 97308  
(503) 315-7257

National Library of Medicine  
Booth 1615  
New York Academy of Medicine  
1216 Fifth Avenue  
New York, NY 10029  
(212) 822-7349

NeuroCare Group  
Booth 1117  
8401-102nd Street, Suite 200  
Pleasant Prairie, WI 53158  
(414) 947-4900

Neurodynamics, Inc.  
Booth 1305  
P.O. Box 603, Lenox Hill Station  
New York, NY 10021  
(800) 551-6070

Newport Medical International  
Booth 1143  
27660 Woodside Road  
Shorewood, MN 55331  
(612) 474-6672

Nicolet Biomedical, Inc.  
Booth 806  
5225-2 Verona Road  
Madison, WI 53711  
(608) 273-5000

NOMOS Corporation  
Booth 243  
2591 Wexford Bayne Road, Suite 400  
Sewickley, PA 15143  
(412) 934-8200

NS Recruitment, Inc.  
Booth 1345  
P.O. Box 36096  
Louisville, KY 40233  
(888) 673-6096

NYMOX Corporation  
Booth 128  
5516 Nicholson Lane, Suite 100A  
Rockville, MD 20895  
(800) 93NYMOX

OEC Medical Systems, Inc.  
Booth 943  
384 Wright Brothers Drive  
Salt Lake City, UT 84116  
(801) 328-9300

Olympus America, Inc.  
Booth 1039  
2 Corporate Center Drive  
Melville, NY 11747  
(516) 844-5435
OMI Surgical Products
4900 Charlemar Drive
Cincinnati, OH 45227
(513) 561-2241

OMNA Medical Partners
2255 Glades Road, Suite 416-A
Boca Raton, FL 33431
(561) 988-2227

Omni Medical Designs, Inc.
12296 Hubbard Drive
Livonia, MI 48150
(313) 513-7450

Omni-Tract Surgical
1100 New Brighton Boulevard
Minneapolis, MN 55413
(612) 623-0396

Oratec Interventions, Inc.
3700 Haven Court
Menlo Park, CA 94025
(650) 369-9904

Orion Medical, Inc.
12187 S. Business Park Drive
Draper, UT 84020
(801) 571-9774

Ortho Biotech, Inc.
700 US Highway 202
Raritan, NJ 08869
(908) 704-5000

Orthofix, Inc.
250 E. Arapaho Road
Richardson, TX 75081
(972) 918-8300

Orthopedic Systems, Inc.
30031 Ahern Avenue
Union City, CA 94587
(510) 429-1500

Osteomed Corporation
3750 Realty Road
Dallas, TX 75244
(972) 241-3401

Osteonics
59 Route 17 South
Allendale, NJ 07401
(201) 825-4900

Osteotech, Inc.
51 James Way
Eatontown, NJ 07724
(732) 542-2800

OUR Scientific International, Inc.
1 World Trade Center, Suite 7871
New York, NY 10048
(212) 524-9739
Parke-Davis  
201 Tabor Road  
Morris Plains, NJ 07950  
(201) 540-4182  

The Patient Education Institute  
The University of Iowa  
100 Oakdale Campus, Room W108-OH  
Iowa City, IA 52242  
(319) 335-4613  

Philadelphia Cervical Collar Co.  
P.O. Box 185  
Westville, NJ 08093  
(800) 923-9760  

Philips Medical Systems  
710 Bridgeport Avenue  
Shelton, CT 06484  
(203) 926-7674  

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

Photoelectron  
5 Forbes Road  
Lexington, MA 02173  
(781) 861-2069  

Picker International  
595 Miner Road  
Cleveland, OH 44143  
(440) 473-3544  

PMT Corporation  
1500 Park Road  
Chanhassen, MN 55317  
(800) 626-5463  

Practical Anatomy Workshop  
3839 Lindell Blvd.  
St. Louis, MO 63108  
(314) 535-4000  

Practice Promotion  
4828 12th Avenue South  
Minneapolis, MN 55417  
(612) 827-1874  

Precision Therapeutics, Inc.  
3636 Boulevard of the Allies  
Pittsburgh, PA 15213  
(412) 622-7249  

Quality Medical Publishing, Inc.  
11970 Borman Drive, #222  
St. Louis, MO 63146  
(314) 878-7808  

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

85
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ThermoGenesis Corp. Booth 233
3146 Gold Camp Drive
Rancho Cordova, CA 95670
(916) 858-5100

Thieme Medical Publishers Booth 1620
333 Seventh Avenue
New York, NY 10001
(212) 760-0888

Thompson Surgical Instruments, Inc. Booth 802
10170 E. Cherry Bend Road
Traverse City, MI 49684
(616) 922-0177

Transonic Systems Booth 1145
34 Dutch Mill Road
Ithaca, NY 14850
(607) 257-5300

Transplantation Research Foundation Booth 326
4545 Bissonnet, #285
Bellaire, TX 77401
(713) 666-0518

Universal Pain Technology, Inc. Booth 1703
908-B Powerline Road
Pompano Beach, FL 33069
(954) 968-4075

University of Florida Tissue Bank, Inc. Booth 1402
1 Innovation Drive
Alachua, FL 32615
(904) 462-3097

U.S. Army Recruiting Command Booth 1513
1307 3rd Avenue
Ft. Knox, KY 40121
(502) 626-1979

V. Mueller, Allegiance Healthcare Booth 609
1435 Lake Cook Road
Deerfield, IL 60015
(800) 388-2838

Vasamedics Booth 1238
2963 Yorkton Blvd.
St. Paul, MN 55117
(612) 490-0999

Vital Images Booth 1543
3100 West Lake Street, Suite 100
Minneapolis, MN 55416
(612) 915-8000

Voxel Booth 1339
26081 Merit Circle, Suite 117
Laguna Hills, CA 92653
(714) 348-3200

W.B. Saunders Co. Booth 1626
625 Walnut Street/300 East
Philadelphia, PA 19106
(215) 238-7800
W.L. Gore & Associates, Inc.  
Booth 939  
4747 E. Beautiful Lane #3  
Phoenix, AZ 85044  
(888) 686-4673

Walter Lorenz Surgical, Inc.  
Booth 815  
1520 Tradeport Drive  
Jacksonville, FL 32218  
(800) 874-7711

Welch Allyn, Inc.  
Booth 1606  
4341 State Street Road  
Skaneateles Falls, NY 13153  
(315) 685-4560

Williams & Wilkins  
Booth 1635  
351 W. Camden Street  
Baltimore, MD 21201  
(410) 528-4230

Wright Medical Technology, Inc.  
Booth 236  
5677 Airline Road  
Arlington, TN 38002  
(901) 867-4546

ZEPPELIN Instruments  
Booth 1501  
Gistlstrasse 99  
Pullach D-82049  
Germany  
(49) 89-7936880
1998 AANS ANNUAL MEETING
INSTITUTIONAL EXHIBITORS

AANS
22 S. Washington Street
Park Ridge, IL 60068
(847) 692-9500
Booth 623

AANS Publications
22 S. Washington Street
Park Ridge, IL 60068
(847) 692-9500
Booth 1627

AANS/CNS Joint Section on Pain
22 S. Washington Street
Park Ridge, IL 60068
(847) 692-9500
Booth 140

AANS/CNS Joint Sections
22 S. Washington Street
Park Ridge, IL 60068
(847) 692-9500
Booth 143

Acoustic Neuroma Association
3109 Maple Drive, Suite 406
P.O. Box 12402
Atlanta, GA 30305
(404) 237-8023
Booth 139

American Syringomyelia Alliance Project, Inc.
P.O. Box 1586
Longview, TX 75606
(903) 236-7079
Booth 1541

Brain Injury Association
105 N. Alfred Street
Alexandria, VA 22314
(202) 296-6443
Booth 1731

Congress of Neurological Surgeons
1813 Sixth Avenue, S. MEB504
Birmingham, AL 35294
(205) 934-3546
Booth 138

Journal of Neurosurgery/Neurosurgical Focus
1224 West Main Street, Suite 450
Charlottesville, VA 22903
(804) 924-5503
Booth 701

Neurosurgery
1975 Zonal Avenue, KAM 415
Los Angeles, CA 90033
(213) 342-3001
Booth 1632

NEUROSURGERY://ON-CALL®
22 S. Washington Street
Park Ridge, IL 60068
(847) 692-9500
Booth 700

North American Spine Society
6300 N. River Road, Suite 500
Rosemont, IL 60018
(847) 698-1630
Booth 136
SANS VI
22 S. Washington Street
Park Ridge, IL 60068
(847) 692-9500

THINK FIRST Foundation
22 S. Washington Street
Park Ridge, IL 60068
(847) 692-9500

Trigeminal Neuralgia Assoc.
710 Bayview Avenue
P.O. Box 340
Barnegat Light, NJ 08006
(609) 361-1014

Booth 1633

Booth 1139

Booth 139A
## 1998 AANS Annual Meeting
### Technical Exhibitor Product/Service Listing

### Allografts/Human Tissue

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<tr>
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<td>Biodynamics International, Inc.</td>
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<td>GenSci Regeneration Sciences, Inc.</td>
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<td>LifeNet</td>
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<td>Osteotech, Inc.</td>
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<td>Precision Therapeutics, Inc.</td>
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<td>Transplantation Research Foundation</td>
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<td>University of Florida Tissue Bank, Inc.</td>
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### Anatomical Charts/Models

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<td>Kilgore International, Inc.</td>
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<td>National Biological Labs, Inc.</td>
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### Aneurysm Clips & Accessories

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### Aspirators

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### Bone Growth Stimulators

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<td>Orthofix, Inc.</td>
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### Bone Substitute

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## COMPUTER HARDWARE

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## COMPUTER SOFTWARE FOR OFFICE MANAGEMENT

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## COMPUTER SOFTWARE FOR SURGICAL APPLICATIONS

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## CONTINUING MEDICAL EDUCATION COURSES

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## CRANIOTOMES, DRILLS & ACCESSORIES

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CSF DRAINAGE DEVICES

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CT/MRI/MAGNETIC SOURCE IMAGING

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DURA SUBSTITUTE

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Disclosure Information

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<table>
<thead>
<tr>
<th>Faculty Name</th>
<th>Disclosure</th>
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<tbody>
<tr>
<td>Apfelbaum, Ronald I., MD</td>
<td>Consultant: Aesculap</td>
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<tr>
<td>Bakay, Roy, A.E., MD</td>
<td>Grants/Research Support: NIH</td>
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<td>Barnett, Gene H., MD</td>
<td>Consultant: Picker International</td>
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<td>Barolat, Giancarlo, MD</td>
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<td>Benabid, Alim L., MD, PhD</td>
<td>Other: Medtronic</td>
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<td>Benzel, Edward C., MD</td>
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<td>Berger, Mitchel S., MD</td>
<td>Grants/Research Support: NIH</td>
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<td>Bucholz, Richard D., MD, FACS</td>
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<td>Campbell, James N., MD</td>
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<td>Dickinson, Lawrence D., MD</td>
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<td>During, Matthew, M.D.</td>
<td>Grants/Research Support: Canavan Disease Foundation</td>
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<td>Dickman, Curtis A., MD</td>
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<td>Garton, Hugh, MD</td>
<td>Grants/Research: Medtronic PS Medical and Cordis/Elekta</td>
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<td>Giannotta, Steven L., MD</td>
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Grand, Walter, MD  
Haid, Regis W., Jr., MD  
Haines, Stephen J., MD  
Hamilton, Allan J., MD  
Hardy, Jules, MD  
Harkey, H. Louis, III, MD  
Hassenbusch, Samuel J., MD, PhD  
Heilbrun, M. Peter, MD  
Hopkins, L.N., III, MD  
Jho, Hae-Dong, MD, PhD  
Johnson, J. Patrick, MD  
Kalfas, Iain H., MD  
Kambin, Parviz, MD  
Kestle, John, MD  
Kliot, Michel, MD  
Kondziolka, Douglas S., MD, FRCSC  
Laske, Douglas W., MD  
Levy, Robert M., MD, PhD  
Loftus, Christopher M., MD  
Lozano, Andres M., MD, PhD  
Lunsford, L. Dade, MD  
Maiman, Dennis J., MD, PhD  
Marshall, Lawrence F., MD  
Martin, Neil, MD  
Maroon, Joseph, MD  
McLaughlin, Mark, M.D.
<table>
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<th>Name</th>
<th>Relationship/Company</th>
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<td>Mullan, John F., MD</td>
<td>Grants/Research Support: Cook Inc.</td>
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<td>Muraszko, Karin M., MD</td>
<td>Grants/Research Support: NY Comed Corp.</td>
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<td>North, Richard B., MD</td>
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<td>Oro, John J., MD</td>
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<td>Ray, Charles D., MD</td>
<td>Consultant: Surgical Dynamics</td>
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<td>Stock Shareholder: U. S. Surgical Co.</td>
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<td>Rhoton, Albert, Jr., MD</td>
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<td>Sonntag, Volker, KH, MD</td>
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<td>Other: Codman/Johnson &amp; Johnson</td>
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<td>Sundaresan, Narayan, MD</td>
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<td>Tabar, Vivian, MD</td>
<td>Grants/Research Support: Swiss Foundation for Biomedical Grants</td>
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<td>Tasker, Ronald R., MD, FRCSC</td>
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<td>Tew, John M., Jr., MD</td>
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<td>Stock Shareholder: Ohio Medical Instruments</td>
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<td>Wanebo, John E, MD</td>
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<td>Young, Ronald F., MD</td>
<td>Grants/Research Support: Elekta Radiosurgery, Inc.</td>
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</table>
Saturday Morning

PRACTICAL CLINIC 001

8:00 AM – 12:00 NOON (Room 114)

SPINAL BIOMECHANICS

Director: Edward C. Benzel, MD (Albuquerque, NM)*

Faculty:
Vincent C. Traynelis, MD (Iowa City, IA)*
Eric Woodard, MD (Boston, MA)
Nevan Baldwin, MD (Albuquerque, NM)
Saturday Morning

PRACTICAL CLINIC 002

8:00 AM – 5:00 PM (Room 103B/C)

MICROSURGICAL ANATOMY OF THE VENTRICLES, DEEP CISTERNS, AND CRANIAL NERVES

Director: Albert L. Rhoton, Jr., MD (Gainesville, FL)*

Faculty:
Evandro De Oliveira, MD (Sao Paulo, SP, Brazil)
Alberto C. Cardoso, MD (Gainesville, FL)
Eduardo Seoane, MD (Gainesville, FL)
Xiangen Shi, MD (Gainesville, FL)
Andrew Fine, MD (Gainesville, FL)
Ron Smith, MD (Gainesville, FL)
Tsutomu Hitotsumatsu, MD (Gainesville, FL)
Saturday Morning

PRACTICAL CLINIC 003

8:00 AM – 5:00 PM (Room 108A/B)

SURGICAL ANATOMY OF THE THORACIC AND LUMBAR SPINE

Directors: Richard G. Fessler, MD (Gainesville, FL)*
David Cahill, MD (Tampa, FL)

Faculty:
Christopher Shaffrey, MD (Detroit, MI)
Eric Woodard, MD (Boston, MA)
Paul McCormick, MD (New York, NY)
Andrea Halliday, MD (Dallas, TX)
Sanford Larson, MD (Milwaukee, WI)
Robert Heary, MD (Newark, NJ)
Stephen Ondra, MD (Wilmette, IL)
Bruce McCormack, MD (San Francisco, CA)
Paul Sawin, MD (Phoenix, AZ)
Saturday Afternoon

PRACTICAL CLINIC 004

1:00 PM – 5:00 PM (Room 204C)

TRIGEMINAL NEURALGIA

Director: Jeffrey Brown, MD (Toledo, OH)

Faculty:
Harry Van Loveren, MD (Cincinnati, OH)
G. Robert Nugent, MD (Morgantown, WV)
Jan Gouda, MD (Toledo, OH)
Thomas Lovely, MD (Pittsburgh, PA)
Hae-Dong Jho, MD (Pittsburgh, PA)
Saturday Afternoon

PRACTICAL CLINIC 005

1:00 PM – 5:00 PM (Room 203A/B)

CRANIO-CERVICAL JUNCTION: APPROACHES AND TREATMENT

Director: Arnold Menezes, MD (Iowa City, IA)

Faculty:
Eric Woodard, MD (Boston, MA)
Noel Perrin, MD (New York, NY)
Timothy Ryken, MD (Syracuse, NY)
Vincent C. Traynelis, MD (Iowa City, IA)
Wade Mueller, MD (Milwaukee, WI)
Michael Fehlings, MD (Toronto, ON)
Saturday Afternoon

PRactical Clinic 006

1:00 PM – 5:00 PM (Room 111A/B)

Cervical Spine Instability: Instrumentation and Other Methods of Management

Directors: Volker K.H. Sonntag, MD (Phoenix, AZ)*
Stephen M. Papadopoulos, MD (Ann Arbor, MI)

Faculty:
Regis W. Haid, Jr., MD (Atlanta, GA)*
Iain Kalfas, MD (Cleveland, OH)*
Paul Sawin, MD (Phoenix, AZ)
Joseph T. Alexander, MD (Jacksonville, FL)
Maurice Smith, MD (Memphis, TN)
Charles L. Branch, MD (Winston-Salem, NC)*
Paul Marcotte, MD (Philadelphia, PA)
Christian Zimmerman, MD (Boise, ID)
Gerald E. Rodts, Jr., MD (Atlanta, GA)
Saturday Afternoon

**PRACTICAL CLINIC 007**

1:00 PM – 5:00 PM (Room 113B/C)

**INTERACTIVE IMAGE GUIDED NEUROSURGERY**

**Director:** Robert Maciunas, MD (Nashville, TN)

**Faculty:**
- Richard Bucholz, MD, FACS (St. Louis, MO)
- M. Peter Heilbrun, MD (Salt Lake City, UT)
- Lucia Zamorano, MD (Detroit, MI)
- Jaimie Henderson, MD (St. Louis, MO)
- Patrick J. Kelly, MD (New York, NY)
- Manuel Dujoyovy, MD (Detroit, MI)
- Christopher Duma, MD (Los Angeles, CA)
- Gene Barnett, MD (Cleveland, OH)
- Tyrone Hardy, MD (El Cajon, CA)
- William Tobler, MD (Cincinnati, OH)
- John Adler, Jr., MD (Stanford, CA)
Saturday Afternoon

**PRACTICAL CLINIC 008**

1:00 PM – 5:00 PM (Room 105A/B)

**ABLATIVE NEUROSURGERY FOR PAIN**

**Director:** Nicholas Barbaro, MD (San Francisco, CA)

**Faculty:**
- Michael Munz, MD (Philadelphia, PA)
- Samuel Hassenbusch, MD, PhD (Houston, TX)
- Blaine S. Nashold, Jr., MD (Durham, NC)
- Dennis E. Bullard, MD (Raleigh, NC)
Saturday Afternoon

PRACTICAL CLINIC 009

1:00 PM – 5:00 PM (Room 114)

BUILDING A BOOMING PRACTICE USING LUMBAR STENOSIS: THE SCIENCE AND MARKETING PROGRAM (SMART)

Director: Stanley Pelofsky, MD (Oklahoma City, OK)

Faculty:
Russell Travis, MD (Lexington, KY)
Susan Nowicki (Park Ridge, IL)
Sunday Morning

PRACTICAL CLINIC 010

8:00 AM – 12:00 NOON (Room 202A/B)

NEUROENDOSCOPY: INDICATIONS, TECHNIQUES, AND INSTRUMENTATION

Directors: Kerry Crone, MD (Cincinnati, OH)*
Adam Lewis, MD (Jacksonville, MS)

Faculty:
Antonio DeSalles, MD (Los Angeles, CA)
John Frazee, MD (Los Angeles, CA)
Hae-Dong Jho, MD (Pittsburgh, PA)*
David Jimenez, MD (Columbia, MO)
Charles Teo, MD (Little Rock, AR)
Alan Turtz, MD (Philadelphia, PA)
PRACTICAL CLINIC 011

8:00 AM – 12:00 NOON (Room 112A/B)

PALLIDOTOMY: STEREOTACTIC TREATMENTS OF MOVEMENT DISORDERS

Director: Roy A.E. Bakay, MD (Atlanta, GA)*

Faculty:
Ronald Alterman, MD (Philadelphia, PA)
Jerrold Vitek, MD (Marietta, GA)
L. Dade Lunsford, MD (Pittsburgh, PA)
Frederick Lenz, MD (Baltimore, MD)
Andres Lozano, MD (Toronto, ON)*
Kim Burchiel, MD (Portland, OR)*
Sunday Morning

PRACTICAL CLINIC 012

8:00 AM – 12:00 NOON (Room 113A)

STEREOTACTIC SPINE SURGERY: TECHNIQUES AND APPLICATIONS

Director: Kevin Foley, MD (Memphis, TN)*

Faculty:
Stephen Papadopoulos (Ann Arbor, MI)
Iain H. Kalfas, MD (Cleveland, OH)
Nevan Baldwin, MD (Albuquerque, NM)
Michael Fehlings, MD, PhD (Toronto, ON, Canada)
Sunday Morning

PRACTICAL CLINIC 013

8:00 AM – 12:00 NOON (Room 108A)

LUMBAR INTERBODY FUSION AND INTERBODY TECHNIQUES

Directors: Charles Branch, MD (Winston-Salem, NC)
            Peter Klara, MD (Norfolk, VA)

Faculty:
        Curtis Dickman, MD (Phoenix, AZ)
        Richard Fessler, MD (Gainesville, FL)
        Fred Geisler, MD, PhD (Chicago, IL)
        Robert Heary, MD (Newark, NJ)
        J. Patrick Johnson, MD (Los Angeles, CA)
        Paul J. Marcotte, MD (Philadelphia, PA)
        Michael Potter, MD (Medford, OR)
        Kenneth Yonemura, MD (Seattle, WA)
Sunday Morning

**PRACTICAL CLINIC 014**

8:00 AM – 12:00 NOON (Room 111A/B)

**THORACIC AND LUMBAR STABILIZATION TECHNIQUES**

**Director:** Regis Haid, Jr., MD (Atlanta, GA)*

**Faculty:**
- Eric Woodard, MD (Boston, MA)
- Nevan Baldwin, MD (Albuquerque, NM)
- Scott Erwood, MD (Atlanta, GA)
- Patrick Johnson, MD (Los Angeles, CA)
- Gregory Trost, MD (Madison, WI)
- Christopher Shaffrey, MD (Detroit, MI)
- Joseph Alexander, MD (Jacksonville, FL)
- Gerald Rodts, Jr., MD (Atlanta, GA)
- Brian Cuddy, MD (Charleston, SC)
- Carl Lauryssen, MD (St. Louis, MO)
Sunday Morning

PRACTICAL CLINIC 015

8:00 AM – 12:00 noon (Room 103B/C)

LATERAL APPROACHES TO TUMORS AND ANEURYSMS: APPLICATION OF THE TRANSCONDYLAR, FAR LATERAL, AND EXTREME LATERAL APPROACHES

Director: Jeffrey Keller, PhD (Cincinnati, OH)
Harry R. Van Loveren (Cincinnati, OH)

Faculty:
Mario Zuccarello, MD (Cincinnati, OH)
Michael Chicoine, MD (Cincinnati, OH)
Khaled Aziz, MD (Cincinnati, OH)
Maurali Guthikonda, MD (Detroit, MI)
Troy Payner, MD (Indianapolis, IN)
Michael Link, MD (Jacksonville, FL)
Sunday Morning

PRACTICAL CLINIC 016

8:00 AM – 12:00 NOON (Room 104A/B)

PRACTICAL AND TECHNICAL ASPECTS OF TRANSSPHENOIDAL AND TRANSORAL SURGERY

Director: William Chandler, MD (Ann Arbor, MI)

Faculty:
Mary Louise Hlavin, MD (Cleveland, OH)
Hae-Dong Jho, MD (Pittsburgh, PA)*
Sunday Morning

PRACTICAL CLINIC 017

8:00 AM – 12:00 NOON (Room 114)

SURGICAL ANATOMY FOR RESIDENTS

Director: Albert Rhoton, Jr., MD (Gainesville, FL)*
Sunday Morning

PRACTICAL CLINIC 018

8:00 AM – 12:00 NOON    (Room 103A)

CAROTID ENDARTERECTOMY:
AN INTERACTIVE VIDEO CLINIC

Director: Robert E. Harbaugh, MD (Lebanon, NH)

Faculty:
Issam Awad, MD (New Haven, CT)
Julian Bailes, MD (Maitland, FL)
Christopher Loftus, MD (Iowa City, IA)
Harold Pikus, MD (Key Biscayne, FL)
Philip Stieg, MD (Boston, MA)
PRACTICAL CLINIC 019

8:00 AM – 5:00 PM  (Room 204B)

CRITICAL CARE FOR THE NEUROSURGEON

Director: Jack Wilberger, MD (Pittsburgh, PA)

Faculty:
Alex Valadka, MD (Houston, TX)
Issam Awad, MD (New Haven, CT)
Jeffrey Lobosky, MD (Chico, CA)
Nelson Oyesiku, MD (Atlanta, GA)
Lawrence Dickinson, MD (Ann Arbor, MI)*
P. David Adelson, MD (Pittsburgh, PA)
Christopher Farber, MD (Pittsburgh, PA)
Sunday Morning

PRACTICAL CLINIC 020

8:00 AM – 5:00 PM (Room 201C)

TEMPORAL BONE ANATOMY

Director: Steven Giannotta, MD (Los Angeles, CA)*

Faculty:
John Diaz Day, MD (Burlington, MA)
Takanori Fukushima, MD (Pittsburgh, PA)
Anil Nanda, MD (Shreveport, LA)
Carl Heilman, MD (Boston, MA)
Dennis Maceri, MD (Los Angeles, CA)
Sunday Morning

PRACTICAL CLINIC 021

8:00 AM – 5:00 PM   (Room 109A/B)

SURGICAL EXPOSURE, DECOMPRESSION AND STABILIZATION OF THE CERVICAL SPINE

Directors: Richard Saunders, MD (Lebanon, NH)
           Dennis Vollmer, MD (San Antonio, TX)
           Vincent C. Traynelis, MD (Iowa City, IA)*

Faculty:
       Perry Ball, MD (Lebanon, NH)
       Kevin T. Foley, MD (Memphis, TN)
       Brian Cuddy, MD (Charleston, SC)
       Edward Benzel, MD (Albuquerque, NM)
       Fraser Henderson, MD (Washington, DC)
       Seth Zeidman, MD (Baltimore, MD)
       Timothy Ryken, MD (Syracuse, NY)
       Peter Klara, MD, PhD (Norfolk, VA)
Sunday Afternoon

**Practical Clinic 022**

1:00 PM – 5:00 PM (Room 107B)

**Brain Mapping and Epilepsy Surgery**

**Directors:** Mitchel Berger, MD (San Francisco, CA)
Nicholas Barbaro, MD (San Francisco, CA)

**Faculty:**
George Ojemann, MD (Seattle, WA)
Warwick Peacock, MD (San Francisco, CA)
Carl Sartorius, MD (Indianapolis, IN)
John P. Weaver, MD (Worcester, MA)
Michael M. Haglund, MD, PhD (Durham, NC)
Sunday Afternoon

PRACTICAL CLINIC 023

1:00 PM – 5:00 PM (Room 103B/C)

ANTERIOR AND ANTEROLATERAL APPROACHES TO TUMORS AND ANEURYSMS

Director: Laligam Sekhar, MD (Washington, DC)

Faculty:
Donald Wright, MD (Washington, DC)
Akio Morita, MD (Washington, DC)
Ann Marie Yost, MD (Washington, DC)
Ghassan Bejjani, MD (Washington, DC)
Zachary Levine, MD (Washington, DC)
Russell Buchanan, MD (Washington, DC)
Sunday Afternoon

PRACTICAL CLINIC 024

1:00 PM – 5:00 PM (Room 203A/B)

SURGICAL TECHNIQUES IN INTRACRANIAL ANEURYSMS

Director: Arthur L. Day, MD (Gainesville, FL)

Faculty:
Ralph G. Dacey, Jr., MD (St. Louis, MO)
Robert A. Solomon, MD (New York, NY)
Duke S. Samson, MD (Dallas, TX)
Sunday Afternoon

**Practical Clinic 025**

1:00 PM – 5:00 PM (Room 204A)

**Stereotactic Surgery: Principles, Techniques, and Instrumentation**

**Director:** Robert Breeze, MD (Denver, CO)

**Faculty:**
Richard Bucholz, MD, FACS (St. Louis, MO)
Peter Dempsey, MD (Burlington, MA)
Lucia Zamorano, MD (Detroit, MI)
Richard G. Perrin, MD (Vancouver, BC, Canada)
William Tobler, MD (Cincinnati, OH)
A. Angelo Patil, MD (Omaha, NE)
Robert Maciunas, MD (Nashville, TN)
Roy A.E. Bakay, MD (Atlanta, GA)
Sunday Afternoon

**PRACTICAL CLINIC 026**

1:00 PM – 5:00 PM (Room 202A/B)

**NEUROENDOSCOPY: INDICATIONS, TECHNIQUES, AND INSTRUMENTATION**

**Directors:** Kerry Crone, MD (Cincinnati, OH)*
Adam Lewis, MD (Jacksonville, MS)

**Faculty:**
Antonio DeSalles, MD (Los Angeles, CA)
John Frazee, MD (Los Angeles, CA)
Hae-Dong Jho, MD (Pittsburgh, PA)*
David Jimenez, MD (Columbia, MO)
Charles Teo, MD (Little Rock, AR)
Alan Turtz, MD (Philadelphia, PA)
Sunday Afternoon

PRACTICAL CLINIC 027

1:00 PM – 5:00 PM (Room 112A/B)

PALLIDOTOMY: STEREOTACTIC TREATMENTS
OF MOVEMENT DISORDERS

Director: Roy A.E. Bakay, MD (Atlanta, GA)*

Faculty:
Ronald Alterman, MD (Philadelphia, PA)
Jerrold Vitek, MD (Marietta, GA)
L. Dade Lunsford, MD (Pittsburgh, PA)
Frederick Lenz, MD (Baltimore, MD)
Andres Lozano, MD (Toronto, ON)*
Kim Burchiel, MD (Portland, OR)
Sunday Afternoon

PRACTICAL CLINIC 028

1:00 PM – 5:00 PM  (Room 113A)

STEREOTACTIC SPINE SURGERY: TECHNIQUES AND APPLICATIONS

Director: Kevin T. Foley, MD (Memphis, TN)*

Faculty:
Stephen Papadopoulos (Ann Arbor, MI)
Iain H. Kalfas, MD (Cleveland, OH)
Nevan Baldwin, MD (Albuquerque, NM)
Michael Fehlings, MD, PhD (Toronto, ON, Canada)
Sunday Afternoon

PRACTICAL CLINIC 029

1:00 PM – 5:00 PM    (Room 108A)

LUMBAR INTERBODY FUSION AND INTERBODY TECHNIQUES

Directors: Charles Branch, Jr., MD (Winston-Salem, NC)
            Peter Klara, MD (Norfolk, VA)

Faculty:
          Curtis Dickman, MD (Phoenix, AZ)
          Richard Fessler, MD (Gainesville, FL)
          Fred Geisler, MD, PhD (Chicago, IL)
          Robert Heary, MD (Newark, NJ)
          J. Patrick Johnson, MD (Los Angeles, CA)
          Paul J. Marcotte, MD (Philadelphia, PA)
          Michael Potter, MD (Medford, OR)
          Kenneth Yonemura, MD (Seattle, WA)
Sunday Afternoon

PRACTICAL CLINIC 030

1:00 PM – 5:00 PM (Room 111A/B)

THORACIC AND LUMBAR STABILIZATION TECHNIQUES

Director: Regis Haid, Jr., MD (Atlanta, GA)*

Faculty:
- Eric Woodard, MD (Boston, MA)
- Nevan Baldwin, MD (Albuquerque, NM)
- Scott Erwood, MD (Atlanta, GA)
- Patrick Johnson, MD (Los Angeles, CA)
- Gregory Trost, MD (Madison, WI)
- Christopher Shaffrey, MD (Detroit, MI)
- Joseph Alexander, MD (Jacksonville, FL)
- Gerald Rodts, MD (Atlanta, GA)
- Brian Cuddy, MD (Charleston, SC)
- Carl Lauryssen, MD (St. Louis, MO)
Saturday–Sunday

COMPUTER EDUCATION

PRACTICAL CLINIC 031

SATURDAY
1:00 PM – 5:00 PM (Room 110A/B)

MICROSOFT WORD FOR WINDOWS® 95
FUNDAMENTALS

Director: David Reid (Park Ridge, IL)

PRACTICAL CLINIC 032

SUNDAY
8:00 AM – 12:00 NOON (Room 110A/B)

MICROSOFT EXCEL FOR WINDOWS® 95
FUNDAMENTALS

Director: David Reid (Park Ridge, IL)

PRACTICAL CLINIC 033

SUNDAY
1:00 PM – 5:00 PM (Room 110A/B)

MICROSOFT POWERPOINT FOR WINDOWS® 95
FUNDAMENTALS

Director: David Reid (Park Ridge, IL)
Saturday–Sunday

COMPUTER EDUCATION

Practical Clinics 034 – 039

(Room 102A/B)

AN INTRODUCTORY TOUR OF THE INTERNET AND NEUROSURGERY://ON-CALL®

034 – Saturday, 1:00 – 2:30 PM
035 – Saturday, 3:00 – 4:30 PM
036 – Sunday, 8:00 – 9:30 AM
037 – Sunday, 10:00 – 11:30 AM
038 – Sunday, 1:00 – 2:30 PM
039 – Sunday, 3:00 – 4:30 PM

Director: John Oro, MD (Columbia, MO)*

Faculty:
Joel MacDonald, MD (Tucson, AZ)
David McKalip, MD (Chapel Hill, NC)
Thomas Ellis, MD (Gainesville, FL)
Jaimie Henderson, MD (St. Louis, MO)
Domenic Esposito, MD (Valdosta, GA)
Monday, April 27, 1998

BREAKFAST SEMINARS
6:45 AM – 9:30 AM

Breakfast—Grand Hall
6:45 AM – 7:30 AM

Seminars
7:30 AM – 9:30 AM

#101 ANEURYSM CLIPPING: ADVANCED TECHNIQUES
Room 102A/B

Moderator:
Ralph Dacey, MD (Saint Louis, MO)

Panelists:
Philip E. Stieg, MD, PhD (Boston, MA)
Robert Solomon, MD (New York, NY)
Robert Spetzler, MD (Phoenix, AZ)
H. Hunt Batjer, MD (Chicago, IL)

#102 MICROVASCULAR DECOMPRESSION
Room 105B

Moderator:
John Tew, MD (Cincinnati, OH)*

Panelists:
Peter Jannetta, MD (Pittsburgh, PA)
Takanori Fukushima, MD (Pittsburgh, PA)
Albert Rhoton, Jr., MD (Gainesville, FL)
#103 NEUROSURGICAL MANAGEMENT OF INTRACTABLE PAIN
Room 110A

Moderator:
Richard North, MD (Baltimore, MD)*

Panelists:
Robert Levy, MD (Chicago, IL)*
Kim Burchiel, MD (Portland, OR)*
Samuel Hassenbusch, MD (Houston, TX)*
Yucel Kanpolat, MD (Ankara, Turkey)

#104 PEDIATRIC EPILEPSY
Room 110B

Moderator:
Robert Maxwell, MD (Minneapolis, MN)

Panelists:
Glenn Morrison, MD (Miami, FL)
Paul Kanev, MD (Detroit, MI)
Anne-Christine Duhaime, MD (Philadelphia, PA)
Philip Villanueva, MD (Miami, FL)
Gordon Baltuch, MD (Philadelphia, PA)

#105 CONTROVERSIES IN PEDIATRIC CRANIOFACIAL SURGERY
Room 112B

Moderator:
Gordon McComb, MD (Los Angeles, CA)

Panelists:
Mark Dias, MD (Buffalo, NY)
David Jimenez, MD (Columbia, MO)
James Goodrich, MD (Bronx, NY)
Derek Bruce, MD (Dallas, TX)

#106 SURGERY OF THE CRANIOCERVICAL JUNCTION IN PEDIATRIC PATIENTS
Room 111A

Moderator:
Arnold Menezes, MD (Iowa City, IA)

Panelists:
David Gordon McLone (Chicago, IL)
Gerald Tuite, MD (Saint Petersburg, FL)*
Benjamin Carson, MD (Baltimore, MD)
J. Parker Mickle, MD (Gainesville, FL)
#107  STEREOTACTIC SURGERY FOR MOVEMENT DISORDERS  
Room 104B  

Moderator:  
M. Peter Heilbrun, MD (Salt Lake City, UT)*  

Panelists:  
Robert Grossman, MD (Houston, TX)  
Andres Lozano, MD (Toronto, ON)*  
Roy A.E. Bakay, MD (Atlanta, GA)*  
Alim Benabid, MD (New York, NY)*  

#108  SURGICAL OPTIONS IN EPILEPSY  
Room 104A  

Moderator:  
George Ojemann, MD (Seattle, WA)  

Panelists:  
Guy Clifton, MD (Houston, TX)  
Robert Goodman, MD (New York, NY)  
Webster Pilcher, MD (Rochester, NY)  
Steven Roper, MD (Gainesville, FL)  

#109  EVALUATION AND MANAGEMENT OF TRAUMATIC C1–C2 INSTABILITY  
Room 107B  

Moderator:  
Ronald Apfelbaum, MD (Salt Lake City, UT)*  

Panelists:  
Volker K.H. Sonntag, MD (Phoenix, AZ)*  
Nevan Baldwin, MD (Albuquerque, NM)  
Michael Fehlings, MD (Toronto, ON)  
R. Patrick Jacob, MD (Gainesville, FL)  

#110  PERIPHERAL NERVE INJURY AND ENTRAPMENT: EVALUATION AND MANAGEMENT  
Room 202A  

Moderator:  
David Kline, MD (New Orleans, LA)  

Panelists:  
James Campbell, MD (Baltimore, MD)*  
Allan Friedman, MD (Durham, NC)  
Eric Zager, MD (Philadelphia, PA)  
Allan Belzberg, MD (Baltimore, MD)
#111 MANAGEMENT OF SPINAL CORD TUMORS
Room 202B

Moderator:
Paul Cooper, MD (New York, NY)

Panelists:
Fred Epstein, MD (New York, NY)
Jacques Brotchi, MD (Brussels, Belgium)
Michael Ebersold, MD (Rochester, MN)
Ian McCutcheon, MD (Houston, TX)

#112 MINIMALLY INVASIVE SURGERY OF THE LUMBAR DISC
Room 204B

Moderator:
Richard G. Fessler, MD (Gainesville, FL)*

Panelists:
Kevin T. Foley, MD (Memphis, TN)*
Paul Young, MD (Saint Louis, MO)
Parvin Kambin, MD (Philadelphia, PA)*
Manucher Javid, MD (Madison, WI)

#113 CERVICAL SPONDYLOTIC MYELOPATHY: SURGICAL MANAGEMENT AND LONG-TERM OUTCOME
Room 203B

Moderator:
Ulrich Batzdorf, MD (Los Angeles, CA)

Panelists:
Gary Rea, MD (Columbus, OH)
Richard Saunders, MD (Lebanon, NH)
Barth Green, MD (Miami, FL)
George Sypert, MD (Fort Myers, FL)
#114  SPINAL STABILIZATION: STATE OF THE ART
PRINCIPLES OF SELECTION, APPLICATION,
AND CONSTRUCTION DESIGN
Room 204C

Moderator:
Edward C. Benzel, MD (Albuquerque, NM)*

Panelists:
Vincent C. Traynelis, MD (Iowa City, IA)*
Dennis Maiman, MD (Milwaukee, WI)*
Regis Haid, Jr., MD (Atlanta, GA)*
Eric Woodard, MD (Boston, MA)

#115  NEUROLOGICAL INJURIES AND THE ATHLETE
Room 112A

Moderator:
Michael L.J. Apuzzo, MD (Los Angeles, CA)

Panelists:
Charles Tator, MD (Toronto, ON)
Joseph Maroon, MD (Pittsburgh, PA)
Robert Cantu, MD (Concord, MA)
Arthur L. Day, MD (Gainesville, FL)
Ronald Barnes, MD (East Rutherford, NJ)

#116  CAVERNOUS SINUS SURGERY: TECHNIQUES,
PITFALLS, AND COMPLICATIONS
Room 203A

Moderator:
Ossama Al-Mefty, MD (Little Rock, AR)

Panelists:
Laligam Sekhar, MD (Washington, DC)
Harry Van Loveren, MD (Cincinnati, OH)
J. Diaz Day, MD (Boston, MA)
Wolfgang Koos, MD (Vienna, Austria)
#117  LOW-GRADE GLIOMAS: CURRENT TREATMENT AND CONTROVERSIES
Room 111B

Moderator:
Mitchel Berger, MD (San Francisco, CA)

Panelists:
Michael Salcman, MD (Baltimore, MD)
James Rutka, MD (Toronto, ON)
Kevin Judy, MD (Philadelphia, PA)
Jeffrey Wisoff, MD (New York, NY)

#118  CRANIAL BASE APPROACHES TO THE POSTERIOR FOSSA
Room 109A

Moderator:
Steven Giannotta, MD (Los Angeles, CA)*

Panelists:
Johnny B. Delashaw, MD (Portland, OR)
Donald Wright, MD (Washington, DC)
Jack Rock, MD (Detroit, MI)
Chandranath Sen, MD (New York, NY)

#119  ESTABLISHING STROKE CENTERS AND STROKE TEAMS
Room 109B

Moderator:
Marc Mayberg, MD (Seattle, WA)

Panelists:
Issam Awad, MD (New Haven, CT)
Julian Bailes, MD (Pittsburgh, PA)
Lee Guterman, MD (Buffalo, NY)

#120  FROM LABORATORY TO THE OPERATING ROOM: TECHNOLOGY TRANSFER IN NEUROSURGERY
Room 105A

Moderator:
Forcht Dagi, MD (Atlanta, GA)

Panelist:
George Dohrman, MD (Chicago, IL)
Theodore Roberts, MD (Seattle, WA)
#121  CONSULTANTS CORNER: CEREBROVASCULAR  
Room 201C

Moderator:  
David Piepgras, MD (Rochester, MN)

Panelists:  
Roberto Heros, MD (Miami, FL)  
Duke Samson, MD (Dallas, TX)  
Daniel Barrow, MD (Atlanta, GA)  
Robert Rosenwasser, MD (Philadelphia, PA)

#150  3-D VIDEO SEMINAR—8TH NERVE TUMORS  
Room 204A

Moderator:  
Ken Smith, MD (Saint Louis, MO)

Demonstrating Surgeon:  
Madjid Samii, MD (Hannover, Germany)
Monday Morning

**POSTER PROGRAM**

9:00 AM – 4:30 PM

Exhibit Hall A/B/C

479 posters have been chosen for presentation. All will be on display Monday through Wednesday. Presentations will be given between 1:45 PM and 2:45 PM on Wednesday, April 29, 1998, for uninterrupted viewing.

(See Poster Program for complete listing of papers.)
P A P E R #701  MONDAY, 9:45-10:00 AM
Randomized Trial of Pallidotomy for Parkinson’s Disease

Roy A.E. Bakay
Jerrold L. Vitek
Alan Freeman
Marian L. Evatt
Joanne Green
William McDonald
Michael Habar
Huan Bahr
Shirley Triche
Klaus Mewes
Vijay Chockkan
Jian-Yu Zhang
Natalie Wahlay
Mahlon R. DeLong (Atlanta, GA)

Discussant: David Cahill

KEY WORDS: pallidotomy, Parkinson’s disease

The effect of posterolateral “sensorimotor” pallidotomy on parkinsonian motor signs as well as neuropsychological and psychiatric function was examined.

The beneficial effects of pallidotomy on parkinsonian motor signs have been reported in uncontrolled studies by several centers. Based on the success of these studies, we designed a randomized trial comparing best medical therapy to pallidotomy.

Patients with idiopathic Parkinson’s disease (PD) were assessed pre- and postoperatively using the Unified Parkinson’s Disease Rating Scale (UPDRS), timed tests of motor function, Schwab and England measure of functional independence, as well as a detailed neuropsychological battery and psychiatric evaluation. Following the second of three baseline evaluations, patients were randomized to surgical or medical management. Pallidotomy was performed using electrophysiological guidance. Lesion locations were reconstructed using high-resolution MR images.

Of the 44 patients enrolled, 27 have completed 6-month follow up in surgical (14) and medical control arms (13) of the study. The study was closed after the third interim analysis. Following surgery, there was a statistically significant reduction in the total UPDRS score, from 77 to 51 (p < 0.0001). The medical control arm showed a slight worsening, from 73 to 77. The surgical group also showed significant (p, 0.0001) improvement in the “off” motor and
ADL subscores, improving from 42 to 28, and 23 to 17, respectively. The Schwab and England score improved from 51 to 71% when “off” and from 81 to 89% when “on.” In addition, drug-induced dyskinesias were significantly reduced (p = 0.0019). In the “off” state, improvement in overall motor function was reflected by a substantial reduction in contralateral tremor (p = 0.0012), rigidity (p < 0.001), and bradykinesia (time test 2-point movement [p < 0.0001]). Significant (p < 0.0001) ipsilateral benefit in bradykinesia and rigidity was also observed and sustained over the 6-month follow-up period. Statistical comparisons did not suggest a difference in neuropsychological or psychiatric function between the two groups.

This randomized prospective clinical trial demonstrates significant improvement in the cardinal motor features of PD following unilateral GPI pallidotomy. (Supported by NIH grant #NS-32047.)

PAPER #702 MONDAY, 10:00-10:15 AM
Gamma Knife Radiosurgery for Cerebral Arteriovenous Malformations: A Multi-Institutional Study in Japan

Masaaki Yamamoto (Tokyo, Japan)
Tatsuya Kobayashi (Sendai, Japan)
Hidefumi Jokura (Komak, Japan)
Seiji Fukuoka (Sapporo, Japan)
Hiromichi Hosoda (Chigasaki, Japan)

Discussant: William Friedman*

KEY WORDS: radiosurgery, arteriovenous malformation

No multi-institutional studies on radiosurgery for cerebral AVMs have been published. The results of greater than 3 years’ follow-up data in 885 patients with AVMs who underwent gamma knife radiosurgery between May, 1991, and March, 1994, at one of the 11 gamma knife centers in Japan are analyzed.

Postradiosurgically, 681 (77%) of the 885 patients were periodically examined by means of angiography. Complete nidus obliteration was angiographically confirmed in 443 patients (50%), subtotal obliteration in 114 (13%), shrinkage in 99 (11%), and no significant change in the remaining 25 (3%); these rates correspond to 65%, 17%, 15%, and 4%, respectively, of the 681 patients who had follow-up angiography. Hemorrhages occurred during the postradiosurgical latency period, ranging from 1 to 48 months, in 41 patients (4.6%); annual rates were 2.4% during the first postradiosurgical year, 0.9% during the second, 0.8% during the third, and 0.3% during the fourth. Although 14 (34%) of the 41 patients showed full recovery, 13 (32%) died and the other 14 (34%) have persistent deficits. Radiation-related aggravation of clinical symptoms was seen in 24 (2.7%) of the 885 patients at between 5 and 36 postradiosurgical months. Steroid treatment was required in all 24 patients, 16 of whom recovered completely while the other 8 had persistent deficits.

The overall obliteration rate reported here seems relatively low as compared with those reported previously. However, the annual incidence of (re-)bleeding during the latency period was low as compared with those reported for untreated AVMs.

Monday, 10:15–10:18 AM
Distinguished Service Award Presentation—Mark J. Kubala
(To be introduced by Russell L. Travis, MD)
Posterior C1-2 Transarticular Screw Fixation for Atlantoaxial Arthrodesis

Curtis A. Dickman
Volker K.H. Sonntag (Phoenix, AZ)

Discussant: Kevin T. Foley

KEY WORDS: atlantoaxial instability, arthrodesis, screw fixation

One hundred twenty-one patients were treated for atlantoaxial instability with posterior C1-2 transarticular screws and an autologous wired posterior C1-2 interposition bone strut. Atlantoaxial instability was due to rheumatoid arthritis (n = 48), fractures of C-1 or C-2 (n = 45), transverse atlantal ligament disruption (n = 11), os odontoideum (n = 9), tumors (n = 6), or infection (n = 2). In all, 226 screws were placed. Bilateral C1-2 transarticular screws were placed in 105 patients; 16 patients had only one transarticular screw placed because of an anomalous course of the vertebral artery (n = 13), tumor (n = 1), joint erosion (n = 1), or C-1 fracture (n = 1). All screws were placed under lateral fluoroscopic guidance. All patients had postoperative radiographs and CT scans to assess screw positioning, stability, and osseous union.

Two hundred twenty-one (98%) of the 226 screws were positioned satisfactorily; five were malpositioned (2% malpositioning), none had clinical sequelae. Four of the five malpositioned screws were reoperated on (1 screw was repositioned, 3 screws were removed). There were no neurological complications, strokes, or TIAs associated with the surgical procedures. Long-term follow up was achieved in 114 patients (mean follow-up duration 22 months). One hundred twelve (98%) of the patients achieved fusion. There were two nonunions (1.9%) associated with delayed screw fractures and recurrent C1-2 hypermobility which required occipitocervical fusion. In comparison, our prior C1-2 fixations for similar pathology with C1-2 wires and autograft (n = 89) had an 86% union rate. C1-2 transarticular screw fixation rigidly fixes the unstable motion segments resulting in a significantly higher fusion rate (98% vs 86%) than wiring and grafts alone.

The risk of screw malpositioning and catastrophic vascular or neural injury is small and can be minimized by assessing the position of the foramen transversarium on preoperative CT scans, by using intraoperative fluoroscopy, and by using frameless stereotaxis to guide the screw trajectory.

Results and Expectations From Trigeminal Neuralgia Radiosurgery

Douglas Kondziolka*
L. Dade Lunsford
Bernardo Perez
John C. Flickinger (Pittsburgh, PA)

Discussant: Ronald Apfelbaum

KEY WORDS: radiosurgery, trigeminal neuralgia, pain relief

Over the last 5 years, stereotactic radiosurgery has been performed for the management of patients with trigeminal neuralgia (TGN). Initially, radiosurgery was used for patients with recurrent pain after prior surgeries. More recently radiosurgery has been advocated as a minimally invasive surgical strategy for TGN. Our hypothesis was that radiosurgery could provide pain relief with low risk for facial sensory loss and no other morbidity. We reviewed our 5-year experience in patients and evaluated the factors of age, prior surgical history, radiosurgery dose, multiple sclerosis, imaging appearance of the nerve, and need for further treatment.
One hundred twenty-one patients underwent gamma knife radiosurgery with a 4-mm isocenter (3 patients had a 2-isocenter plan). A maximum dose of 70 Gy (n = 54), 80 Gy (n = 60), 85 Gy (n = 5), or 90 Gy (n = 2) was administered. Prior operations included microvascular decompression (n = 42), glycerol rhizotomy (n = 57), and radiofrequency rhizotomy (n = 19). Mean patient age was 67 years (32–92 years), and the mean duration of pain was 11 months. Seventy-nine patients had radiosurgery with the Model U gamma knife and 42 with the Model B.

Median follow up after radiosurgery was 18 months. Initial improvement was noted in 106 patients (88.3%). Degree of pain relief was coded as total (100% relief off medications) in 64 patients (60.4%); 50 to 90% relief in 18 patients (17%); 10 to 50% relief in 9 patients (9%); and no relief in 15 patients (14%). Significant pain relief was noted by 77% of patients at last follow up. There was no difference in the spectrum of pain relief according to radiosurgery dose, patient age, imaging identification of the nerve, or whether prior surgery had been performed. Patients with multiple sclerosis-related TGN fared worse (p = 0.04). Post-radiosurgery paresthesias (numbness) was described by 12 patients (10%). Relapse of pain was noted in only 6 (10%) of 64 patients who attained complete relief. There was no other morbidity after radiosurgery.

Radiosurgery is a minimally invasive surgical approach for the management of trigeminal neuralgia that is associated with a low rate of facial paresthesia and no other morbidity. At the current range of dose and nerve length irradiated, significant and lasting pain relief was noted in 77% of patients. Radiosurgery should be considered part of the armamentarium for TGN, especially in patients with concomitant medical illness or in whom other surgical procedures have failed.

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**Monday, 10:48–10:51 AM**

**THINK FIRST Announcement**

**Thomas G. Saul, MD (Cincinnati, OH)**

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**PAPER #705 MONDAY, 10:51-11:06 AM**

**Transplantation of Fetal Pig Cells for Parkinson’s Disease: Preliminary Results**

James M. Schumacher (Sarasota, FL)*
Prather Palmer
Samuel A. Ellias (Burlington, MA)
Steven Fink (Boston, MA)
Ole Isacson (Belmont, MA)

Discussant: Robert Breeze

**KEY WORDS:** Parkinson's disease, pig cell transplantation, outcome

This series includes 12 patients with moderate to severe Parkinson's disease for an average of 13.9 years (5–24 years), age 61 years (48–70 years), Hoehn and Yahr stage of 3.7 off, 2.2 on. The patients underwent unilateral transplantation of pig ventral mesencephalic cell suspensions into the striatum in an open-label study. Six patients were immunosuppressed with cyclosporin and six received cells treated with a F(ab)’2 antibody directed to MHC1. Ten patients were tested both presurgically and at 3-month intervals postsurgery using a modified CAPIT protocol including the Unified Parkinson's Disease Rating Scale (UPDRS), timed tests, neuropsychological testing, and home diaries. F-DOPA PET scans were performed.

Specific patients in both treated groups showed clinical improvement. One patient improved 81 points on the UPDRS from 9 months to 21 months after surgery. Two patients showed sustained improvement on the UPDRS at 15 months. Ten patients had an average improvement of 13.2 UPDRS points.
at 6 months (p < 0.05). Seven of 10 patients showed improvement on the UPDRS, UPDRS motor, and UPDRS activities of daily living. There were no major side effects or decrement in neuropsychological scores. One patient died 7 months after surgery of a pulmonary embolism unrelated to the procedure. Autopsy revealed surviving pig dopaminergic grafts in the patient’s striatum. Xenotransplantation of pig neural cells is possible in Parkinson’s disease and has shown clinical efficacy in some patients.

Monday, 11:06–11:21 AM

Special Presentation—Philadelphia Art
Michael Salcman, MD (Baltimore, MD)

Gamma Knife Radiosurgery of Cavernous Sinus Meningiomas: Results of a Two-Institution, 10-Year Experience

Christopher M. Duma (Los Angeles, CA)
L. Dade Lunsford
Douglas Kondziolka
Brian R. Subach (Pittsburgh, PA)
Deane B. Jacques (Los Angeles, CA)

Discussant: Laligam Sekhar

Key Words: radiosurgery, cavernous sinus, meningioma

To evaluate the response of cavernous sinus meningiomas to stereotactic radiosurgery we reviewed the combined 10-year experience, from two institutions, of 92 patients. This review included long-term data in 34 patients (median follow up 5.8 years) from a previously reported series. All patients underwent radiosurgery using a cobalt-60 gamma knife. Fifty-four patients (59%) had histological confirmation of meningiomas (1–5 skull base craniotomies per patient, average 2.6); 38 (41%) were treated on the basis of neuroimaging criteria alone. The single-fraction radiation tumor margin dose (5-20 Gy, median 14 Gy) was designed to conform to the irregular tumor volumes and required an average of 6.9 isocenters.

None of the 92 patients had tumor growth (100% tumor control) during the follow-up interval (median 3.1 years, range 0.3–9.5 years). Tumor regression was observed in 39% of patients imaged at an average of 18 months. Thirteen patients (14%) improved clinically. Three patients (3%) had new or worsened visual field deficits with varying improvement during the follow-up interval. One patient developed deterioration of a previously present 6th nerve paresis. Three patients developed a partial-complex seizure disorder. Two patients died in the follow-up interval of unrelated causes. The overall permanent complication rate was 7.6%. In the initial 34-patient series, followed up to 9.5 years (median 5.8 years), the tumor control rate remained 100%. Nineteen patients (56%) showed tumor regression. The overall complication rate was 8.8%.

Gamma knife radiosurgery is an accurate, safe, and effective adjunct or alternative to craniotomy to prevent growth of tumors involving the cavernous sinus. The long-term results show safety and efficacy. Conformal radiosurgery should be considered the treatment of choice for residual or recurrent meningiomas as well as selected tumors discovered de novo.
SPECIAL LECTURE I
DECADE OF THE BRAIN MEDALIST

11:40 AM – 12:15 PM  (Ballroom A/B)

FROM STEM CELLS TO CIRCUITS, EARLY STEPS IN BRAIN DEVELOPMENT

Ronald D.G. McKay, PhD
(To be introduced by William F. Chandler, MD)
Monday Afternoon

PRESIDENTIAL ADDRESS

12:15 PM – 1:00 PM  (Ballroom A/B)

Edward R. Laws, Jr., MD
(To be introduced by William Shucart, MD)
Monday Afternoon

**SCIENTIFIC SESSION I**

2:45 PM – 5:15 PM  (Room 113A/B/C)

**Moderators:** Russell Travis, MD (Lexington, KY)
Steven Gianotta, MD (Los Angeles, CA)

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**PAPER #707 MONDAY, 2:45-3:00 PM**

Management of Cerebral Vasospasm: Timing of Endovascular Options

Robert H. Rosenwasser
Jeffrey E. Thomas
Patricia Gannon
Rodney Bell
Dara Jamieson (Philadelphia, PA)

Discussant: L.N. Hopkins

**KEY WORDS:** vasospasm, endovascular surgery

This series includes a total of 367 patients with SAH treated between July, 1993, and March, 1997. Ninety-two percent of all patients admitted were operated on within the first 24 hours of admission to our institution, regardless of the day of the original hemorrhage. The remainder were operated on within a 48-hour window pending medical stabilization. Postoperatively, all patients were managed in the routine fashion, with prophylactic hypervolemic hemodilutional therapy, and twice-daily transcranial Doppler ultrasound evaluation. Treatment protocol was extremely aggressive, in that if a patient's neurological deficit from cerebral vasospasm did not reverse within a 60-minute period, the patient was then taken to the interventional suite for angioplasty and papaverine infusion.

Eighty patients (22%) underwent endovascular management during this period of time. Angioplasty protocol involved placement of a No. 9 French femoral sheath, full heparinization, continuation of dextran infusion (40 cc/hr), and general anesthesia. Materials utilized included the ITC silicone balloon, as well as the Stealth system, with inflations of approximately 0.5 to 1 atmosphere for 1- to 5-second inflation periods. Balloon angioplasty was followed by papaverine infusion, administering 300 mg total over a 30- to 45-minute period.

Analysis of the data indicated that there appears to be a 2-hour window in which angioplasty is most successful in reversing the deficit and maintaining a sustained clinical improvement. Forty-nine patients (61%) were treated in < 2 hours. The remainder were treated in a > 2-hour period, but no longer than an 8-hour interval. In those patients treated in < 2 hours, there was a 90% rate of angiographic improvement after treatment, with a 70% rate of immediate and sustained clinical improvement. At > 2 hours, there was 88% angiographic improvement with only a 40% sustained clinical improvement; in this group, asymptomatic salt and pepper reperfusion hemorrhages were noted. No patients developed intracranial hematomas necessitating surgical evacuation or elevating ICP to dangerous levels. Eight patients required retreatment; all were in Fisher Grade III or above, with sustained transcranial Doppler flow velocities > 200 cm/sec. There were no dissections and no surgical hematomas for the entire group treated with angioplasty.
Aggressive and early intervention should be undertaken in patients who are refractory to medical measures to reverse the symptoms of cerebral vasospasm. It appears in this preliminary series that the 2-hour window may be important. Rationales and details of treatment will be discussed.

**PAPER #708 MONDAY, 3:00-3:15 PM**

**Mercury Water and Cauterizing Stones: Nicolaus André and Tic Douloureux**

Jeffrey A. Brown (Toledo, OH)
Catherine Simon
Devedutta G. Sangvai (Groton, MA)
Mark C. Preul (Detroit, MI)

Discussant: Peter Jannetta

**KEY WORDS:** tic douloureux, Nicolaus André, cautery, neurosurgical history

In his 1756 publication, *Observations Pratiques sur les Maladies de l’Urethre et sur Plusiers Faits Convulsifs*, Nicolaus André included a series of 5 case reports wherein he coined the term “tic douloureux” and described a novel treatment for the disease known before as “spasme cynique.” This paper represents the first translation of this aspect of his work into English and reviews André’s understanding of the etiology and treatment of trigeminal neuralgia two centuries ago. André hypothesized that tic douloureux occurs because of peripheral compression of the inferior maxillary or orbital nerves. In his first case there was a traumatic origin to the pain and associated infection, presumably osteomyelitis. The other cases had no known cause. André treated the pain with cauterizing stones and mercury water to bloodlessly expose the peripheral nerve branches over the periosteum of the maxilla. He first compressed and squeezed the associated nerve branches with tweezers, then left the wound open to decompress it.

Testimonies confirm the success of his pain relief, but in two cases there was a recurrence of pain about 6 months after treatment which was treated successfully again with the same technique. His patients were followed for several years and often had had pain for a decade or longer before reaching his attention. André believed in a peripheral etiology and he treated pain both by nerve compression and decompression. He was able to identify a specific trigeminal branch which mediated the triggering sensation and suggested in his observation that the nerve was injured, observing a probable neuroma. By gradually approaching the nerves with cautery he limited the blood loss. He suggested that the clot which formed after direct surgical approach continued to compress the sensitive nerve branch and trigger pain. André’s treatment was successful because he used the modern medical model of clinical and surgical observation, applied anatomy, careful hemostasis, and innovative technique.

**PAPER #709 MONDAY, 3:15-3:30 PM**

**The Value of Clinical Symptoms and Signs in Predicting Shunt Failure**

Hugh Garton*
John Kestle (Vancouver, BC, Canada)
James Drake (Toronto, ON, Canada)

Discussant: Alexa Canady

**KEY WORDS:** shunt failure, grading system, prognosis

Shunt malfunctions are a common occurrence in neurosurgical practice. Predictive values for signs and symptoms are important in deciding which patients need an imaging study and/or transfer to regional care facility. Likewise, symptoms or lack thereof that correlate with a low probability of shunt
failure could simplify management. Data from the recently completed Shunt Design Trial were analyzed. Predictive values were calculated for the signs and symptoms of shunt failure. To refine predictive capability, a Symptom Score, based on a cluster of signs and symptoms, was then developed and assessed.

A total of 570 encounters from 344 patients (median age at entry 71 days) were analyzed. For patients with recent shunt insertion (<5 months), the following factors were most useful in predicting shunt failure: bulging fontanelle (positive predictive value [PPV] = 94.3%, likelihood ratio [LR] = 37.0), CSF leak (PPV = 87.5%, LR = 16.31), decreased level of consciousness (LOC) (PPV = 85.7%, LR = 13.9), fluid tracking around shunt (PPV = 82.8%, LR = 11.2), and nausea and vomiting (PPV = 81.4%, LR = 10.3). For patients further out from shunt insertion (>9 months), fewer factors were predictive: decreased LOC (PPV = 77.8%, LR = 27.5) and nausea and vomiting (PPV = 60%, LR = 11.8). No factors were independently successful in predicting the lack of malfunction in either group. Using the Symptom Score, in the recent insertion group, the lowest LR obtained was 0.2, while in the late group the lowest LR was 0.6.

Effectively ruling out a shunt malfunction based on symptoms alone remained a problem, particularly in the late group. Based on the prevalence of shunt failure of 30% and 10% for the two groupings, with a minimum Symptom Score, the likelihood of malfunction could be decreased to 7% and 6%, respectively.

PAPER #710 MONDAY, 3:30-3:45 PM
Outcome Analysis of Patients With Isolated Neck Pain in the United States: Results of the Cervical Spine Research Society Study

John Davis
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Mohammed Bendebba (Baltimore, MD)
Thomas Ducker (Annapolis, MD)

Discussant: Paul McCormick

Key Words: neck pain, multicenter study, pain treatment

The impact of medical and surgical treatment on neck pain is poorly understood. The Cervical Spine Research Society (CSRS) Study is a multicenter study of a large heterogeneous group of patients who were referred to a specialist for the evaluation and treatment of subacute or chronic recognized cervical spine pain problems. In this study, we focus on patients who have neck pain; patients with radiculopathy or myelopathy were studied separately. The goals of the study are to characterize these patients, assess their outcome with respect to the treatment received, treatment prescribed, and expected outcome, identify the most effective forms of treatment, and examine factors that influence outcome.

A total of 503 patients were enrolled in the study. Of these, 195 (39%) presented with neck pain as the predominant complaint with no associated radiculopathy or myelopathy. Forty-one CSRS surgeons participated in the study. Each study patient completed a CSRS questionnaire on initial presentation and at follow-up appointment after receiving a treatment. The physician then completed a form. Statistical analysis was performed by a blinded third party using the SAS program.

Of 195 patients who presented with neck pain as the predominant clinical feature, 122 (63%) returned for follow up and completed the questionnaire. The prescribed treatment was medical treatment for 101 patients (83%), surgical treatment for 10 (8%), and no treatment for 11 (9%). In actuality, 107 (88%) received medical treatment and 14 (12%) received surgical treatment. In the patients who were prescribed and received surgical treatment (with or without fusion), 11 (79%) still had significant neck pain, with only 21% reporting improvement. The patients receiving medical treatment fared better with 60% improvement. Complete data on medical and surgical treatments, pain scales, social activity, work status, litigation, workers’ compensation, and patient satisfaction will be presented. Factors that influence outcomes in all patients will be discussed in detail.
Currently, it appears there are minimal benefits of surgery for patients with isolated neck pain without signs of myelopathy or radiculopathy, even with fusion. This is the first prospective, multisurgeon study that examines this important issue and correlates with present clinical opinion.

**PAPER #711 MONDAY, 3:45-4:00 PM**

Thoracoscopic Microsurgical Excision of Herniated Thoracic Discs

Curtis A. Dickman (Phoenix, AZ)
Daniel Rosenthal (Frankfurt, Germany)
Luiz Madalozzo
Volker K.H. Sonntag (Phoenix, AZ)

Discussant: David Cahill

**KEY WORDS:** intervertebral disc, thoracoscopy, discectomy

From 1992 through 1996, 55 patients were treated with thoracoscopy to resect herniated thoracic discs. Thirty-six patients presented with myelopathies and 19 had incapacitating thoracic radicular pain. Forty-three patients had a single-level discectomy, 11 had two-level discectomies, and one had a three-level discectomy. The mean operative time for thoracoscopic microdiscectomy was 3 hours 25 minutes (range 80–542 minutes), and the average blood loss was 327 cc (range 124–1500 cc).

Compared to a cohort treated with thoracotomy (n = 18), thoracoscopy involved an average of 1 hour less operative time and less than half the blood loss, chest tube drainage, pain medication usage, and hospital stay. Compared to costotransversectomy for thoracic discs (n = 15), thoracoscopy provided more complete resection of calcified and midline discs because it provided a direct view of the entire anterior surface of the dura. Thoracotomy was associated with significantly greater incidence of prolonged, disabling intercostal neuralgia compared to the mild transient episodes of intercostal neuralgia associated with thoracoscopy (50% vs 16%). Thoracotomy was also associated with a higher incidence of postoperative atelectasis and pulmonary dysfunction than thoracoscopy (30% vs 14%).

Excellent clinical and neurological outcomes were achieved (mean follow up 15 months). Among the 36 myelopathic patients, 22 completely recovered neurologically, 5 had functional improvement with some residual myelopathic symptoms, and 9 had stabilization of their myelopathy. Among the 19 patients with isolated thoracic radiculopathies, 15 completely recovered, and 4 had moderate improvement; none had worsened radicular pain. Thoracoscopic microdiscectomy is a reliable surgical technique that can be performed safely with excellent clinical and neurological results.

**PAPER #712 MONDAY, 4:00-4:15 PM**

Predictive Value of Temporary External Drainage in Normal-Pressure Hydrocephalus

Dimitris Zevgaridis
Julian Krott
Adolf Seichert
Ingo Uttner
Hans-Juergen Reulen (Munich, Germany)

Discussant: Peter McL. Black

**KEY WORDS:** normal-pressure hydrocephalus, lumbar drainage, shunt

The clinical triad of gait disturbance, dementia, and urinary incontinence is often incomplete in normal-pressure hydrocephalus (NPH). Furthermore, the radiological picture is not pathognomonic for the disease. In many patients the effect of shunting becomes manifest only after a few days. Therefore, we
decided to use external lumbar drainage for 3 consecutive days to establish a precise predictive tool in the diagnosis of NPH.

Twenty-two patients admitted in our clinic underwent surgery on the basis of well-established clinical and radiological criteria suggestive of NPH. All 22 patients were studied before operation with measurement of the resorptive capacity of CSF using the constant-rate infusion test (CRIT) and by means of external lumbar drainage (120 ml/d) for 3 days. Gait evaluation with apparative gait-analysis and complete neurological and psychometric investigations were performed before and after drainage and 6 months after operation.

Twenty patients (90.9%) had shown improvement in gait and 12 (54.5%) in cognitive properties in the preoperative evaluation; 19 of them showed improvement after operation. Two patients showed neither preoperative (by means of lumbar drainage) nor postoperative improvement. Only 14 patients (63.6%) had a decreased resorptive capacity of CSF. We conclude that external lumbar drainage is a safe and precise (19/20, 95%) test for predicting the outcome of definitive shunt procedures in patients with NPH. The CRIT showed a sensitivity of 63.6% (14/22).

Microendoscopic Discectomy: Surgical Technique and Initial Clinical Results

Kevin Foley*
Maurice Smith (Memphis, TN)
Discussant: Charles Fager

KEY WORDS: discectomy, endoscopy, lumbar disc disease, radiculopathy

Percutaneous endoscopic approaches to lumbar discectomy remain controversial. In general, they have not proven to be as efficacious as conventional open surgery. We have developed a new endoscopic technique which allows the surgeon to address free fragment disc and bone pathology while providing easy access to the entire lumbar spine. The purpose of this study was to determine the feasibility and efficacy of this novel endoscopic approach to lumbar disc disease.

Forty-one patients with single-level radiculopathy secondary to lumbar disc herniation were treated with microendoscopic discectomy (MED). The patient population consisted of 28 males and 13 females with ages ranging from 23 to 67 years. Disc levels included L2-3 (2 patients), L3-4 (3 patients), L4-5 (11 patients), and L5-S1 (25 patients). Five patients had far-lateral discs; the remaining disc herniations were within the spinal canal. Twenty-five (61%) of 41 cases had free fragment pathology. The surgery was performed under epidural anesthesia on an outpatient basis. A 16-mm tubular retractor was inserted via a small paramedian stab wound. An adjustable endoscope was attached to the retractor. Under direct endoscopic vision, a hemilaminotomy, medial facetectomy, resection of ligamentum flavum, retraction of nerve root, and removal of disc were performed as necessary. Concomitant lateral recess stenosis (12% of cases) was addressed with a specially modified high-speed drill. Follow up ranged from 7 to 18 months, with a mean of 13 months.

All patients had substantial relief of their radiculopathy. Under modified Macnab criteria, all patients had good to excellent results. All patients were discharged home within 6 hours of surgery except our first patient, who sustained a dural tear and was admitted for 48 hours. There were no other complications. Return to work ranged from 2 to 40 days in the non-workers’ compensation population (mean 14 days) and 14 to 107 days in the workers’ compensation population (15% of cases, mean 58 days).

The MED procedure combines standard lumbar microsurgical technique with endoscopy, enabling the surgeon to successfully address free fragment disc pathology and lateral recess stenosis. The endoscopic approach allows for a smaller incision and less tissue trauma than standard open microdiscectomy. Routine outpatient application and the avoidance of general

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anesthesia lower hospital stays and costs. We conclude that MED is both feasible and efficacious for the management of lumbar disc disease.

PAPER #714.MONDAY, 4:30-4:45 PM
Results of Long-Term Follow-Up of 405 Temporal Lobectomies Over a 20-Year Period

Michael O'Connor
Michael Sperling
Joyce Liporace
Joseph Sirven (Philadelphia, PA)
Elizabeth Barry (Baltimore, MD)

Discussant: Mitchel Berger

KEY WORDS: temporal lobectomy, epilepsy, outcome

Temporal lobectomy has been the standard for treating intractable temporal lobe epilepsy for more than the last 50 years. The senior surgeon (MOC) has performed 405 of these operations over the last 20 years, and during that time there has been a gradual change in the evaluation and surgical performance of this procedure. In general, the more recent patients have had a more extensive mesial resection and less extensive lateral resection, and have benefited from pre- and postoperative evaluation that included MRI. Depth electrode implantation has been used more commonly in the last 10 years. All patients undergoing temporal lobectomy performed by MOC between 1974 and 1996 for whom follow up was available were included in this study. As a result, fewer than 10% of the patients operated on in that period have been excluded.

Of the 48 patients followed from our surgical series in the first 12 years (1974-1986), 65% are seizure free and 8% have less than three seizures a year, whereas 27% remain in a higher classification and were therefore judged to have no significant improvement. This category included 8% who had an 80% reduction in their seizure frequency. In the last 10 years of the study (1986-1996), the current classification reveals that 249 (70%) of 357 patients remained seizure free during the year when their status was last evaluated, whereas 28 (8%) of the 357 were Class II (rare seizures, < 3 per year) during that year. In 1986 to 1996, 26 patients were Class III, with an 80% reduction, whereas 21 were Class IV, 2 were Class V, and 10 had died; 21 patients had no classification recorded. Other than one hemiplegia in 1977, there was no significant morbidity or mortality. There were occasionally mild and rarely moderate changes in memory.

Of the patients with temporal lobectomies performed in the last 10 years who have been followed to date, the current classification revealed 78% seizure free or with rare seizures (< 3 per year). This compares with 73% of those with surgery from 1976 to 1986 (p < 0.05). The seizure-free category was slightly higher in the more recent series, being 70% compared to 65% (p = 0.05). The ramifications of these findings and the updated figures will be discussed.

PAPER #715.MONDAY, 4:45-5:00 PM
Deep-Brain Stimulation in Parkinson's Disease: Indications and Results

Giovanni Broggi
Domenico Servello
Angelo Franzini
Ivano Dones
Floriano Girotti (Via Celoria, Italy)

Discussant: Roy A.E. Bakay*

KEY WORDS: brain stimulation, Parkinson's disease, electrode implantation

The efficacy of chronic deep-brain stimulation in the treatment of movement disorders led to renewed interest in surgical therapy for Parkinson's dis-
ease (PD). From 1991 to 1997, 41 patients (pts) underwent this surgery. In the same period, 15 pts underwent lesioning surgery (thalamotomy). We are reporting on 36 pts affected by PD implanted with a deep-brain electrode (Activa Medtronic system mod. 3837) at the INNCB in Milan from January, 1996, to June, 1997. In 29 pts (age 35–75 years, mean 62 years) with disabling lateralized and drug-resistant tremor, the chosen target for electrode implant was the nucleus ventralis intermedius of the thalamus (Vim). One pt underwent bilateral Vim neurostimulation. In 7 pts (age 39–60 years, mean 45 years) affected by dopa-induced on-off phenomena and dyskinesias (2 with bilateral symptoms), the target was the ventroposterolateral part of the pallidum internum. In all pts a postoperative MRI control was performed during the test period (1–3 days). A stereotactic electrode implant procedure by means of fused CT and MR images compared to a stereotactic atlas was performed in all cases. In 2 cases a target error was presumed from the absence of effect and ascertained by postoperative MRI, leading to an immediate repositioning of the electrode. The tremor disappeared in 28 pts (96%) while failure of treatment occurred just in 1 case, due to a severe psychic side effects (unbearable fear and illusions). All pts who underwent pallidal implantation showed a marked reduction or disappearance of dyskinesias and rigidity. There were no deaths.

In spite of the short follow up of these series, the results strongly support the use of this procedure in the treatment of drug-resistant or drug-induced tremor, dyskinesias, and rigidity in selected parkinsonian pts. The advantages and limits of these surgical techniques, as well as the clinical indication to perform neurostimulation or lesioning surgery, will be discussed.

PAPER #716 WEDNESDAY, 5:00-5:15 PM
Prolongation of Survival in Mice With Intracerebral Glioma Treated With Semi-Allogeneic/Syngeneic Fibroblasts

Roberta P. Glick
Terry Lichtor
Edwin DeZoeten
Edward P. Cohen (Chicago, IL)

Discussant: Mark Rosenblum

KEY WORDS: glioma, fibroblast, immunotherapy

The current prognosis for patients with malignant brain tumors remains poor. We have previously shown that prolongation of survival can be achieved in C57BL/6 mice (H-2b) with a syngeneic intracerebral or subcutaneous glioma if treated with allogeneic mouse fibroblasts (H-2k) genetically engineered to secrete interleukin-2 (IL-2). Like other antigens, tumor-associated antigens are recognized by cytotoxic T lymphocytes in the context of determinants specified by the major histocompatibility complex (MHC) class I locus. Since the rejection of allogeneic MHC determinants has the property of an immune adjuvant, immunotherapy of glioma with a cellular immunogen that combines the expression of both syngeneic class I determinants and allogeneic antigens could have advantages over an immunogen that expresses syngeneic or allogeneic determinants alone.

To investigate this question in a mouse glioma model, we further modified allogeneic mouse fibroblasts (H-2k) not only for IL-2 secretion but also for the expression of H-2Kb class I determinants. We tested the immunotherapeutic properties of these semi-allogeneic/syngeneic cells in C57BL/6 mice with G1261 glioma in both subcutaneous and intracerebral models.

The results indicate that C57BL/6 mice with either a subcutaneous or intracerebral glioma treated solely by injection of these IL-2-secreting semi-allogeneic/syngeneic cells significantly prolonged survival compared to untreated mice or mice treated with cells secreting IL-2 or cells lacking H-2Kb determinants. In addition, in some instances the mice treated with the semi-allogeneic/syngeneic cells survived indefinitely, suggesting total eradication of the glioma. Thus, the augmented immune response against glioma in mice treated with the semi-allogeneic/syngeneic IL-2-secreting cells points toward a new form of immunotherapy for patients with malignant intracerebral glioma.
Endoscopic transsphenoidal surgery via a natural nasal cavity can now be performed without the use of a nasal speculum or a transsphenoidal retractor. Endoscopic surgical techniques have evolved into standardized simple and easy ones. The average operation time is now 1 to 2 hours for a pituitary adenoma. Endoscopic transsphenoidal surgery has been performed in 94 patients with pituitary and other skull base lesions at the University of Pittsburgh Medical Center during the period from September 1993 through July 1997. Our patients included 54 females and 40 males (age range 14–88 years; median 38 years). Eighty-one patients had pituitary adenomas and 13 had other skull base lesions. Among 81 patients with pituitary adenomas, 25 patients had microadenomas, 22 had intrasellar macroadenomas, 21 had macroadenomas with suprasellar extension, and 13 had invasive macroadenomas involving the cavernous sinus with suprasellar extension. Forty-one adenomas were hormone-secreting (12 associated with Cushing’s disease, 25 prolactinomas and four acromegaly). Two patients underwent surgery as outpatients and 63 patients stayed in the hospital one postoperative night. No nasal packing was required and postoperative discomfort was minimal.

Ten patients with Cushing’s disease were improved clinically with normal ACTH and urinary cortisol levels. Two patients with incompletely treated Cushing’s disease were treated by gamma knife surgery. Among 25 patients with prolactinomas, 16 were improved clinically with normal prolactin level, four were improved clinically with mildly elevated prolactin levels, and five had residual tumors in the cavernous sinuses. Two patients with residual prolactinomas in the cavernous sinuses were treated with gamma knife surgery and three patients were treated with bromocriptine. Among 40 patients with nonsecreting adenomas, 32 underwent total resection and eight had residual tumors in the cavernous sinuses. Two patients had residual tumors: one was treated with conventional radiation therapy and one was treated by gamma knife surgery. The remaining six patients were observed. Three patients developed CSF leakage postoperatively, which was repaired with endoscopic abdominal fat graft immediately.

Endoscopic transsphenoidal surgery has been performed via a nostril in 94 patients; these patients have experienced a fast recovery, short hospital stay, and minimal morbidities. Evolved surgical techniques and improved surgical tools will be presented with patient outcome.
Treatment of Essential Tremor by Gamma Knife Thalamotomy

Ronald F. Young
Arthur Ginsberg
Sandra S. Vermeulen
Allen Posewitz (Seattle, WA)

Discussant: Ronald Tasker

KEY WORDS: essential tremor, thalamotomy, radiosurgery, tremor

Fourteen patients (9 male, 5 female, mean age 67 years) underwent ventral intermediate nucleus (Vim) thalamotomy using the Leksell gamma knife between February, 1994, and May, 1997, for treatment of medically refractory, disabling essential tremor. The lesions were performed contralateral to the dominant hand in all patients. The target was localized by stereotactic MRI and the lesions were made with a single isocenter, the 4-mm secondary collimator helmet and radiosurgical dose maximum levels between 120 and 140 Gy. Pre- and postoperative assessments were carried out by trained but disinterested observers using the clinical rating scale (CRS) for tremor developed by Fahn, Tolosa, and Marin.

Coincidental with the appearance of lesions on MR images and within 2 to 3 months of treatment all but one patient began to experience a clinically observable progressive decline in tremor and by 6 months after treatment 12 (85.7%) of 14 patients were tremor free or nearly so. Of the other two patients, one had significant improvement in tremor and the other showed no change in tremor with a follow-up period over 1 year. Both the mean global examiners scores and the mean global subjects scores on the CRS showed statistically significant improvements (p < 0.001). Ten of the 14 patients had illegible handwriting and could not drink from a cup preoperatively and 9 (90%) of these 10 were able to write legibly and drink from a cup without spilling the contents by 6 months posttreatment. No complications of any kind were experienced by any of the patients.

We conclude that Vim thalamotomy is a safe and very effective treatment for essential tremor. It is minimally invasive, probably safer than radiofrequency thalamotomy, and probably safer and less expensive than the placement of thalamic stimulators.

Effects of Surgical Revascularization on Outcome of Patients With Pediatric Moyamoya Disease

Tatsuya Ishikawa
Kiyohiro Houkin
Hiroshi Abe (Sapporo, Japan)

Discussant: R. Michael Scott

KEY WORDS: moyamoya disease, revascularization, outcome

We reviewed surgically treated patients with pediatric moyamoya disease to examine whether vasoreconstructive surgeries reduced the risk of recurrent ischemic attacks and changed the overall outcomes in terms of the patients' performance and intellectual status. Sixty-four hemispheres in 34 pediatric moyamoya disease patients who received surgical treatment were examined. We performed a superficial temporal artery to middle cerebral artery (STA-MCA) bypass and encephaloduroarteriomyosynangiosis (EDAMS) on 48 sides (combined group) and indirect bypass surgery such as EDAMS on 16 sides (indirect group). These 34 patients were observed postoperatively from 1 to 14 years (mean ± SD: 6.6 ± 3.8 years) and were examined for the incidence of recurrent ischemic attack. Of the 34 patients, 23 were followed for more than 5 postoperative years, and their overall outcomes in terms of their performance and intellectual status were determined.
Perioperative ischemic events (within 2 weeks after surgery) occurred in 5 surgeries (31%) in the indirect group and in 6 (13%) in the combined group (p = not significant). The incidence of postoperative ischemic events (more than 2 weeks after surgery) was significantly reduced in the combined group (10%) compared to the indirect group (56%, p < 0.01). Of the 23 patients observed for more than 5 years, 7 (30%) were mentally retarded and regarded as having a fair outcome.

Combined surgery (STA-MCA bypass with EDAMS) for pediatric moyamoya disease was more effective in reducing the risk of postoperative ischemic attacks compared to indirect surgery. Surgical revascularization may be effective to prevent intellectual deterioration and improve the overall outcome.

PAPER #720 MONDAY, 3:30-3:45 PM
Genetically Engineered Cytotoxic T Cells Targeted Against Angiogenesis: A Novel Antitumor Strategy

Zoher Ghogawala
Richard C. Mulligan (Boston, MA)

Discussant: Henry Brem*

KEY WORDS: angiogenesis, T cell receptor, gene therapy

The growth of solid tumors including malignant glioma is dependent on angiogenesis. The characterization of the basic molecular mediators of angiogenesis has established a critical interaction between a specific endothelial mitogen called vascular endothelial growth factor (VEGF), which is secreted by many tumors including glioblastoma, and an endothelial receptor called Flk-1, which is upregulated in angiogenic endothelium and is not present in quiescent endothelium. Interruption of the VEGF/Flk-1 interaction has shown promise as an antitumor strategy in several animal models. The objective of this work is to modify T cells genetically so that the cells specifically bind endothelial Flk-1 and cause the lysis of angiogenic endothelial cells.

We have constructed a murine chimeric T cell receptor (TCR) that contains VEGF as its extracellular domain with the hypothesis that the receptor would confer upon T cells the ability to bind and kill Flk-1-expressing cells. This chimeric TCR gene was cloned into a retroviral vector (CMMMP), and retroviral particles were produced by transient transfection of the 293 GPG (VSV G) packaging cell line. Primary mouse T cells were isolated from C57/B6 mouse spleens using CD8+ immunoselection columns.

Transient transfection of 293 GPG cells reproducibly yielded a titer of 5 x 10^6 cfu/ml as determined by Southern blotting and by FACS analysis of infected 3T3 cells. Successful retroviral gene transfer into primary T cells was shown by demonstrating cell surface expression of this chimeric TCR on primary T cells (FACS analysis). Finally, chromium-51 release assays showed that primary T cells transduced with this chimeric TCR are capable of potent and specific cytotoxic activity against Flk-1-expressing cells. At effector/target ratios of 12/1, the cytotoxicity approached 100% as measured by cytotoxicity assays in vitro (p < 0.001, one-way ANOVA).

This work raises the novel concept of directing T cells against angiogenesis as a tumor therapy. Such cells, in principle, would be capable of inhibiting many types of tumors. The next step, which is in progress, is to adoptively transfer anti-angiogenic T cells into glioblastoma-bearing mice in order to assess the antitumor potential of these genetically modified T cells. In anticipation of its use in human T cells, we have constructed a human VEGF-chimeric TCR, which is currently under in vitro evaluation in the laboratory.
Results of Phase I Trials of \(^{131}\text{I}\)-Labeled Anti-Tenascin Monoclonal Antibody Injected via Surgically Created Resection Cavities in Patients With Recurrent Malignant Brain Tumors

John H. Sampson
Allan H. Friedman
Herbert E. Fuchs
Darell D. Bigner
Henry S. Friedman (Durham, NC)

Discussant: Ian Pollack*

**KEY WORDS:** radiolabeled monoclonal antibody, tenascin, recurrent tumor

The murine immunoglobulin G\(_{2b}\) monoclonal antibody 81C6 is reactive against the extracellular matrix protein, tenascin, present in gliomas and some carcinomas but not in normal adult brain. A Phase I trial of a single injection of \(^{131}\text{I}\)-radiolabeled 81C6 (\(^{131}\text{I}\)-81C6) into surgically created resection cavities (SCRCs) was conducted in patients with recurrent malignant brain tumors. Escalating doses of \(^{131}\text{I}\) on a fixed dose of 81C6 were used.

In 34 patients with prior external beam radiation therapy, dose-limiting neurological and hematological toxicity was reached at a \(^{131}\text{I}\) dose of 120 mCi. None of the patients treated with less than 120 mCi developed serious neurological toxicity, and only one other patient developed hematological toxicity. The estimated average absorbed radiation dose to the SCRC interface at the 100 mCi administered activity level was 36,800 cGy. Median time to progression for these patients from the time of \(^{131}\text{I}\)-81C6 treatment was 5.1 weeks (4.1–12.1 weeks, 95% confidence index [CI]). Although there have been no complete or partial responses to date, 14 of 33 evaluable patients had stabilized disease, and the median estimated survival for these patients with recurrent malignant brain tumors was 62 weeks (50.3 weeks-infinity, 95% CI). In 23 patients without prior radiation therapy, the maximum tolerated dose has not been reached with \(^{131}\text{I}\) dosages of 140 mCi. Fifteen of these patients remain alive. Survival times have ranged between 30 and 186 weeks with a median survival time of 82 weeks.

The high levels of radiation that can be delivered by this technique with acceptable toxicity are encouraging and suggest that radiolabeled antibodies specific for tumor-associated antigens may improve current therapy for malignant brain tumors.

**PAPER #722 MONDAY, 4:00-4:15 PM**

Endovascular Cervical Carotid Revascularization

Lee R. Guterman
Ajay K. Wakhloo
L.N. Hopkins (Buffalo, NY)

Discussant: Robert Harbaugh

**KEY WORDS:** carotid stenosis, angioplasty, stent, revascularization

To evaluate the safety and efficacy of endovascular revascularization for cervical carotid stenosis in a high-risk surgical population, we studied 96 patients. These were designated for endovascular revascularization of the carotid artery for one or more of the following reasons: multiple medical comorbidities, unstable angina, restenosis after carotid endarterectomy, radiation-induced stenosis, high cervical bifurcation, and long segment stenosis. A No. 9 French guide catheter was placed into the common carotid artery. Angioplasty was performed at 8 to 12 atm for 30 to 60 seconds. In 62 patients, angioplasty was followed by stent placement. In some cases, transvenous atrial or ventricular pacers were used. In the 62 stented patients, heparin was not used postoperatively unless a new neurological deficit occurred.
In the group that was treated with angioplasty and stent, there were no major and two minor strokes. There were two deaths, one from cardiac arrest in a patient awaiting bypass surgery and one immediately after four-vessel coronary artery bypass grafting and two valve replacements. There was one reversible ischemic neurological deficit in a patient whose fine motor deficits cleared within 30 days. Another patient developed minor fine motor deficits, and MRI revealed a small capsular infarction. One patient suffered from iliac occlusion requiring axillobifemoral bypass. He subsequently developed acute renal failure requiring dialysis. He is neurologically intact. Patients have been followed with carotid Doppler ultrasound at 1, 3, 6, and 12 months after stent placement. One patient developed significant restenosis distal to the stent and required retreatment and an additional stent placement.

Endovascular revascularization of the cervical carotid artery using angioplasty and stent placement is a safe and efficacious procedure in patients considered at high risk for surgical carotid revascularization.

PAPER #723 MONDAY, 4:15-4:30 PM
Mortality After Epilepsy Surgery

Michael Sperling
Harold Feldman
Judith Kinman
Michael O’Connor (Philadelphia, PA)

Discussant: Dennis Spencer

KEY WORDS: epilepsy surgery, mortality risk, seizure relief

The mortality rate is increased in people with epilepsy, particularly those with uncontrolled seizures. Since epilepsy surgery eliminates seizures in some people, we used an epilepsy surgery population to examine how seizure control influences mortality. We tested the hypothesis that patients with complete seizure relief after surgery would have a lower mortality rate than those who had persistent seizures.

We followed 393 patients who had epilepsy surgery between January, 1986, and January, 1996. Of these, 347 had focal resection or transection, and 46 had anterior or complete corpus callosotomy. Survival analysis using a time variable technique was performed, contrasting those who had seizure recurrence with those who remained seizure free. Analyses were adjusted for type of surgery, age at seizure onset, preoperative seizure frequency, and age at surgery, since these variables potentially affect the likelihood of seizure relief.

For the entire cohort, seizure-free patients had a lower mortality rate than those with persistent seizures ($p = 0.0117$), after adjusting for the above variables. This was true as well for the subset of patients with localized resection or transection ($p = 0.0352$). Of 194 patients with seizure recurrence, 11 died, whereas no patients died among 199 with no seizure recurrence. Six deaths were sudden and unexplained, 3 others were probably epilepsy related, and 2 were non-neurological. Most patients who died had seizure reduction after surgery, and 6 had less than monthly seizures. Standardized mortality ratio in patients with recurrent seizures was 4.69, and the risk of death in these patients was 1 in 73 person-years.

Patients who have complete seizure relief after epilepsy surgery have a lower mortality rate than those with persistent seizures. This suggests that epilepsy surgery may reduce the risk of death. In contrast, recurrent seizures, even relatively infrequent ones, carry a high mortality risk.
Factors Associated With Surgical Complications for Basilar Bifurcation Aneurysms: An Analysis of 101 Patients

Peter LeRoux
Rajiv Sethi
J. Paul Elliott (New York, NY)
Gerald Grant
H. Richard Winn (Seattle, WA)

Discussant: John Tew*

KEY WORDS: aneurysm, basilar bifurcation, surgical complication

We retrospectively analyzed 101 patients admitted to Harborview Medical Center to determine the incidence and impact of surgical complications during operative treatment of basilar bifurcation aneurysms. Median aneurysm size was 10 mm (range 3–40 mm). There were 26 unruptured aneurysms and 53 patients in good and 22 in poor clinical grade after aneurysm rupture. Fifty-two (70%) of the 75 patients underwent surgery within 3 days of SAH. Postoperatively all 101 patients underwent head CT and 94 underwent angiography.

The incidence of surgical complications including intraoperative cerebral swelling (4%), aneurysm rupture (16%), or vessel injury (2%) and postoperative radiographic evidence for perforator injury (2%), retraction contusions (4%), aneurysm remnants (6%), or vessel occlusion (6%) was similar to that observed in 434 anterior circulation aneurysms treated during the review period. Intraoperative rupture (p = 0.001), vessel occlusion (p = 0.01), and perforator injury (p = 0.0001) were associated with poor outcome. Surgical complications, however, did not prolong hospital stay. Bivariate analysis was performed to determine what preoperative clinical, CT, and angiographic factors were associated with surgical complications. Intraoperative rupture was more frequent during ruptured aneurysm repair (p = 0.06), particularly during early surgery (p = 0.008). No clinical or radiographic factors were associated with vessel occlusion or aneurysm remnants. Perforator injury was significantly associated with large aneurysm size (p = 0.002) and posterior orientation (p = 0.0001). Among 20 patients who underwent posterior-oriented aneurysm repair, 14 (70%) suffered perforator injury and 15 (75%) had a poor outcome.

We conclude that, overall, patients undergoing surgical repair of basilar bifurcation aneurysms are at no greater risk for surgical complications than patients undergoing anterior circulation aneurysm repair. However, direct surgical repair of posterior-oriented aneurysms may not always be warranted.

Development and Impact of a Craniotomy for Tumor Critical Pathway

Gregory P. Licholai
Peter McL. Black
Sharon P. Kleefield (Boston, MA)

Discussant: Stephen Haines

KEY WORDS: craniotomy, brain tumor, cost/efficiency

Much interest has been focused on health expenditures, but few studies examining the benefits of specific cost-control methods have been reported in peer-reviewed literature. In 1994, a craniotomy for tumor critical pathway was developed at the Brigham and Women's Hospital to determine if length of stay (LOS) and costs could be reduced while not increasing readmission rates, morbidity, mortality, or dissatisfaction. All patients with brain tumors admitted from 1994 to 1996 at our hospital were included. Variables examined were LOS, direct cost per case, hospital charges per case, in-house morbidity and mortal-
ity, 30-day readmission rates, and degree of patient satisfaction. There were 764 subjects enrolled, including 461 pathway and 303 non-pathway patients.

Pathway and non-pathway patients had similar demographics and average age of 49.3 years. It was found that LOS for patients in the critical pathway group was 5.7 days compared to 7.3 days for non-pathway patients. This was statistically significant when adjusted for age, sex, and comorbid factors. Charges per case were $22,299 for pathway and $25,366 for non-pathway patients, a difference of $3,067. Direct cost per case was $12,206 for pathway and $14,812 for non-pathway patients, a difference of $2,606. Readmission rates were 9.8% in pathway and 18.4% in non-pathway patients. In-house morbidity and mortality rates were similar and patient satisfaction, based on follow-up questionnaires, was above 95% in both groups. It was also determined that the only significant variable to affect LOS was presentation through the emergency ward.

A postoperative critical pathway for brain tumor patients is a safe and effective method of controlling costs and reducing LOS without increasing readmission rates, morbidity and mortality, or decreasing satisfaction.

PAPER #726 MONDAY, 5:00-5:15 PM
The Opportunity Costs of Training to Become a Neurosurgeon: Relevance for the Expectation of Appropriate Compensation

Frederick Simeone
Andrew Freese (Philadelphia, PA)
Discussant: John Kusske

Key Words: neurosurgical training, compensation, opportunity cost

As reimbursements for neurosurgical procedures continue to decline, salaries for neurosurgeons are likely to decrease significantly over the next decade. Although neurosurgeons appear to receive high compensation for their work when compared to the general populace, such a comparison fails to account for the extraordinarily high opportunity costs associated with training to become a neurosurgeon. We have calculated opportunity costs of training, including 4 years of college, 4 years of medical school, and 6 or 7 years of residency training. We have also compared these costs to the training costs required for four other careers: lawyer, MBA executive, stockbroker, and truck driver. In deciding career paths, college and medical students often become aware of such considerations, explaining in part the current precipitous decline in applications for anesthesiology residencies. At some point in the decline of compensation for neurosurgical procedures, the effect on the applicant pool for highly qualified neurosurgical residents may become significant.

Economic parameters (salary, tuition costs, costs of living, and debt) were calculated using information obtained from federal sources, trade unions, and professional schools and organizations. Conservative estimates of salary incomes were used and investment income was estimated using the annualized return from five well-established mutual funds for the past 15 years. The cost of living was normalized to the standard of living first of a medical student for 4 years, and thereafter for a resident. For all careers except the truck driver, the analysis of opportunity costs began at the end of college, whereas the truck driver received credit for income and savings associated with working rather than attending college.

If a college senior were to consider starting medical school in 1998 and thereafter enter a 7-year neurosurgery program, by his completion of training in 2009 his indebtedness would approximate $200,000, assuming no parental financial support throughout the training period. In contrast, a truck driver who began work after graduating high school in 1994 would, by 2009, have almost $1 million in savings if he maintained the same standard of living during this entire time period as the neurosurgeon-in-training. Similarly, the savings differential between the neurosurgeon and a moderately successful stockbroker who started at the end of college is also in excess of $1 million. The savings differential between the neurosurgeon and a moderately successful lawyer or
midelevel MBA executive would exceed $700,000. It is impossible to predict the future earning potential of a neurosurgeon in 2009.

As our society continues to permit payors to devalue surgical specialists, in particular neurosurgeons, the implicit and fragile balance of opportunity costs and earnings potential for neurosurgeons becomes threatened. At some point, the many sacrifices, both economic and personal, of becoming a neurosurgeon may no longer be adequately compensated by our society.
PAPER #727 MONDAY, 2:45-3:00 PM
Results of Transsphenoidal Microsurgery for Growth Hormone-Secreting Pituitary Adenomas

Aviva Abosch
J. Blake Tyrrell
Lisa Hannegan
Kathleen Lamborn
Charles B. Wilson (San Francisco, CA)

Discussant: Edward R. Laws, Jr.

Key Words: pituitary adenoma, acromegaly, growth hormone level

We performed a retrospective review of 254 patients with acromegaly who underwent transsphenoidal microsurgical resection of a pituitary adenoma at UCSF, between 1974 and 1992. The purpose of this review was to assess patient outcome following surgical treatment of acromegaly. Of these patients, 173 (68%) had growth hormone (GH) levels below 5 µg/L within 30 days after surgery and were considered to be in remission. An additional 20 patients (8%) were deemed to have technical persistence of disease, defined as a single elevated GH level within the first 30 postoperative days, and subsequent normal GH levels with clinical remission of acromegalic symptoms in the absence of further therapy. True disease persistence was found in 61 cases (24%). Overall initial success rate was thus 76%. Univariate analysis revealed larger tumor size, higher tumor stage and grade, and higher preoperative GH levels all to be predictive of disease persistence (p < 0.01). Younger age and earlier surgery were marginally predictive of disease persistence (p = 0.05 and p = 0.08, respectively). No relationship was found between disease persistence and gender, prior irradiation, prior medical therapy, or symptom duration.

Of the original 173 patients demonstrating initial remission, 130 could be contacted for long-term follow up. Of these, 9 (7%) had suffered disease recurrence. Five of these patients underwent further therapy, 4 of whom are currently in remission. Median time to recurrence was 3.3 years. Technical recurrences, defined as a single elevated GH level unaccompanied by clinical symptoms, and followed by subsequent normal GH levels without further treatment, occurred in 11 cases (9%). In contrast to disease persistence, no variables were found to be predictive of disease recurrence. The total postoperative complication rate among the 254 patients was 13%, including 6% hyponatremia, 2% CSF leaks, 2% meningitis, and 2% hypopituitarism. There were no known cases of postoperative hemorrhage and no postoperative deaths linked to surgery. In contrast to the 2- to 5-fold increased mortality rate among untreated acromegalics, the mortality rate among patients with postsurgery GH levels of < 5 µg/L was equivalent to that predicted by age-adjusted mortality statistics. Aggressive therapy to reduce GH levels should therefore be instituted at the time of diagnosis.
The ability to immediately predict long-term outcome following transsphenoidal resection of prolactin-secreting pituitary adenomas is important in determining the vigilance with which residual or recurrent tumor is sought and in allaying the concerns of patients who want to know whether their tumor has been cured. Therefore, we retrospectively reviewed our experience with prolactinomas followed for at least 5 years after surgery in order to assess the prognostic value of serum prolactin levels drawn on the first postoperative day.

Between 1979 and 1991, 241 patients underwent transsphenoidal surgery for prolactinomas. Nineteen were lost to follow up, while the remaining 222 had fasting AM prolactin levels drawn on postoperative day (POD) 1 and random serum levels taken at 6 weeks, 12 weeks, and then every 6 months for a minimum of 5 years.

Of the 222 patients, 176 had normal serum prolactin levels (< 20 ng/ml in our lab) on the morning after surgery; 133 of these were < 10 ng/ml (Group 1) and 43 were between 10 and 20 ng/ml (Group 2). At 12 weeks postoperatively, 132 patients (99%) in Group 1 had normal prolactin levels, compared with only 32 (74%) of those in Group 2. By 5 years, 130 patients (98%) in Group 1 still had normal levels, compared with only 5 patients (12%) in Group 2. No patient who had a prolactin level < 3 ng/ml on POD 1 had an elevated prolactin at 5 years.

Prolactin levels < 10 ng/ml drawn the morning after surgery predict cure, whereas patients with normal levels between 10 and 20 ng/ml on POD 1 are still at high risk for treatment failure. Reported recurrences at 5 years thus most likely represent persistent tumor that was not originally removed, and patients with immediate prolactin levels > 10 ng/ml should undergo early postoperative imaging to determine if resectable residual tumor exists.
(48%), and ophthalmoplegia (7%), prompted diagnosis in most cases. Seven percent presented with apoplexy. Symptoms consistent with hypopituitarism were reported by a third of patients. Endocrine workup disclosed pituitary insufficiency in half of the patients, with deficits of ACTH (35%) and gonadotropins (33%) being the most frequent. Seven patients exhibited alpha-subunit hypersecretion. Nearly all tumors were macroadenomas (93%), with a median size of 2.3 cm. Immunostaining revealed that truly null tumors were less common than previously supposed (23.4%) with immunoreactivity for FSH (47%), LH (43%), and alpha-subunit (50.6%) higher than expected. Immunoreactivity to other hormones suggests that EIA include a significant subpopulation of clinically silent endocrine tumors (ACTH, 15%; TSH, 16%; GH, 7.9%; prolactin, 12.8%). Although total resection was reported in 80% of cases, only 29% were free of tumor on follow-up imaging. Only 16% developed symptomatic recurrences despite the high incidence of residual tumor. Patients undergoing postoperative radiotherapy (RR = 2.8, p < 0.01) and those receiving complete resections (RR = 5.5, p < 0.01) were less likely to develop recurrences. Surgery improved headaches (92%) and visual field deficits (90%) in most patients. Thyroid insufficiency (35%) appeared to increase following surgery while steroid dependency (28%) decreased. Surgery infrequently reversed hypogonadism.

Based on these data we conclude that: 1) postoperative radiotherapy likely reduces the recurrence rate; 2) surgeon impression of resection underestimates residual tumor; 3) a complete resection lowers the recurrence risk; 4) surgery alleviates symptoms of mass effect but is less successful for treating hypopituitarism; and 5) most EIA are immunoreactive to LH, FSH, or alpha-subunit with a significant portion being clinically silent endocrine tumors.

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**PAPER #730**
**MONDAY, 3:30-3:45 PM**

**Pallidotomy in Advanced Parkinson’s Disease: Two-Year Follow-Up**

Andrés M. Lozano*

Anthony E. Lang

Erwin B. Montgomery

Jan Duff

Ronald R. Tasker (Toronto, ON, Canada)

Discussant: Douglas Kondziolka

**KEY WORDS:** pallidotomy, Parkinson’s disease, outcome

Recent claims that posteroventromedial (PVM) pallidotomy has striking ameliorative effects in Parkinson’s disease come from studies involving small numbers of patients, short follow up, or nonvalidated rating methods. We studied 40 patients with detailed assessment of parkinsonism using validated rating scales after drug withdrawal (OFF) and under optimal drug response (ON). Scores 6 months after surgery were compared to baseline scores in the full cohort (n = 39), and stability of response was assessed in patients reaching 1 year (n = 27) and 2 years (n = 11) of follow up.

At 6 months total motor and daily activities (ADL) OFF scores had improved by 30% and contralateral limb scores by 40 to 55%. At the same time, ADL ON scores had improved by 30% and levodopa-induced dyskinesias by 83% in the contralateral limbs and 50% in the ipsilateral limbs. Improvement in the scores for total parkinsonism, contralateral bradykinesia, rigidity, and dyskinesias were sustained for the 2-year follow-up period. Benefit to ipsilateral dyskinesias was lost after 1 year and postural stability and gait improvements lasted only 3 to 6 months. PVM pallidotomy had a major impact on functional capacity with approximately 50% of patients dependent in measures of ADL in the OFF state before surgery becoming independent after surgery. Medication changes were not significant.

We conclude that PVM pallidotomy significantly reduces levodopa-induced dyskinesias and the disability experienced in OFF periods in later-stage Parkinson’s disease. The benefit from this procedure is largely sustained for
at least 2 years although certain features, such as improvements on the ipsilateral side and in axial symptoms, wane within the first year. ON period symptoms resistant to dopaminergic therapy do not benefit from surgery.

PAPER #731 MONDAY, 3:45-4:00 PM

Improved Outcome and Reduced Cost in Cervical Spinal Cord Injury Using Emergency MRI and Surgical Decompression

Nathan R. Selden
Nayna Patel
Susan Grube
Stephen Papadopoulos (Ann Arbor, MI)

Discussant: Volker K.H. Sonntag

KEY WORDS: spinal cord compression, cervical spine, magnetic resonance imaging

Ninety-one consecutive patients with acute traumatic cervical spinal cord injury (1991–1996) initially received conventional treatment with cervical traction, as needed, and steroids. Sixty-six patients then underwent emergency MRI to determine the presence of persistent spinal cord compression, followed, if indicated, by immediate operative decompression and stabilization (protocol group). Twenty-five patients were managed outside the protocol because of contraindication to MRI, need for other emergency surgical procedure, or surgeon preference (reference group). The protocol and reference groups had similar sex and age distributions, admitting Frankel scores, levels of neurological injury, and injury severity scores.

Protocol patients improved an average of 0.7 Frankel grades more than reference patients between admission and most recent follow up (p < 0.01). Fifty percent of protocol patients but only 24% of reference patients improved from their admitting Frankel grade. Eight protocol patients (12%) but no reference patients improved from complete motor quadriplegia (Frankel A or B) to independent ambulation (Frankel D or E). Protocol patients required significantly shorter ICU, general care, rehabilitation hospital stays and fewer days of ventilator support than reference patients (p < 0.01), representing an average savings per case of approximately $50,000. MR images obtained during protocol treatment also provided useful prognostic information regarding neurological outcome.

We conclude that immediate MRI to determine the need for emergency operative decompression and definitive stabilization significantly improves neurological outcome and reduces cost in the management of acute traumatic cervical spinal cord injury.

PAPER #732 MONDAY, 4:00-4:15 PM

Results of Linear Accelerator-Based Radiosurgery for Intracranial Meningiomas

Peter McL. Black
Rodolfo Hakim
Eben Alexander, III
Jay S. Loeffler
Dennis Shrieve (Boston, MA)

Discussant: Kevin Gibbons

KEY WORDS: radiosurgery, meningioma, linear accelerator

We report the outcome of the patients treated with LINAC radiosurgery for intracranial meningiomas at our institution. We reviewed 127 patients with 155 meningiomas treated with stereotactic radiosurgery (SRS) at the study institutions between October, 1988, and December, 1995.
There were 86 females and 41 males (median age 61.5 years, range 19.9–87.9 years). Median follow up was 31 months (range 1.2–79.8 months). The median tumor volume was 4.1 cc (range, 16–51.2 cc) and the median marginal dose was 15 Gy (range 9–20 Gy). The tumor locations were: 31 convexity, 39 parasagittal/falcine, 82 skull base, and 3 ventricular/pineal. There were 106 benign meningiomas, 26 atypical, 18 malignant, and 5 meningiomatosis. SRS was given to 48 lesions as initial treatment and to 107 lesions as adjunct therapy. Twenty patients had disease progression (16 marginal and 4 local) at a median time of 19.6 months (range 4.1–69.3 months); 6 patients (4.7%) had permanent complications attributable to SRS (median time months; range 4.3–18.0 months); 13 patients died of causes related to the meningioma (median 17.5 months; range 4.3–37.3 months). The 1-, 2-, 3-, 4-, and 5-year survival rates in benign meningiomas, excluding death from intercurrent disease, were 97.6%, 94.8%, 91%, and 91%, respectively.

Even though complications from SRS are more probable with larger tumors and with those near critical structures, it offers a safe and effective means of treating selected meningiomas.

PAPER #733  MONDAY, 4:15-4:30 PM
Anterior Cervical Surgery Without Plate Instrumentation in 245 Patients

Nancy Epstein (New Hyde Park, NY)
Discussant: Stephen Papadopoulos*

Key Words: cervical spine, instrumentation, plating

To examine the efficacy of anterior cervical surgery without plate instrumentation, 248 patients with anterior cervical discectomy (ADF) or corpectomy (ACF) and fusion performed by the author over a 7.5-year period were reviewed. Only three were plated. Of the other 245 patients, 179 had ADF averaging 1.6 levels, 56 had ACF averaging 1.6 vertebrae, while 10 had ADF/ACF averaging 1.1 levels. Fusion was assessed with flexion/extension x-ray, and/or 3D-CT studies, performed at 2 months and repeated every month for a minimum of 6 months, or longer if fusion had not occurred. Based on White and Panjabi’s definition of clinical instability, patients were placed into one of three categories: 1) fused; 2) clinically stable but with radiographic lucency; or 3) clinically unstable. The mean follow up was 52 months.

Fusion was demonstrated in 210 patients, clinical stability with radiographic lucidity in 25, and clinical instability in 10 patients. There was no relationship between the type and severity of the original operation and clinical instability. In 4 of these 10 patients, a secondary posterior wiring and fusion was performed within 4 to 22 months of the original surgery. The other 6 patients are being followed conservatively. Postoperative vertebral fractures led to graft extrusions/complications and reoperations in another 7 patients.

The frequencies of clinical instability (4.1%) and secondary surgery due to clinical instability (1.6%) or fractures/graft extrusions (2.9%) compare favorably with the frequencies of complications (up to 15%) and reoperations (up to 12.5%) reported in plated series.

PAPER #734  MONDAY, 4:30-4:45 PM
Current Patterns of Inflicted Head Injury in Children

Shervin Rahimdashti
Debra Decker
Ashfaq Razzaq
Alan Cohen (Cleveland, OH)

Discussant: Ann-Christine Duhaime

Key Words: head injury, child abuse, subdural hematoma
Although inflicted trauma is a major cause of serious head injury in young children, it has received little systematic examination. To define current patterns of child abuse associated with head trauma, we analyzed the clinical presentation, socioeconomic status, mechanism of injury, and outcome in patients carrying this diagnosis. We reviewed 353 consecutive cases of head trauma admitted to Rainbow Babies and Childrens Hospital between January, 1995, and July, 1997, and identified 30 children with deliberately inflicted injuries. There were 18 boys and 12 girls (median age 5 months). All but 3 children were less than 2 years old. Of the patients' families, 67% lived in the inner city, 75% were on welfare, and 20% had a history of drug or alcohol abuse.

In most cases, the mechanism of injury was unknown or the history was insufficient to explain the injury. Median Glasgow Coma score was 7. Retinal hemorrhages were present in 11 patients (37%). There were 7 skull fractures (23%). Seventeen patients (57%) had acute subdural hematomas, 6 (20%) had subarachnoid hemorrhage, and 4 (13%) had epidural hematomas. Most patients (24) had isolated head injuries. In those sustaining multiple trauma, associated injuries included human bite (2), rib fracture (2), facial fracture (1), liver contusion (1), and pancreatic contusion (1). No patient had a long bone fracture. Four patients died, 3 from acute subdural hematoma. Of the survivors, 9 were placed in foster care, 3 were left in the custody of other family members, and 14 were returned to a safe caregiver in the home.

We stratified the patients by age and identified 86 children under 2 years old admitted for head trauma. Of these, the head injury was inflicted in 27 (31%) and accidental in 59 (69%). Acute subdural hematoma occurred in 16 (59%) of the 27 children with inflicted trauma but only 5 (8%) of the 59 with accidental trauma. Four (80%) of the 5 accidental subdural hematomas were caused by motor-vehicle accidents.

Deliberately inflicted injury remains exceedingly common. In our series, inflicted injury accounted for almost a third of all hospital admissions for head trauma in children under 2 years old. Child abuse was strongly correlated with low socioeconomic status. Inflicted head injuries occurred most often as isolated wounds. No patient in this series had an extremity fracture, calling into question the routine use of skeletal surveys. Acute subdural hematomas occurring in children under the age of 2 years, in the absence of a motor-vehicle accident, are most likely related to abuse.

PAPER #735 MONDAY, 4:45-5:00 PM

Head Injury in Abused Children

Javad Hekmat-Panah (Chicago, IL)
Linda Callans
Pia Pannaraj (Philadelphia, PA)

Discussant: Thomas Luerssen

KEY WORDS: head injury, child abuse

We reviewed the clinical and social findings of abused children with head injury admitted to our hospital over the past 30 years. We selected the files of 190 children in whom head injury was severe enough to require hospitalization. There were 119 (63%) male and 71 (37%) female infants. The age distribution was 63% below 1 year, 36% below 6 months, and 23% below 3 months. There were 88% Blacks, 9% Caucasian, and 3% Hispanic. Of the parents, 26% were married and 72% were unmarried; 7% of mothers were alcohol abusers and 19% drug abusers.

There was a depressed level of consciousness in 79 (42%) of the children, respiratory depression in 61 (32%), seizures in 60 (32%), and vomiting in 43 (23%). Suppressed level of consciousness varied in its degree; 20 (10.5%) arrived comatose. The respiratory distress also varied in intensity; 50% had apnea. The alteration of the pupils was recorded in 172 patients, and funduscopic examination was recorded in 143. Thirty-two (19%) had dilatation of
pupils, bilateral in 25 (14.5%); 58 (41%) suffered retinal hemorrhage and 52% had associated injuries. Information about the fontanel was available in 149 patients; the fontanel was open in 101 (68%); in 44 (43%) it was bulging or tense. There were 78 (41%) skull fractures, 11 (6%) cerebral contusion, 10 (5%) SAH, 54 (29%) subdural hematoma, and 7 (3%) epidural hematoma; 9% required craniotomy or other operations. At discharge, 29 (15%) had physical, 8% mental, and 2% both impairments. There were 22 (12%) deaths.

Of all abused children, 67% are below the age of 9 years, while 80% of head injury victims are under 2 years old when the condition could mimic other diseases of infancy. This is especially true when a history of trauma is minimized or denied. This study reveals that the clinical symptoms are so distinct that a history of trauma is not necessary for diagnosis.

P APER #736 MONDAY, 5:00-5:15 PM

Failed Radiosurgery and the Role of Microsurgery for Acoustic Neuroma

L. Dade Lunsford (Pittsburgh, PA)*
Bruce E. Pollock (Rochester, MN)
Douglas Kondziolka
Raymond Sekula
Brian Subach (Pittsburgh, PA)

Discussant: Kalmon Post

KEY WORDS: acoustic neuroma, radiosurgery, microsurgery

The indications, operative findings, and outcomes of acoustic neuroma microsurgery are controversial when it is performed after a patient has undergone stereotactic radiosurgery. We reviewed our experience at two academic medical centers during a 10-year interval in which 523 patients underwent stereotactic gamma knife radiosurgery. Ten patients with unilateral tumors (2%, three men, seven women, age range 35–79) underwent delayed microsurgery 6–49 months (mean 29.9 months) after radiosurgery. Six patients had undergone microsurgery before radiosurgery. The mean marginal dose to the tumor margin was 16 Gy, and an average of 6.2 isocenters were used for each tumor. The indications for surgery were tumor growth in four patients (substantiated in one), increased symptoms in seven, persistent symptoms in two, and cystic enlargement in two. Microsurgery consisted of suboccipital removal in eight patients, translabyrinthine in one, and a combined approach in one. Gross-total removal was possible in seven patients. Tumor consistency was described as cystic in two patients, adherent to normal structures in five (three had undergone prior microsurgery), fibrous in two, and normal in five. Histopathological examination revealed a typical Schwann cell neoplasm in all cases; three tumors showed suggesting radiation effects. Postoperative complications included hydrocephalus in two patients, meningitis in two, CSF leak in two, new onset deafness in two, worsened facial nerve function in five, diplopia in one, and ninth and tenth cranial nerve palsies in two. At last follow up seven patients had House-Brackmann Grade 6 facial palsies, two had Grade 4, and one had Grade 3. All patients were deaf. Two patients remained bedridden.

The goal of radiosurgery is the prevention of further growth. Failed radiosurgery was rare at our two institutions, occurring in less than 2% of patients, and failure occurred before 5 years had elapsed since radiosurgery. Failed microsurgery was more common in patients in whom prior microsurgery had already failed, and for whom radiosurgery was performed as a microsurgical alternative. Clinical worsening appears to be common after microsurgery performed after radiosurgery, and patient outcomes are usually worse. No clear relationship could be detected between the use of radiosurgery and the subsequent ease or difficulty of delayed microsurgery. The indications for microsurgery after radiosurgery should reflect definitive, sustained tumor volume increase in association with progressive, new neurological symptoms. Because 1 to 2% of patients have slight tumor growth before subsequent growth arrest, the need for delayed microsurgical resection should be reviewed with the surgeon who performed radiosurgery.
Monday Afternoon

Scientific Session IV

2:45 PM – 5:15 PM     (Room 108A/B)

Moderators: Stewart Dunsker, MD (Cincinnati, OH)
Arthur L. Day, MD (Gainesville, FL)

PAPER #737 MONDAY, 2:45-3:00 PM
Hearing Preservation in 75 Acoustic Neuroma Surgeries
Catalino D. Dureza
Takanori Fukushima
Moises Arriaga (Pittsburgh, PA)

Discussant: Albert Rhoton, Jr.

Key Words: hearing preservation, acoustic neuroma, facial nerve function

In this series of patients selected for hearing preservation in acoustic neuroma surgery the emphasis was placed on a meticulous microsurgical skull base technique which was tailored to fit each patient. In the hands of a highly skilled microsurgical team with appropriate selection criteria, the majority of acoustic neuromas can be removed safely with excellent facial nerve function and as nearly successful rate of hearing preservation. This study reviews 75 consecutive hearing preservation acoustic neuroma surgeries in a total series of 360 acoustic neuromas performed over an 8-year period. Patients with speech reception threshold (SRT) of 50 dB and at least a 60% speech discrimination score (SDS) were preferred candidates for hearing preservation. We have attempted hearing preservation in patients with 50 dB SRT and 50% SDS if other factors were favorable.

Patients with largely intracanalicular tumors underwent a middle fossa approach, whereas tumors located predominantly in the cerebellopontine angle were excised via a retrosigmoid transtemporal approach. Postoperatively 80% of the patients in this series had some form of hearing preserved. Of the total number of patients undergoing surgery for hearing preservation, 69% had usable hearing based on the American Academy of Otolaryngology hearing classification. Of the 33 retrosigmoid cases, 73% had hearing preserved; 64% had usable hearing; and 9% measurable hearing. Of 42 middle fossa operations, 86% had hearing preserved; 74% had usable hearing; and 12% had measurable hearing. No significant complications were encountered. Excellent immediate postoperative facial nerve function was present in 90% of the cases. There were no cases of complete facial nerve paralysis. Details of our current operative techniques and sophisticated microinstrumentation will be discussed using illustrative cases.
Late Angiographic Follow Up of Surgically Treated Aneurysms: Recurrence, De Novo Formation, and the Fate of Residuals

Carlos David (Phoenix, AZ)
A. Giancarlo Vishteh
Michael Lemole
Michael Lawton (San Francisco, CA)
Shahram Partovi
Robert F. Spetzler (Phoenix, AZ)

Discussant: Michael Horowitz

KEY WORDS: aneurysm recurrence, angiography, residual aneurysm

The long-term angiographic follow up of surgically treated aneurysms is unknown. This study was undertaken to evaluate the long-term angiographic outcome of surgically treated aneurysms, specifically, to determine the incidence of recurrent aneurysms, the fate of residual necks, and the de novo formation of aneurysms. Ninety surgically treated aneurysm patients underwent late follow-up angiography. There were 70 females and 20 males with a mean age of 48.5 years (range 12 to 78 years) and a total of 155 aneurysms. One hundred forty-eight aneurysms were surgically treated. Late angiographic follow up was obtained at a mean of 4.4 ± 1.6 years (range 2.6–9.2 years). Total patient follow-up time was 393 years.

There were 2 (1.3%) recurrent aneurysms, yielding a recurrence rate of 0.50%/yr. Sixteen aneurysms had known residuals of which 3 (18.7%) enlarged in size and 13 (81%) remained unchanged. Two residuals (12.5%) ruptured yielding a yearly hemorrhage risk of 2.4%/yr. Eight de novo aneurysms were found in six patients for a yearly risk of 2.0%/yr. A previous history of multiple aneurysms was associated with de novo aneurysm formation (p = 0.031, Fisher’s exact test).

This study confirms the long-term efficacy of aneurysmal clip ligation. In addition, there is a small but significant risk of de novo aneurysm formation, particularly in multiple-aneurysm patients. Most residual aneurysmal rests appear to remain stable, although a subset may enlarge or rupture. These findings support the use of late angiographic follow-up evaluation in aneurysm patients.

Failed Microsurgery and the Role of Radiosurgery for Acoustic Neurinoma

Bruce E. Pollock (Rochester, MN)
L. Dade Lunsford
Douglas S. Kondziolka
Brent C. Clyde
John C. Flickinger (Pittsburgh, PA)

Discussant: Steven Giannotta

KEY WORDS: radiosurgery, microsurgery, acoustic neuroma, outcome

To assess the outcomes of stereotactic radiosurgery (SR) for acoustic neurinoma in patients who have failed prior microsurgical resection, we reviewed our experience in 72 consecutive patients referred during a 9-year interval. The 72 patients (74 tumors) had a minimum of 1 year follow up after SR using the gamma knife. Multiple isocenter dose planning was used in all patients (doses ranged from 12–20 Gy at the tumor margin, depending on tumor volume). All tumors were either recurrent after total resection (34%) or persistent after subtotal resection (66%). Twenty-eight patients (38%) had undergone multiple resections. Forty-one patients (55%) had significant impairment of facial nerve function (House-Brackman Grade III–VI) after their microsurgical procedure; 50% had trigeminal sensory loss, and 96% had unserviceable hearing (PTA > 50, SDS < 50%). Median follow up after radiosurgery was 40 months (range 12–97 months).
In-hospital stay varied from 6 to 24 hours after radiosurgery. Patients returned to their preradiosurgical functional status the next day. Tumor control after radiosurgery was achieved in 69 patients (93%). Six patients required additional resection despite radiosurgery (median of 32 months afterwards) and one patient had repeated radiosurgery for tumor progression outside the previously irradiated volume. Nine (20%) of 45 patients with Grade I to III facial function prior to radiosurgery developed increased facial weakness after radiosurgery. Ten patients (14%) developed new trigeminal symptoms.

The recurrence of progression rates after surgical resection of acoustic neuromas is thought to be as low as 1 to 3%. Nonetheless, we have performed radiosurgery on more than 100 patients whose tumors had progressed despite initial surgery. The majority of these patients had significant cranial nerve or other neurological deficits after microsurgery. Radiosurgery proved to be a safe and effective surgical alternative to additional microsurgery in patients who failed initial microsurgical removal. Cranial nerve function and functional status were preserved in the majority of patients. Radiosurgery should be performed for all patients with symptomatic regrowth, recurrence, or progression of previously operated acoustic neuromas.

PAPER #740 MONDAY, 3:30-3:45 PM
The Long-Term Prospective Natural History of Venous Malformations

Mark McLaughlin*
Douglas Kondziolka
Stephanie Lunsford
L. Dade Lunsford (Pittsburgh, PA)

Discussant: Daniel Barrow

Key Words: venous malformation, hemorrhage, conservative management

A 12-year prospective study was undertaken to determine the long-term natural history of venous malformations with respect to hemorrhage rate, morbidity, and mortality. From 1984 to 1996, 80 patients with venous malformations were referred to the University of Pittsburgh multidisciplinary vascular malformation group for evaluation. Observation was recommended for all patients. Follow-up clinical information was obtained from patients or their referring physicians through questionnaire or telephone conversation.

Of the 80 patients with venous malformations, 22 presented with neurological signs or symptoms related to the malformation (9 with headaches, 4 seizures, 3 sensory symptoms, 3 motor deficits, 2 trigeminal neuralgia, and 1 extrapyramidal disorder). Twenty-three patients presented with headaches not considered related to the malformation. Sixteen patients had sustained prior intracranial hemorrhage in the region of the venous malformation, 2 of whom suffered two hemorrhages. Two patients suffered rehemorrhages during a prospective follow up totaling 295 patient-years of observation. One of these patients remained asymptomatic while the second developed temporary worsening of facial paresthesias. We suspect that bleeding may have been due to an associated but not visualized cavernous malformation. This represented a hemorrhage rate of 0.68% per year, similar to that we have reported for cavernous malformations without prior symptomatic hemorrhage. No patient died as a result of the venous malformation.

Conservative management of venous malformations should remain the mainstay of therapy since intervention risks probably greatly exceed the low risk of morbidity related to lesion hemorrhage.
PAPER #741 MONDAY, 3:45-4:00 PM
Morbidity and Mortality Associated with Operative and Conservative Management of Unruptured Intracranial Aneurysms: The International Study of Unruptured Intracranial Aneurysms (ISUIA)

David G. Piepgras, MD
David Wiebers, MD (Rochester, MN)

Discussant: Roberto Heros

Key Words: morbidity, intracranial aneurysms

The International Study of Unruptured Intracranial Aneurysms is a NIH/NINDS-sponsored study to assess the risk of hemorrhage of unruptured intracranial aneurysms and to determine the morbidity and mortality from intracranial procedures to prevent future hemorrhage. A prospective cohort of 1176 patients were entered from 52 centers in the United States, Canada, and Europe from 1991 to 1995 for whom surgical or endovascular treatment of their intracranial aneurysm was intended. Overall 962 patients had intracranial aneurysms without a history of SAH (Group I) while 214 patients with multiple intracranial aneurysms had a prior SAH (Group II) and now were undergoing repair of an unruptured aneurysm. Of Group I patients 696 had clipping of the aneurysm, 119 had an endovascular procedure, 100 had multiple procedures, and 47 had some other intracranial procedure. For Group II the distribution was 186, 10, 9, and 9, respectively. Operative morbidity and mortality will be presented for both Group I and Group II patients including functional status at 1 year following the procedure. Factors related to selection for the procedures will be presented. Also variables related to morbidity and mortality will be discussed. These results will be presented in the context of the risk of hemorrhage without surgery among various patient groups.

PAPER #742 MONDAY, 4:00-4:15 PM
Ten-Year Experience With Slit Ventricle Syndrome in Children

Robert Sanford
Michael Muhlbauer
Stephanie Einhaus
Errol Thomas (Memphis, TN)

Discussant: Fred Epstein

Key Words: slit ventricle, shunt malfunction

Slit ventricle syndrome is a controversial clinical entity which is poorly characterized in the literature. The authors define slit ventricle syndrome as a transient shunt malfunction in a patient that appears to have a “functioning” shunt. Further criteria that must be satisfied are bilateral small or slit ventricles, no dilated CSF collections, and a paroxysmal episode that starts and stops abruptly. A parental description should match that of typical shunt malfunction that resolved spontaneously. Clinical examination during the episode demonstrates the child with signs and symptoms compatible with increased ICP and a shunt that refills slowly.

In our series, we have allowed children to undergo at least 3 such episodes ("3 strikes you're out") before instituting operative treatment. Almost exclusively surgical treatment would consist of placement of a siphon control device. We eliminated from the series shunted hydrocephalics who had episodic proximal malfunction, pseudotumor syndromes, unilateral slit ventricles with dilated or contralateral ventricles, trapped fourth ventricles, or nonshunted cystic structures such as tectal plate cysts. We encountered 32 children with this syndrome and treated them with siphon control devices with a 95% success rate defined as immediate cessation of clinical episodes and no evidence of shunt malfunction over the next year. The failures will be discussed in detail, along with the differential diagnosis.
Rats underwent 2 hours of occlusion of the left MCA. In the first study (I) the animals were kept (intra-ischemically) either normothermic (37°C; n = 8), hypothermic (33°C; n = 8), or hypothermic (30°C; n = 8). In Study 2 (II) the rats were randomized into 4 groups: 1) normothermia controls (37°C; n = 8), 2) hypothermia (33°C; n = 9) for 30 minutes at ischemic onset, 3) hypothermia for 1 hour (n = 8), and 4) hypothermia for 2 hours (n = 8). In Study 3 (III) 6 groups of rats underwent 1.5-hour MCA occlusion: these included hypothermic animals (33°C; n = 36) that survived for 3 days, 1 week, or 2 months, and a parallel set of normothermic controls (n = 36). After suture removal, the rats were returned to 37°C and reperfused for 24 hours (I), 72 hours (II), and up to 2 months (III).

The neurological score for hypothermic rats was significantly better than for controls (I and II) at both 24 and 72 hours with the exception of the 30-minute group (II). In I, histopathology revealed an 83% and 68% reduction in infarct size for the 33°C group (p < 0.01) and the 30°C (p < 0.025), respectively. In II, 2 hours of hypothermia reduced the hemispheric infarct area by 69%; 1 hour reduced injury by 84% (p < 0.05); and 30 minutes did not show protection. Two hours of hypothermia decreased neutrophil accumulation by 76%, 1 hour by 80%, and 30 minutes by 15%. Normalized for infarct size, 2 hours hypothermia decreased neutrophil accumulation by 52%, while both 1 hour and 30 minute were not effective. At 72 h, 2 hours of hypothermia decreased TUNEL staining by 97%. In III, mortality was significantly reduced by hypothermic treatment, and average postanesthesia recovery time showed hypothermic animals tended to recover at a faster rate (24 ± 6.7 minutes; p = 0.068) compared to controls (31.2 ± 11.9 minutes). We conclude that intra-ischemic mild hypothermia (33°C) must be maintained for 1 to 2 hours to obtain optimal neuroprotection against ischemic cell death due to necrosis and apoptosis.

Basal metabolism was suppressed by reducing rectal core temperatures to 86°F (30°C). Activation metabolism was suppressed with pentobarbital. Diphenylhydantoin and dexamethasone were used for membrane stabilization and further suppression of the inflammatory response. Hypokalemia, hypotension, hypocap-
nia, alkalemia, and reduced fluid requirements were physiological by-products of the protocol and were utilized for prophylaxis of vasogenic edema and intracranial hypertension during the period of cooling. Shivering was blocked with nondepolarizing muscle relaxants and phenothiazines. Requirements for mannitol were minimal with this protocol. Hypothermia was maintained for an average of 4 days following head injury and limited to the perioperative period with elective surgery. Patients were then warmed slowly at 1°F/hr. Good outcome was improved over conventional management of traumatic coma (GCS score < 8) and mortality was significantly reduced (p < 0.0001): 66% good outcome, 3% moderate disability, 9% poor outcome, and 22% mortality. No patients were vegetative.

Appropriate guidelines for the management of patients during prolonged metabolic suppression with hypothermia and pentobarbital were developed. These differ substantially from conventional, normothermic management. Adaptation to the altered physiological state induced by hypothermia at 30°C combined with a metabolic drug regimen, rather than attempts to correct parameters to normothermic values, was paramount in the successful utilization of these modalities.

PAPER #745  MONDAY, 4:45-5:00 PM
Application of Transcranial Doppler Ultrasonography for the Diagnosis of Brain Death

Moshe Hadani
Bella Bruk (Tel Hashomer, Israel)

Discussant: Donald Becker

KEY WORDS: brain death, ultrasound, circulatory arrest

The clinical diagnosis of brain death may be discouraged by the presence of pharmacological agents and legal considerations. Therefore an objective and a noninvasive method is essential to the early and accurate diagnosis of brain death. Transcranial Doppler ultrasonography (TCD) is a noninvasive technique that detects changes in cerebral circulation such as total circulatory arrest, which is pathognomonic for brain death. A number of TCD patterns such as systolic peaks without diastolic blood flow, oscillating patterns with retrograde flow in diastole, short systolic spikes, and absence of TCD signals have been previously reported as specific for intracranial circulatory arrest.

The TCD recordings of the extradural carotid artery, MCA, VA, and BA arteries were performed in 117 comatose patients (GCS score < 5). Sixty-seven patients met the clinical criteria of brain death. In 65 of them (97%) circulatory arrest was confirmed; TCD confirmation of total circulatory arrest was based on the presence of specific patterns in both the MCA and the VA and BA. In two patients a false negative result was evident by the presence of positive flow in diastole in more than two vessels. In the 50 patients who did not meet the clinical criteria for brain death, no TCD signs of total cerebral circulatory arrest were found. The specificity of TCD tests for confirmation of brain death was 100% and the sensitivity 97%. We conclude that TCD should be included as an additional test to the protocol for the assessment of brain death.
James Conway
Eric Oshiro
Rafael Tamargo (Baltimore, MD)

Discussant: H. Hunt Batjer

**KEY WORDS:** subarachnoid hemorrhage, ventricular blood, outcome

The admission GCS score has been shown to be predictive of outcome for patients with aneurysmal SAH. The admission CT scans for patients presenting with aneurysmal SAH were analyzed to identify CT features which complement the predictive value of the admission GCS score. The admission CT scans for 126 patients were retrospectively graded on ordinal scales for the presence and severity of 33 different features.

Using univariate contingency analysis, the CT features which correlated significantly with the discharge GOS score were: blood thickness (p = 0.04), the presence of a mass lesion (p = 0.04), the presence of ventricular blood (p < 0.0001) and the presence of blood in several different cisterns and fissures. The clinical variables of age (p = 0.04), the occurrence of vasospasm (p = 0.005), and the admission GCS score (p < 0.0001) significantly correlated with GOS score. Stepwise logistic regression with backward elimination was used to identify truly independent variables and included all CT features and clinical variables in the analysis. The admission GCS score and the presence of ventricular blood were the only variables significantly correlated with the GOS score in the logistic regression (p < 0.05 for retention of the variable in the model) with calculated odds ratio of 2.3 and 6.3, respectively. The presence of blood in the ventricles on the admission CT scan appears to be as strong a predictor of outcome as the admission GCS score. This is the first study which identifies a feature on the admission CT scan after aneurysmal SAH that is a strong predictor of a patient's discharge GOS status.
Monday Afternoon

AANS ANNUAL BUSINESS MEETING

5:15 PM – 6:15 PM    (Room 204 B/C)
Tuesday, April 28, 1998

BREAKFAST SEMINARS
6:45 AM – 9:30 AM

Breakfast—Grand Hall
6:45 AM – 7:30 AM

Seminars
7:30 AM – 9:30 AM

#201 POINT - COUNTERPOINT: BASILAR TIP ANEURYSMS COIL VERSUS CLIP
Room 203B

Moderator:
John Tew, MD (Cincinnati, OH)

Panelists:
Alex Berenstein, MD (New York, NY)
L. N. Hopkins, MD (Buffalo, NY)*

#202 CURRENT MANAGEMENT OF VASOSPASM
Room 107B

Moderator:
Neal Kassell, MD (Charlottesville, VA)

Panelists:
Marc R. Mayberg, MD (Seattle, WA)
Jay Max Findlay, MD (Edmonton, AB)
Michael Levy, MD (Los Angeles, CA)
H. Richard Winn, MD (Seattle, WA)
#203 NEW APPLICATIONS OF SPINAL CORD STIMULATION
Room 109B

Moderator:
Samuel Hassenbusch, MD (Houston, TX)*

Panelists:
Robert Levy, MD (Chicago, IL)*
Giancarlo Barolat, MD (Philadelphia, PA)*
Mario Meglio, MD (Rome, Italy)

#204 PEDIATRIC CRITICAL CARE
Room 110B

Moderator:
Thomas Luerssen, MD (Indiapolis, IN)

Panelists:
Michael Rosner, MD (Birmingham, AL)
Randall Chesnut, MD (Portland, OR)
Ann-Christine Duhaime, MD (Philadelphia, PA)
Harold L. Rekate, MD (Phoenix, AZ)

#205 MANAGEMENT OF PEDIATRIC HYDROCEPHALUS
Room 112B

Moderator:
Dale Swift, MD (Dallas, TX)

Panelists:
Stephen Haines, MD (Minneapolis, MN)*
Leslie Sutton, MD (Philadelphia, PA)
Maurice Choux, MD (Marseilles, France)
Veetai Li, MD (Buffalo, NY)

#206 ADVANCED TECHNIQUES FOR STEREOTACTIC RESECTION OF INTRACRANIAL LESIONS
Room 204C

Moderator:
Richard Bucholz, MD (Saint Louis, MO)*

Panelists:
Michael Sisti, MD (New York, NY)
David Roberts, MD (Lebanon, NH)*
Axel Perneczky, MD (Mainz, Germany)
Robert Maciunas, MD (Nashville, TN)
#207 ENDOSCOPIC SURGERY OF THE SPINE
Room 204B

Moderator:
Curtis Dickman, MD (Phoenix, AZ)*

Panelists:
Daniel Rosenthal, MD (Frankfurt, Germany)*
J. Patrick Johnson, MD (Los Angeles, CA)*
Stephen Ondra, MD (Ann Arbor, MI)
Charles Riedel, MD (New York, NY)

#208 CONTEMPORARY MANAGEMENT OF SYRINGOMYELIA
Room 102A/B

Moderator:
Richard Morawetz, MD (Birmingham, AL)

Panelists:
Thomas Milhorat, MD (Brooklyn, NY)
Barth Green, MD (Miami, FL)
Hiroshi Abe, MD (Sapporo, Hokkaido, Japan)
Richard Ellenbogen, MD (Washington, DC)

#209 MANAGEMENT OF THORACOLUMBAR FRACTURES
Room 202A

Moderator:
Regis Haid, Jr., MD (Atlanta, GA)*

Panelists:
Christopher Shaffrey, MD (Norfolk, VA)
Gerald Rodts, Jr., MD (Atlanta, GA)
David Cahill, MD (Tampa, FL)
Lee Ansell, MD (Houston, TX)

#210 CONTEMPORARY EVALUATION AND MANAGEMENT OF PRIMARY METASTATIC SPINAL COLUMN NEOPLASMS
Room 202B

Moderator:
Arnold Menezes, MD (Iowa City, IA)

Panelists:
Narayan Sundaresan, MD (New York, NY)*
Martin Camins, MD (New York, NY)
Ziya Gokaslan, MD (Houston, TX)
Richard Perrin, MD (Toronto, ON)
#211 Diagnosis and Management of Cervical Spine Instability
Room 203A

Moderator:
Stephen Papadopoulos, MD (Ann Arbor, MI)*

Panelists:
Vincent C. Traynelis, MD (Iowa City, IA)*
H. Louis Harkey III, MD (Jackson, MS)*
Joseph Cusick, MD (Milwaukee, WI)
Peter Klara, MD (Norfolk, VA)

#212 Contemporary Techniques of Carpal Tunnel Syndrome
Room 104A

Moderator:
Scott Shapiro, MD (Indianapolis, IN)

Panelists:
Mario Brock, MD (Berlin, Germany)
Susan Tindall, MD (Atlanta, GA)

#213 Correlative Microvascular Anatomy as a Guide to Better Surgery
Room 105B

Moderator:
Arthur L. Day, MD (Gainesville, FL)

Panelists:
Harry Van Loveren, MD (Cincinnati)
Evandro DeOliveira, MD (Sao Paulo, Brazil)
Gazi Yasargil, MD (Little Rock, AR)
Walter Grand, MD (Buffalo, NY)*

#214 Trauma Guidelines for the Practicing Neurosurgeon
Room 104B

Moderator:
Charles Tator, MD (Toronto, ON)

Panelists:
Jack Wilberger, MD (Pittsburgh, PA)
Beverly Walters, MD (Providence, RI)
Jam Ghajar, MD (New York, NY)
#215 SURGICAL APPROACHES TO THE ANTERIOR SKULL BASE
Room 109A

Moderator:
John Jane, MD (Charlottesville, VA)

Panelists:
Takanori Fukushima, MD (Pittsburgh, PA)
Franco DeMonte, MD (Houston, TX)
Donald Wright, MD (Washington, DC)
Anil Nanda, MD (Shreveport, LA)

#216 FUNCTIONAL PITUITARY TUMORS
Room 111B

Moderator:
Martin Weiss, MD (Los Angeles, CA)

Panelists:
William Chandler, MD (Ann Arbor, MI)
Ivan Ciric, MD (Evanston, IL)
Ian McCutcheon, MD (Houston, TX)
Douglas Kondziolka, MD (Pittsburgh, PA)*

#217 SURGICAL APPROACHES TO LATERAL SKULL BASE
Room 111A

Moderator:
Albert Rhoton, Jr., MD (Gainesville, FL)

Panelists:
Laligam Sekhar, MD (Washington, DC)
Madjid Samii, MD (Hannover, Germany)
J. Diaz Day, MD (Boston, MA)
Griffith Harsh IV, MD (Boston, MA)

#218 ADVANCED TECHNIQUES IN THE TREATMENT OF PITUITARY TUMORS
Room 112A

Moderator:
Jules Hardy, MD (Montréal, PQ)*

Panelists:
Alex Landolt, MD (Zurich, Switzerland)
Kalmon Post, MD (New York, NY)
L. Dade Lunsford, MD (Pittsburgh, PA)
Zvi Ram, MD (Tel-Hashomer, Israel)
#219  EXPANDING YOUR PRACTICE THROUGH NETWORKING AND OTHER INNOVATIVE TECHNIQUES
Room 110A

Moderator:
Joseph Hahn, MD (Cleveland, OH)

Panelists:
Craig Van Der Veer, MD (Charlotte, NC)
Paul Nelson, MD (Indianapolis, IN)
Julian Bailes, MD (Maitland, FL)
Edward Downing, MD (Savannah, GA)

#220  MOUNTAINEERING AND HIGH ALTITUDE MEDICINE FOR THE NEUROLOGICAL SURGEONS
Room 105A

Moderator:
Richard Lehman, MD (New Brunswick, NJ)

Panelists:
Richard Wohns, MD (Tacoma, WA)
W. Ben Blackett, MD (Tacoma, WA)
Rick Leone, MD (Tacoma, WA)
John Krasney, MD (Tacoma, WA)
Neil A. Martin, MD (Los Angeles, CA)

#221  CONSULTANTS CORNER: SPINE
Room 201C

Moderator:
Stewart Dunsker, MD (Cincinnati, OH)

Panelists:
Edward Benzel, MD (Albuquerque, NM)*
Richard G. Fessler, MD (Gainesville, FL)*
Volker K.H. Sonntag, MD (Phoenix, AZ)*
Richard Balderston, MD (Philadelphia, PA)

#250  3-D VIDEO SEMINAR—SURGICAL TREATMENT OF SPINAL ARTERIOVENOUS MALFORMATIONS
Room 204A

Moderator:
Ken Smith, MD (Saint Louis, MO)

Demonstrating Surgeon:
Robert Spetzler, MD (Phoenix, AZ)
Tuesday Morning

POSTER PROGRAM

9:00 AM – 4:30 PM

Exhibit Hall A/B/C

479 posters have been chosen for presentation. All will be on display Monday through Wednesday. Presentations will be given between 1:45 PM and 2:45 PM on Wednesday, April 29, 1998, for uninterrupted viewing.

(See Poster Program for complete listing of papers.)
Tuesday Morning

PLENARY SESSION II

9:45 AM – 11:40 AM (Ballroom A/B)

Moderators: Edward R. Laws, Jr., MD (Charlottesville, VA)
L. N. Hopkins, MD (Buffalo, NY)

PAPER #747 TUESDAY, 9:45-10:00 AM
Long-Term Outcomes After Acoustic Tumor Radiosurgery
Douglas Kondziolka*
L. Dade Lunsford
John Flickinger
Ann Maitz
David Bissonette (Pittsburgh, PA)

Discussant: Madjid Samii

KEY WORDS: radiosurgery, acoustic tumor

Little information exists regarding the long-term outcomes after radiosurgery for benign tumors. To evaluate results after radiosurgery for acoustic tumors, we studied all patients managed at a single center from 1987 to 1992 using serial imaging studies, physician-based clinical assessments, audio- grams, and a patient questionnaire. Our hypothesis was that stereotactic radiosurgery would provide good clinical outcome (prevention of tumor growth and maintenance of brain function) over a long-term interval. We also sought to identify patient expectations, levels of satisfaction, and complication rates.

One hundred sixty-two patients had radiosurgery (median age 65 years). Radiosurgery was performed with CT-based imaging (1987–1991) and MRI (1992). The mean tumor margin dose was 16 Gy (range 12–20 Gy). The mean number of isocenters was 3 (range 1–11). The mean transverse tumor length (pons-petrous + canal) was 22 mm (range 8–39 mm). Prior surgery was performed in 42 cases (26%): 13 total and 29 subtotal resections. Normal facial function was present in 123 cases (76%) and useful hearing (GR I + II) in 32 (20%).

For the entire series, the clinical tumor control rate (no resection required) was 98% (97% for patients with at least 8 years of follow up). One hundred tumors were smaller (62%), 53 remained unchanged in size (33%), and nine were larger (6%). Resection was performed in 4 patients (2.4%). Normal facial nerve function was preserved in 77% of patients, normal trigeminal function in 72%, and no change in hearing (GR grade) in 47%. No deficits occurred after 28 months of radiosurgery. Ten patients died of unrelated causes during follow up. A patient survey was returned by 115 patients (71%). Prior surgery had been performed in 30 patients (26%). Fifty-four patients (47%) were employed at the time of radiosurgery and 37 remained so (69%). Older patients retired. Patient activity levels remained unchanged in 68%, increased in 8%, and decreased in 24%. Gamma knife radiosurgery met patient expectations in 106 cases (92%), 97% in the prior-surgery group and 91% in the no-resection group. Complications were described by 36 patients (31%), 55% of which resolved. These included balance problems (n = 7), facial twitching (n = 6), facial weakness (n = 4), tinnitus (n = 3), hydrocephalus (n = 3), numbness or headache (n = 3),...
= 2), and one each of face pain, watery eye, low blood pressure, tumor bleed, dizziness, dry eye, and “stroke.” Radiosurgery was believed “successful” for 30 of 30 patients who had undergone prior surgery and 81 (96%) of those without prior resection. When asked if they would recommend this treatment for a friend or family member, 109 (95%) said yes, two said no, and four did not know.

In a 5- to 10-year follow up, 97% of patients believed that radiosurgery was a good treatment for their acoustic tumor. Overall, 98% of patients required no further tumor surgery. Morbidity in this early series of CT-based planning was usually transitory and relatively mild. Radiosurgery provides long-term tumor control and is associated with high rates of neurological function preservation and patient satisfaction.

PAPER #748 TUESDAY, 10:00-10:15 AM
Increased Oxygen Extraction Fraction in Symptomatic Patients With Complete Carotid Artery Occlusion Predicts a Subsequent Increased Incidence of Ipsilateral Ischemic Stroke

Robert Grubb
William Powers
Collin Derdeyn
Kent Yundt
David Carpenter
Thomas O. Videen
Susanne M. Fritsch (St. Louis, MO)

Discussant: Marc Mayberg

KEY WORDS: oxygen extraction fraction, stroke prediction, carotid artery occlusion

The hypothesis that increased cerebral oxygen extraction fraction (OEF) in the cerebral hemisphere distal to complete ICA occlusion is an independent predictor of subsequent risk of ipsilateral ischemic stroke was tested in medically treated patients. A total of 117 patients with atherosclerotic carotid artery occlusion were blindly and prospectively studied by clinical evaluation, laboratory testing, and positron-emission tomography (PET) measurements of OEF. Of these, 81 patients had ischemic symptoms ipsilateral to the occluded carotid artery and 36 patients were asymptomatic. Multiple other baseline risk factors were assessed. The patients were followed for an average of 30.6 months (5–62 months). The risk of ipsilateral ischemic stroke in patients with and without increased OEF was compared by Kaplan-Meier survival curves.

Of the 117 patients, 44 had increased OEF in the cerebral hemisphere distal to the occluded carotid artery. Of 13 ipsilateral ischemic strokes, 11 occurred in patients with increased OEF (p = 0.0001). Previous ipsilateral ischemic symptoms occurred in 39/44 high-OEF patients, but in only 42/81 normal OEF patients. Restricting the analysis to symptomatic patients only, there was an approximate sixfold increase in risk for ipsilateral stroke in the patients with increased OEF (p = 0.004). Baseline assessment of other risk factors did not identify this high-risk group.

The PET measurement of high OEF in symptomatic patients with carotid artery occlusion is a major risk factor for stroke. These data suggest that, as opposed to stenosis of the carotid artery, hemodynamic failure is the major mechanism for stroke in symptomatic patients with carotid occlusion, and surgery to improve cerebrovascular hemodynamics may be the treatment of choice in these patients.

Tuesday, 10:15–10:17 AM
Van Wagenen Fellow Presentation—Kamal Thapar, MD (Toronto, ON, Canada)
Physiological Characterization of Malignant Oligodendrogliomas Responding to PCV Chemotherapy

Peter Warnke
Ansgar Berlis
Christoph Ostertag (Freiburg, Germany)

Discussant: Mark Rosenblum

KEY WORDS: oligodendroglioma, chemotherapy, tumor physiology

In order to evaluate the efficacy of procarbazine, CCNU, and vincristine (PCV) chemotherapy in malignant oligodendrogliomas/oligoastrocytomas Grade III, we prospectively studied the tumor physiology in malignant oligodendroglioma patients before and after PCV chemotherapy. Twenty-seven patients with histologically verified malignant oligodendrogliomas were quantitatively studied as to their regional glucose utilization rate using FDG-SPECT, their sodium/potassium ATPase activity using 201 thallium SPECT, and their regional, tumoral, and normal brain blood flow using stable xenon-CT. Patients were treated with classical PCV chemotherapy as published by Cairncross and Hochberg. Four PCV chemotherapy cycles were given to every patient. After each cycle a clinical neurological examination, MRI, and repeated metabolic and physiological studies using SPECT and stable xenon-CT were performed.

Oligodendrogliomas turned out to be a unique entity within the glioma group as their mean pretherapy blood flow was 67.9 ± 13.2 ml/100 g/min, which was significantly higher than blood flow values measured in anaplastic astrocytomas and glioblastomas (p < 0.01). Also all oligodendrogliomas showed a hypermetabolic behavior, with ratios of tumor to contralateral brain of 1.84 ± 0.31; this distinguished them significantly from other malignant gliomas, which were also measured prospectively (p < 0.01). After the first cycle of PCV chemotherapy, 24 of the 27 patients showed an objective response on MRI and the mean glucose utilization dropped from 1.84 to 1.03 µmol/100 g/min, which was a highly significant reduction (p < 0.001). Also the thallium uptake dropped from 2.71 to 0.97 which again was highly significant (p < 0.001). Within this group of 24 patients, 7 showed a complete response after one cycle of PCV chemotherapy and 17 showed a partial response with a tumor volume reduction of more than 50%. After 4 cycles of PCV chemotherapy, 25 of the 27 patients showed either a partial or complete response with the minimum volume reduction of tumor mass of more than 50% and a sustained change in SPECT and xenon-CT images.

Pharmacokinetically, the unique response towards treatment with lipophilic compounds (CCNU, procarbazine) can be explained based on the tumor physiology with excessively high blood flow values within the tumors surpassing those of normal cortex. Also oligodendrogliomas form a physiological and metabolic subentity of malignant gliomas with excessively high glucose utilization ratios and 201 thallium uptakes, which make them easily and significantly distinguishable from other malignant gliomas.
Correlation of Pallidal Lesion Location and Outcome in Parkinson’s Disease: Dissociation Between Motor Signs and Drug-Induced Dyskinesias

Robert E. Gross
Wendy J. Lombardi
Jean A. Saint-Cyr
Anthony E. Lang
Andrés M. Lozano (Toronto, ON, Canada)

Discussant: Thomas Freeman

**KEY WORDS:** pallidotomy, Parkinson’s disease, dyskinesia

Within the globus pallidus, sensorimotor circuits are located posterolaterally, whereas limbic and associative circuits are more anteriorly situated. We therefore hypothesized that posterolateral pallidal lesions would be associated with improved clinical outcomes compared to more anteromedial lesions in patients undergoing pallidotomy for Parkinson’s disease. To test this hypothesis, we analyzed the relationship of lesion location to clinical outcome in a prospective study of the effects of pallidotomy on Parkinson’s disease.

Twenty-four patients who underwent microelectrode-guided pallidotomy were assessed preoperatively and up to 1 year postoperatively using the Unified Parkinson’s Disease Rating Scale (UPDRS) and a dyskinesia rating scale. The stereotactic location of the lesions was determined by two techniques: 1) multiplanar reformatting of 3D-SPGR MR images ($n = 24$), and 2) analysis of MR images acquired with an external stereotactic head frame attached ($n = 13$). Statistical analysis of the relationship of lesion position to clinical outcome measures was performed using multiple linear regression.

**Tuesday, 10:47–10:50 AM**

**Humanitarian Award Presentation—**
Howard L. Finney, MD

*(To be introduced by William Shucart, MD)*

**PAPER #751 TUESDAY, 10:50-11:05 AM**

The Shunt Design Trial: Variation in Surgical Experience Did Not Influence Shunt Survival

John Kestle*
James Drake (Vancouver, BC, Canada)

Discussant: Marion Walker

**KEY WORDS:** hydrocephalus, shunt failure, surgical experience

An international multicenter randomized trial comparing standard pressure-differential valves, Orbis Sigma valves, and PS Medical Delta valves for children with newly diagnosed hydrocephalus failed to show a difference in the time to first shunt failure (power 80%). Surgeons’ prior experience with the three valves varied. This analysis was performed to assess whether lack of surgical experience with any of the valves could explain the overall negative result. We compared shunt survival at high- and low-volume centers. We also compared the 1-year shunt survival rates (95% CI) for patients entered in the first quarter and the last quarter of the 25-month accrual period (for all patients and for each shunt).

Survival curves for high- and low-volume centers were found to be similar. Of the 344 randomized patients, 68 were accrued in the first quarter and 91 in the last quarter. The 1-year shunt survival for all patients entered in the
first quarter was 72 ± 11% compared to 64 ± 10% for patients entered in the last quarter. The shunt-specific results were 66 ± 20% compared to 54 ± 20% for Delta valve patients, 75 ± 20% compared to 70% ± 17% for standard valve patients, and 76 ± 18% compared to 66 ± 16% for Orbis Sigma patients.

Shunt survival did not improve as surgeons accumulated experience over the course of the study. Although participating surgeons had varying levels of experience with the different shunts at the start of the trial, this does not appear to explain the overall negative trial result.
Tuesday Morning

SPECIAL LECTURE II
THE RICHARD C. SCHNEIDER LECTURE

11:23 AM – 12:00 NOON (Ballroom A/B)

VASCULAR LESIONS OF THE SPINAL CORD:
NEW CONCEPTS AND TREATMENTS

Robert Spetzler, MD (Phoenix, AZ)
(To be introduced by L.N. Hopkins, MD)
Tuesday Afternoon

CUSHING MEDAL PRESENTATION

12:00 NOON – 12:05 PM  (Ballroom A/B)

Albert L. Rhoton, Jr., MD (Gainesville, FL)
(To be introduced by Edward R. Laws, Jr., MD)

THE CUSHING ORATION

12:05 PM – 1:00 PM  (Ballroom A/B)

WHAT FLY GENES CAN TELL US ABOUT HOW HUMAN EMBRYOS DEVELOP

Eric Wieschaus, PhD (Princeton, NJ)
(To be introduced by Edward R. Laws, Jr., MD)
Tuesday Afternoon

SECTION SESSIONS

2:45 PM – 5:30 PM

AANS/CNS Section on
Cerebrovascular Neurosurgery
(Room 114)

AANS Section on History of Neurological Surgery
(Room 108A/B)

AANS/CNS Section on
Pediatric Neurological Surgery
(Room 103A/B/C)

AANS/CNS Section on
Stereotactic and Functional Neurosurgery
(Room 113A/B/C)
Tuesday Afternoon

SECTION SESSION

AANS/CNS Section on
Cerebrovascular Neurosurgery

2:45 PM – 5:30 PM     (Room 114)

DONAGHY LECTURE

AANS/CNS Section on
Cerebrovascular Neurosurgery

2:45 PM – 3:15 PM     (Room 114)

EVOLUTION IN THE UNDERSTANDING AND
MANAGEMENT OF CAVERNOUS MALFORMATIONS

Robert Ojemann, MD (Boston, MA)
(To be introduced by Joshua B. Bederson, MD)
Tuesday Afternoon

SCIENTIFIC SESSION

AANS/CNS Section on
Cerebrovascular Neurosurgery

3:15 PM – 4:45 PM     (Room 114)

Moderators: Christopher Loftus, MD (Oklahoma City, OK)
Joshua B. Bederson, MD (New York, NY)

PAPER #801  TUESDAY, 3:15-3:30 PM
Surgical Approaches to Brainstem Cavernous Malformations

Randall Porter
Paul Detwiler
Michael Lawton
Patrick Derksen
Jonathan Baskin
Joseph M. Zabramski
Robert F. Spetzler (Phoenix, AZ)

KEY WORDS: cavernous malformation, brainstem, hemorrhage, outcome

Brainstem cavernous malformations are rare intracranial vascular lesions. They account for 9 to 35% of all cavernous malformations. Their natural history is poorly defined and surgical series heretofore have been relatively small. By reviewing our clinical experience with brainstem cavernous malformations, we determined the hemorrhage rate and factors that influence outcome. From this analysis, we determined the neurological morbidity and mortality resulting from the treatment of these lesions.

From 1985 to 1997, we followed 101 patients with brainstem cavernous malformations. There were 37 males and 64 females, with a mean age of 37 years. The lesions were located throughout the brainstem: the pons (38), pontomesencephalic area (31), medulla (15), midbrain (16), pontomedullary junction (11), midbrain-pons-medulla (2), pons-midbrain-thalamus-third ventricle (2), and midbrain-thalamus (2). The retrospective annual hemorrhage rate was at least 5% per year. Standard skull base approaches were used to resect 87 of 101 lesions. All 87 surgical patients had associated venous malformations. Of the 87 surgically treated patients, 72 (82%) were the same or better after surgical intervention and 12 (14%) were worse; 3 patients (3%) died. The average postoperative Glasgow Outcome Scale score of surgical patients was 4.5, with an average follow-up period of 34 months. Complications occurred in 21% of the cases.

Using the two-point method, frameless stereotactic guidance, and skull-based approaches, brainstem cavernous malformations can be resected safely to prevent rehemorrhage with acceptable morbidity and mortality rates.
We reviewed the clinical and radiographic features of 51 consecutively treated pediatric patients (pts) (< 19 years) with high flow-AVMs treated between 1988 and 1996. The mean age at presentation was 10.3 years (range 2 months–18 years). Most pts presented with intracranial hemorrhage (28 of 50, 56%). The remainder presented with progressive neurological deficit (10 or 20%), seizures (5 or 10%) or headache (6 or 12%), whereas 1 pt (2%) was asymptomatic. The majority were in Spetzler-Martin Grades III to VI (59%). Mean time from presentation to treatment was 12 months (range 0–84 months). Therapy included microsurgery, endovascular embolization, and/or stereotactic radiosurgery. Nine pts (18%) were treated with all 3 modalities, 21 (41%) with 2, and 21 (41%) with one modality. Higher-grade AVMs (III–V) underwent multimodality therapy more frequently (67% vs 54%) and had more stages of treatment (3.3 vs 1.8) when compared to lower-grade lesions. The mean duration of therapy was 15 months (range 0–113 months) and, for 20 pts (39%), treatment remains ongoing.

Radiological follow-up information was available in 43 of 51 pts. Of these, 18 pts (42%) demonstrated complete angiographic obliteration whereas 21 (49%) showed significant (> 10%) reduction in AVM volume. Clinical follow up was available for 47 pts (mean 26 months, range 1–132 months). Forty-three pts (91%) had good to excellent outcomes, 1 pt (2%) had a poor outcome, and 3 (6%) died. In the pediatric population AVMs can be particularly challenging to treat and onset of therapy is often delayed. Despite this, these results demonstrate that good clinical and radiographic results can be achieved with a flexible multimodality approach.
perfusion of fixative. The basilar arteries were prepared histologically and their cross-sectional areas measured. The Fisher LSD test was used for statistical analysis.

Mean basilar artery (BA) cross-sectional area was constricted from 0.324 mm² (100%) in the control group to 0.130 mm² (40.1%), 0.133 mm² (41.0%), and 0.123 mm² (37.9%) in the SAH alone group, vehicle at 24/36 group, and vehicle BID group, respectively. All groups treated with TBC 11251 demonstrated an increase in BA diameter relative to the vehicle groups. The 5 mg/kg TBC BID group had a mean BA area of 0.217 mm² (67.0%) and the 10 mg/kg TBC BID group had a mean BA area of 0.240 mm² (74.1%); both groups statistically improved from the vehicle group (p < 0.05). There were no side effects noted and no differences in the mean arterial pressures between drug and vehicle groups.

These findings demonstrate that systemic administration of the endothelin-A receptor antagonist TBC 11251 significantly attenuates cerebral vasospasm after SAH and provides additional support for the etiological role of endothelin-1 in vasospasm.

PAPER #804  TUESDAY, 4:00-4:15 PM
Endovascular Thrombolysis Using Mechanical Snare Fragmentation and Infusion of Fibrinolytic Agents for Symptomatic and Progressive Combined Dural Sinus and Deep Cerebral Vein Thrombosis

Matthew F. Philips
Robert Hurst
Linda Bagley
Eric Raps
Grant Sinson (Philadelphia, PA)

Key Words: thrombosis, endovascular surgery, urokinase, clot fragmentation

The ability to treat potentially catastrophic intracranial dural and deep cerebral venous thrombosis has been facilitated by improvements in endovascular surgery and microcatheterization. Six patients (aged 14–73 years) presented with progressive symptoms from thrombotic intracranial venous occlusion. Five presented with neurological deficits, while 1 patient had a progressive and intractable headache. Four patients had known risk factors for venous thrombosis (inflammatory bowel disease (2), anti-cardiolipin antibody (1), cancer (1), while 2 had no known risks. Four had combined dural and deep thrombosis, while clot formation was limited to the dural venous sinuses in 2 patients. The patients underwent diagnostic cerebral arteriograms followed by transvenous catheterization and selective sinus and deep venous microcatheterization. Urokinase, 200,000 to 1,000,000 IU, delivered by pulse-spray technique at the cephalad aspect of the thrombus in conjunction with clot fragmentation using a mechanical wire snare resulted in sinus patency in all of the cases. Radiological investigation at 24 hours reconfirmed sinus/vein patency.

All patients' symptoms and neurological deficits improved, and no procedural complications ensued. Follow up ranged from 5 to 28 months, and all 6 patients remain free of any significant venous reocclusion. Factors, including patients' age, pre-existing medical condition, symptom duration, location of thrombus, and venous structures involved had no statistical bearing on outcome. Patients with both dural and deep cerebral venous thrombosis often have a variable clinical course and an unpredictable neurological outcome. With improvements in interventional techniques, aggressive endovascular therapy is warranted in asymptomatic and symptomatic patients early in the disease course, prior to morbidity and potentially fatal neurological deterioration.
Mechanisms of Radiation-Induced Endothelial Injury

Marc Mayberg
Keisuke Onoda
Susan London
Mark Johnson
Corinne Gajdusek (Seattle, WA)

Key Words: radiosurgery, apoptosis, endothelial injury

Stereotactic radiosurgery is a common treatment for cerebral AVMs, yet little is known regarding the mechanisms by which radiation causes vascular obliteration. Confluent cultures of rat aortic endothelium were irradiated with single-fraction doses of gamma radiation (125–1500 cGy) and assayed at 0 to 72 hours.

Dose-dependent double-strand DNA breaks were apparent immediately after radiation and were rapidly repaired to near baseline levels within 2 hours. Apoptosis, demonstrated by TUNEL immunohistochemistry and DNA ladder formation, occurred in a dose- and time-dependent manner, with maximal apoptosis occurring at 16 to 24 hours after irradiation. By rtPCR, expression of gadd45 was maximally increased at 3 hours after irradiation. By Western analysis and immunohistochemistry, endothelial p53 expression was increased in a dose- and time-dependent manner after radiation, with maximum expression at 12 to 16 hours. Both Bax and p21 showed dose- and time-dependent increased expression which were maximal at 3 to 6 hours after irradiation. Using a PARP cleavage assay, interleukin b-1 converting enzyme (ICE) was activated in a dose- and time-dependent manner, with maximal cleavage at 12 hours. The ICE-inhibitor ZVAD-FMK (40 mM) prevented radiation-induced apoptosis.

In vascular endothelium, double-strand DNA breaks induced by ionizing radiation may cause expression of specific genes (p53, Bax, and gadd45) that modulate ICE activation and subsequent apoptosis. Similarly, activation of genes causing G1 cell cycle delay (gadd45 and p21) may inhibit normal endothelial regeneration. We hypothesize that these mechanisms contribute to chronic endothelial injury after radiation, which subsequently leads to intimal hyperplasia and thrombosis in both normal vessels and vascular malformations.

Transfemoral Cerebral Angiography in the Rat

D. Roxanne Todor
John Perl
Martin Porras-Jimenez
Gerry Bruno
Douglas Chyatte (Cleveland, OH)

Key Words: transfemoral cerebral angiography, cerebral vasculature, rat study

The laboratory rat is widely used in neurosurgical research and remains a useful model in the evaluation of cerebrovascular disease. Rodent models of stroke, cerebral vasospasm, and intracranial aneurysms are well represented in the literature and may require evaluation of the cerebral vasculature in vivo. Although the standard technique of direct carotid puncture produces adequate studies, it does not always permit a complete evaluation of the circle of Willis. Additionally, it does not allow the selective catheterization of either cerebral or vertebral arteries. As part of an ongoing study requiring serial cerebral angiograms, we have perfected the technique of transfemoral cerebral angiography in the rat.

Sixty female Sprague-Dawley rats weighing 250 to 275 g were studied. The animals were anesthetized and the femoral artery was exposed using
standard microsurgical technique. A microcatheter (Tracker 10) and guidewire (Dasher 0.01) were inserted into a small arteriotomy and advanced into the aorta. Under fluoroscopic guidance, the catheter was advanced into the desired vessel and digital subtraction angiography was performed using 0.8 cc of nonionic contrast per view.

In 56/60 animals we were able to completely visualize the circle of Willis at the first angiogram. We also had excellent visualization of extracranial carotid and vertebral arteries via aortic arch injections. This technique allows the visualization of aneurysms under 1 mm in size. Early complications such as excessive backbleeding (2 animals), embolism (5 animals), and transient neurological deficit (1 animal) led to 7 deaths.

We have refined the transfemoral cerebral angiogram in the rat and present it as an alternative technique to visualizing the cerebral vasculature in small animals.

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**SPECIAL SYMPOSIUM**

AANS/CNS Section on Cerebrovascular Surgery

4:45 PM – 5:30 PM (Room 114)

COMPLICATIONS OF INTRACRANIAL ANEURYSM TREATMENT

**Moderator:** Joshua B. Bederson, MD (New York, NY)

**Panelists:**
- David Piepgras, MD (Rochester, MN)
- Fernando Vinuela, MD (Los Angeles, CA)
- Robert Ojemann, MD (Boston, MA)
Notes
SECTION SESSION

AANS/CNS Section on History of Neurological Surgery

2:45 PM – 5:30 PM (Room 108A/B)

SPECIAL LECTURE

AANS/CNS Section on History of Neurological Surgery

2:45 PM – 3:30 PM (Room 108A/B)

THE SEPARATION OF CRANIOPAGUS TWINS: AN HISTORICAL PERSPECTIVE

Theodore Roberts, MD (Seattle, WA)
The Conquest of the Skull Base

Ghassan K. Bejjani
Brian J. Sullivan
Donald C. Wright
Laligam N. Sekhar (Washington, DC)

KEY WORDS: skull base, surgical approach, neurosurgical history

Although cranial base surgery has become more popular over the past 2 decades, many of the surgical techniques used date back to the early days of neurosurgery. Technically, the skull base was conquered in small steps by the addition of techniques that progressively removed the cranial base piece-meal: from the orbital rim and ethmoidal sinuses anteriorly, to the zygoma and the middle fossa floor laterally, and to the temporal bone and occipital condyle posteriorly. The orbital osteotomy was originally described by MacArthur (1912) and Frazier (1913) to approach pituitary tumors. The transbasal approach was elaborated by Derome later, in 1972. The use of zygomatic osteotomy in neurosurgery was first described by Rose in 1890 for excision of the trigeminal ganglion, and in 1895 Krogius resected the zygoma to access a middle fossa tumor. Charles Ballance was one of the early pioneers of skull base surgery. He performed radical mastoidectomies with ligation of the jugular vein and facial nerve grafting (1901) and was the first to report successful resection of an acoustic neuroma. Panse suggested the translabyrinthine approach for tumor resection in 1904 and, the same year, Elsberg combined it with the sub-occipital approach. Further refinements to the transpetrous approach were added throughout the years: supralabyrinthine (Eagleton, 1931), infralabyrinthine (Dearmin, 1937), precochlear (Kopetsky and Almour, 1931), transcochlear (Gacek, 1975), partial labyrinthine (Hakuba, 1985), and middle fossa (Parry, 1904, and House, 1963) approaches.

Condylar resection (Seeger, 1978) was another addition to skull base techniques leading the way to the extreme-lateral transcondylar approach. Transfacial approaches were also a useful addition to the skull base surgeon’s repertoire, like the transsphenoidal (Schloffer, 1907) and transoral (Kanavel, 1919) approaches. All these techniques were combined progressively into more complex approaches like the infratemporal fossa approach (Fisch, 1977) and the extended transbasal approach (Cophignon, 1983). However, it is the relatively recent advances in neuroanesthesia, knowledge of the surgical anatomy of the skull base, and new instruments like power tools and the operating microscope that made these approaches safer and more popular.
Cranioplasty is almost as ancient as trephination, yet its fascinating history has been neglected. There is strong evidence that Incan surgeons were performing cranioplasty using precious metals and gourds. Interestingly, early surgical authors, such as Hippocrates and Galen, do not discuss cranioplasty, and it was not until the 16th century that cranioplasty in the form of a gold plate was mentioned by Fallopius. The first bone graft was recorded by Meekeren, who in 1668 noted that canine bone was used to repair a cranial defect in a Russian man. The next advance in cranioplasty was the experimental groundwork in bone grafting, performed in the late 19th century. The use of autografts for cranioplasty became popular in the early 20th century. The destructive nature of 20th century warfare provided an impetus to search for alternative metals and plastics to cover large cranial defects. The metallic bone substitutes have largely been replaced by modern plastics. Methylmethacrylate was introduced in 1940 and is currently the most common material used. Research in cranioplasty is now directed at improving the ability of the host to regenerate bone. As modern-day trephiners, neurosurgeons should be cognizant of how the technique of repairing a hole in the head has evolved.

Even though Washington, DC, is more known for its impact on the national and international political arenas, numerous contributions to neurosurgery were made by neurosurgeons who either practiced or trained in the national capital. Neurosurgery was practiced early in the area by Harry Hyland Kerr, a general surgeon who spent 6 months with Harvey Cushing in the American Expeditionary Force during World War I. Kerr was also one of the earliest members of the Society of Neurological Surgeons. The first fully trained neurosurgeon was John Shugrue who trained at the Mayo Clinic between 1925 and 1928. However, it was not until 1935 that neurosurgery in the national capital gained a different dimension with the arrival of James Watts, after 7 years spent training with various eminent figures of the period. Along with Freeman, he introduced psychosurgery to the United States, further developed it, and promoted it worldwide, putting Washington, DC, on the international neurosurgical map. Among the other figures are William Spence, credited for using methylmethacrylate for cranioplasties and who performed the first carotid endarterectomy, and Ayub K. Ommaya, known for his work on head trauma and his famous “reservoir.” Some of the early steps of endovascular neurosurgery were made at Georgetown University with Alfred Luessenhop, including AVM embolization (and even endovascular aneurysm obliteration). It is at Howard University or Freedman’s Hospital that the first board-certified African-American neurosurgeon, Clarence Greene, practiced. Some of the neurosurgical figures at Walter Reed were Colonel Georges Hayes, and Ludwig Kempe and his well-known book on neurosurgical operative techniques. Hugo Rizzoli is another eminent neurosurgeon in this area, where he practiced since 1945, and helped train generations of neurosurgeons. DC residency programs were established at George Washington University in 1940, at Georgetown University in 1952, and at the Walter Reed Army Medical Cen-
ter, and many of the graduates went on to occupy major positions in national and international neurosurgery, like John Fox, Wolfgang Koos, Manuel Velasco-Suarez, and others.

PAPER #810 TUESDAY, 4:15-4:30 PM
Egyptian Medicine From Ais (Brain) to Tjes (Vertebrae), or A History of Ancient Neurosciences

Mary L. Hlavin (Cleveland, OH)

Key Words: ancient neurosciences, Egyptian medicine, neurosurgical history

The ancient Egyptians displayed some of the earliest knowledge of basic neuroanatomy, physiology, and neurological disease. Although studies were limited by respect for the dead under Pharonic law, external anatomy was quite well described by the ancient Egyptians. The skull received greatest attention and was described in the most detail. Knowledge of the cranial contents or "viscera" is well documented, although there is little evidence to suggest that its function was understood. In fact, the Egyptians related emotion to the heart and, at embalming, the brain was discarded. They were aware, however, that the spinal cord transmitted motor function. Familiarity with multiple neurological diseases, including Pott’s disease, leprosy, polio, and hydrocephalus, as well as the numerous complications of head injury, can be found throughout the medical papyri and on stone carvings. The ancient embalmers demonstrated exquisite technical expertise removing the brain through small openings in the cribriform plate. It appears, however, that there was little contact between embalmers and physicians, and trephination was not an integral part of Egyptian medicine. This presentation will trace the origins of neurosciences in ancient Egyptian civilization.

PAPER #811 TUESDAY, 4:30-4:45 PM
Origins of Neurosurgery in the Seventeenth Century

Eugene S. Flamm (Philadelphia, PA)

Key Words: neurosurgical history, surgical observations, seventeenth century

Origins of neurosurgery can be found in the surgical literature of the 17th century. Based on these works, neurosurgery can be considered a specific area of medical expertise at that time, when a clear definition of anatomy in the works of Willis, Steno, Blasius, and Vieussens developed. Iatromechanical and iatrochemical schools led by Descartes and Willis attempted to provide a physiological system that corresponded to the anatomy. The coupling of anatomical and physiological systems was an important step toward placing the surgical management of neurological disorders on a rational footing. Ultimately this would lead to precise cerebral localization in the 19th century.

Surgical advances were influenced by the need to deal with a persistent array of civilian and military injuries. The general surgical texts of Hildanus, Scultetus, Wiseman, and Woodall were based on case material rather than interpretations of the Hippocratic writings that comprised the literature of the 16th century. In the 17th century surgeons addressed the indications and methods of performing surgery as well as new instrumentation for increasing safety and efficiency. Works by Yonge, Ryder, and others emphasized that patients could recover from cranial operations.

This paper presents the early developments that led to successful surgical management of neurological disorders. It is the hypothesis of this communication that the roots of modern neurosurgery arose gradually through individual surgical observations and not simply as a result of the major advances of anesthesia, aseptic surgery, and cerebral localization in the 19th century.
Trephination of the cranial vault is the oldest known surgical procedure and has often been reported in the literature. Little knowledge, however, exists on the neurosurgical disorders of ancient skulls. In order to examine the incidence of disorders of special interest to the neurosurgeon the present study has been performed. Of a total 115 neolithic (2500–3500 B.C.) skulls found in the region of Mecklenburg-Vorpommern (northeast Germany), 113 were examined. Defects, abrasions, etc., were detected in 31 of these skulls and required further examination (careful microscopic and/or endoscopic examination, CT, and x-ray study).

Five skulls showed defects resulting from trephination, mainly located in the parietal or parietooccipital region. There was good osteological evidence that at least 2 of these operations had been survived. From the interpretation of multiple cuts around two of these trephinations we conclude that a cutting flintstone-technique was used to cause these defects. Other disorders found were: multiple depressed skull fractures (2 cases, both survived), singular depressed skull fracture (survival unclear), mandibular and zygomatic fracture (1 case survived), synostosis of skull sutures (2 cases).

From the present study the authors conclude that the incidence of trephination in neolithic skulls in our region is at least 5% and that these operations were survived in occasional cases. Whether these procedures were intended to be curative or were performed for other reasons still remains unclear.

Three-dimensional models represent a heuristic method through which relevant spatial anatomical information may be analyzed in presurgical planning. The history of anatomical studies of the head and brain and their surgeries are interlinked with the development of anatomical models which provided an important basis for the training of early neurologically oriented practitioners.

It was Leonardo who pioneered the concept of the anatomical model with his wax casting of the ventricular system, but not until Gaetano Giulio Zumbo (1656–1701), a Sicilian artist, did the development of anatomical modeling techniques come into its own. In collaboration with the French surgeon Guillaume Desnoues, Zumbo adapted methods based on wax sculpting techniques to anatomical models. His models are stunning for their anatomical accuracy and fine composition as well as their macabre settings. The school that he founded would have its greatest practitioner 100 years later in Clemente Susini (1757–1814), a Florentine artist commissioned by the then newly created Museum of Specola. Susini established a “Ceroplastica” laboratory where artisans and anatomists in close collaboration created over 2000 specimens, which survive now in greatest number at the Specola as well as at the Josephinum in Vienna.
Anatomy was the most advanced science of the 18th century, and the anatomical waxes were critical to the dissemination of rapidly accruing discoveries. By faithfully reproducing important dissections, they served as a means through which the insights of master anatomists could be preserved in a permanent manner. We will discuss the fabrication of these models, their implications to the state of neuroanatomical understanding of the 18th century, and their significance to the development of neuroanatomy and the practice of the clinical neurosciences.

PAPER #814  TUESDAY, 5:15-5:30 PM
An Analysis of Harvey Cushing’s Philosophy on the Management of Head Injury

William C. Bergman
Sandy A. Shatsky
Raymond A. Schulz (San Jose, CA)

Key Words: Harvey Cushing, head injury, war wound, neurosurgical history

Even before the United States entered the First World War, Major Harvey Cushing had volunteered to serve as a combat surgeon in France. His experience there, with over 100 operations for penetrating wounds of the brain, was published in the British Journal of Surgery, in 1918. This article, considered by many to be the most important contribution to the neurosurgical literature during the war, offers tremendous insight into the philosophy and thought processes of Dr. Cushing at that time. Each patient is described in a concise paragraph, most accompanied by a hand-drawn illustration of the fragment trajectory, the fragment itself, or a facsimile of the temperature chart. The many drawings present in this article are done either by Dr. Cushing himself, or by one of his sergeants, an obviously accomplished medical illustrator, whom Cushing emulated. When this article and a surgical technique companion article published in the British Medical Journal that same year are compared to Dr. Cushing's previous writings on head injury, a significant understanding of what he gleaned from his wartime experience can be ascertained. A prime example of this can be seen when the techniques espoused in the article and subsequent literature are compared with his earlier works, such as his chapter in Dr. Keen’s textbook. This paper analyzes the above-mentioned papers and specifically compares them to other neurosurgical trauma literature from the same era.
Tuesday Afternoon

SECTION SESSION

AANS/CNS Section on Pediatric Neurological Surgery

2:45 PM – 5:30 PM (Room 103A/B/C)

DONALD MATSON MEMORIAL LECTURE

AANS/CNS Section on Pediatric Neurological Surgery

2:45 PM – 3:15 PM (Room 103A/B/C)

REMINISCENCES OF A PEDIATRIC NEUROSURGEON

Luis Schut, MD (Philadelphia, PA)
(To be introduced by Thomas G. Luerssen, MD)
Dr. Michael Drewek was born in Racine, Wisconsin. He earned his Bachelor of Arts degree in Biological Sciences from The University of Chicago with Honors for his work on apolipoprotein gene expression. He obtained his MD from the University of Wisconsin. While in medical school, in the laboratory of Dr. Thomas Duff, he studied the potential role of endothelin on vasospasm. Currently, Dr. Drewek is completing his fifth year of residency in neurological surgery at Vanderbilt University. His current research interests focus on the toxic effects of amniotic fluid on rat fetal spinal cord cultures.
Tuesday Afternoon

SECTION SESSION

AANS/CNS Section on Pediatric Neurological Surgery

3:30 PM – 5:30 PM (Room 103A/B/C)

Moderators: Marion L. Walker, MD (Salt Lake City, UT)
Thomas G. Luerssen, MD (Indianapolis, IN)

PAPER #815 TUESDAY, 3:30-3:45 PM
Cerebellar Astrocytomas in the Age of Modern Imaging

Jeffrey W. Campbell
Ian F. Pollack
Ronald Hamilton
Barbara Shultz (Pittsburgh, PA)

KEY WORDS: astrocytoma, tumor resection, prognosis

Eighty-five patients diagnosed with cerebellar astrocytomas between 1975 and 1993 were identified from the Children’s Hospital of Pittsburgh Tumor Registry. This time span allowed for modern imaging with CT or MRI in all patients as well as allowing a minimum of 3 years follow up. Tumor pathology was independently reviewed by a neuropathologist blinded to patient outcome using the WHO classification system. Patient characteristics and outcome were determined by retrospective chart review, and degree of operative debulking was determined by postoperative imaging. Patients were regarded as having gross-total resection (GTR) of tumor only if both the postoperative imaging and the operative report confirmed this.

After review of the surgical specimens for this series, 9 patients were excluded either because of lack of a specimen to review (n = 6) or because tumors contained a significant nonastrocytic component (n = 3). On chart review, an additional 4 patients were excluded either for a predominant brainstem location of the tumor (n = 2) or because they suffered from neurofibromatosis (n = 2). Of the remaining 72 patients, there were 53 juvenile pilocytic astrocytomas (JPA), 12 low-grade astrocytomas (LGA), and 7 high-grade astrocytomas (HGA). The median age of patients with LGA (54 months) was significantly lower (p = 0.001) than of those with JPA (84 months) or HGA (100 months). Median progression-free survival (PFS) and total survival (TS) in patients with HGA were 6 and 8 months, respectively. While median PFS and TS were not reached in the patients with JPA or LGA, those with JPA had significantly better PFS (p = 0.01) and TS (p = 0.005) than those with LGA.

Life table analysis estimates that, at 5 years after diagnosis, 80% of JPA vs 50% of LGA will not progress, while 100% of patients with JPA vs 73% of patients with LGA will survive. Patients who underwent GTR of their LGA or JPA had nearly identical PFS and TS, with 100% TS in both groups. Conversely, patients who had subtotal resection of tumor did much better in the JPA group, with 5-year PFS of 45% vs 17% (p = 0.02) and 5-year TS of 100% vs 25% (p = 0.003). In patients with incompletely resected JPA, postoperative radiation significantly decreased the incidence of progression (p = 0.0023), while having no impact on TS. High-grade astrocytomas of the cerebellum
carry a uniformly poor prognosis in our experience, with no patient surviving 2 years after diagnosis. Conversely, JPA or LGA that are completely resected carry a very favorable prognosis with no fatalities and only 3/44 patients with any progression. However, patients with incompletely resected lesions suffered a much higher rate of progression and a significant rate of mortality in those with LGA. Pathology was predictive of outcome only in the patients with incomplete resections. The treatment of choice for JPA and LGA is complete surgical excision. Incompletely resected LGA are a fatal disease in many patients and should be treated aggressively.

PAPER #816 TUESDAY, 3:45-4:00 PM
External Hydrocephalus: A Pitfall in the Evaluation of Suspected Child Abuse

Joseph H. Piatt (Portland, OR)

Key Words: hydrocephalus, craniocerebral disproportion, retinal hemorrhage, child abuse

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

Two infants are presented who suffered acute neurological depression after what their caretakers described as minor trauma. Imaging studies in both patients demonstrated diffuse enlargement of the subarachnoid spaces with modest acute subdural hematomas. Funduscopic examination by consultant ophthalmologists disclosed retinal hemorrhages in both cases, documented by fundus drawings and fundus photography. There was no neurosurgical intervention in either case, and both patients recovered their respective neurological and developmental baselines. Because the neurosurgical consultant had to concede that the caretakers’ descriptions of the injuries might be true, both infants were eventually returned to their homes. Neither infant has suffered recurrent injury after 18 and 9 months follow up. Physicians who offer treatment to and testimony about infants with head injuries must be cognizant of the susceptibility of patients with external hydrocephalus to subdural and retinal hemorrhage after minor trauma.

PAPER #817 TUESDAY, 4:00-4:15 PM
Psychopharmacological Treatment of Pediatric Brain Injury

David Mahalick
Peter Carmel
Walter Molofsky
Jacqueline Bartlett
Mark McDonough
John P. Greenberg (Newark, NJ)

Key Words: brain injury, attentional disorder, psychostimulant therapy

The incidence of pediatric traumatic brain injury is high. Recent epidemiological evidence suggests that approximately 294 of every 100,000 children sustain significant head injuries each year. Research conducted on children who have sustained various types of brain injury has demonstrated that acquired attentional disorder are the primary features associated with pediatric brain injury. The usefulness of psychostimulant therapy in treating traumatically induced attentional disorders in pediatric populations has not been studied.
Fourteen children who had sustained traumatic brain injury (TBI) were recruited for participation in this study. The research design was a randomized, double-blind, placebo-controlled, cross-over design. Methylphenidate was administered in a dose of 0.3 mg/kg twice daily. A placebo (lactose) was administered in the form of glucose capsules with an appearance identical to the actual medication. Neuropsychological tests were administered at the end of the first week of each 2-week period. On Day 15, patients who had been receiving methylphenidate began receiving placebo; alternatively, those who had been receiving placebo began receiving methylphenidate. Patients and investigators were both blind to the treatment conditions.

Multiple one-tailed t-tests for related measures were computed on each of the dependent measures and are presented. Inferential statistics were employed to test the hypothesis that: a) performance on baseline measures of attention would not be significantly different than performance during the placebo condition; and b) performance on tasks assessing attention and concentration would be significantly better during the drug vs placebo condition. Both of the latter propositions received statistical support. Differences were considered to be significant at the alpha = 0.05 level. Methylphenidate was found to have a very significant beneficial impact in the cognitive functioning of children with TBI. Generalization of these findings is somewhat limited due to the restricted sample.

PAPER #818 TUESDAY, 4:15-4:30 PM
Radiographic and Morphological Characterization of a New Model of Hydrocephalus in Transgenic Mice

Alan Cohen
David Leifer
Jonathan Lewin
W. David Lust (Cleveland, OH)

KEY WORDS: hydrocephalus model, transgenic mouse

Despite the abundance of available experimental models, the pathophysiology of hydrocephalus is still poorly understood, and many of the models are flawed, expensive, or troublesome to use. This report describes the radiographic and pathoanatomical findings in a new model of hydrocephalus occurring in transgenic mice generated to overproduce transforming growth factor (TGF)-β1. The multigene cytokine TGF-β1 has broad regulatory roles in cell growth and differentiation. Although it and its receptors are expressed in the choroid plexus and meninges, the precise role of TGF-β1 in the developing CNS is unknown. To evaluate its role in CNS development, Messing and collaborators created a transgenic mouse (TgN4Mes) designed to overexpress TGF-β1 in the CNS. Unexpectedly, the transgenic mice developed severe hydrocephalus. We perpetuated a line from this transgenic strain and used it to serve as a model of experimental hydrocephalus. Pups from 5 litters were examined. The transgenic pups developed clinical manifestations of hydrocephalus either at birth or shortly thereafter, manifested by enlargement and doming of the dorsal calvaria and subsequent gait abnormalities. These manifestations were not seen in nontransgenic littermates. Presence of the TGF-β1 transgene was determined by polymerase chain reaction analysis of tail cuts.

To characterize the hydrocephalus anatomically, we performed MRI on animals sedated with intraperitoneal thiopental at 1 and 2 weeks postnatal (weight < 10 g). High-resolution proton density gradient echo MRIs were obtained using a specially designed 1-cm-diameter solenoidal receiver coil and a 1.5-Tesla magnet. Hydrocephalic animals showed massive enlargement of the lateral, third, and fourth ventricles on 3-D volumetric scans compared with nonhydrocephalic controls. The aqueduct was narrow but patent. Morphological examination of frozen brains demonstrated that marked ventriculomegaly was present at birth.

This study demonstrates that transgenic mice bred to overexpress TGF-β1 in the CNS can serve as a reproducible model of communicating hydro-
cephalus. It also demonstrates the feasibility of using high-resolution MRI to characterize hydrocephalus in the small neonatal mouse. The precise role of TGF-β1 in the pathogenesis of hydrocephalus is currently under investigation.

PAPER #819  TUESDAY, 4:30-4:45 PM
Surgical Results for Frontal Lobe Epilepsy in Children

Mark R. Lee
Joseph R. Smith
Mark D. Smith (Augusta, GA)

Key Words: epilepsy surgery, frontal lobe

Epilepsy surgery is increasingly accepted as a treatment for intractable epilepsy in children. Frontal lobe epilepsy is relatively common; however, it poses a particular challenge in diagnosis and surgical management. Thus, we find it timely to review our surgical results and experience in children with frontal lobe epilepsy. Seventeen children (age at surgery, 6–18 years, mean 13.3 years) underwent ablative frontal resections at our institution between 1987 and 1996. All children had medically intractable epilepsy (duration of seizure disorder 0.5–14 years, mean 5.6 years), and had failed multiple medical regimens. All children underwent evaluations that included 24-hour video EEG monitoring and Wada tests in age-appropriate patients. Eleven children (65%) underwent further evaluation utilizing invasive monitoring: 4 children had depth electrodes only, 4 children had subdural grid electrodes only, and 3 children had both depth and subdural grid electrodes implanted. Surgical resection was tailored according to the results of invasive monitoring, intraoperative cortical EEG, and intraoperative functional mapping in selected patients.

Nine children had lesions and 8 did not have lesions. All children with lesions also underwent resection of cortical tissue in addition to lesion resection. Seizure outcome was reviewed after 1-year follow up, and was graded as seizure free or not seizure free. Outcomes were analyzed in regard to age at seizure onset, duration of seizure disorder, age at surgery, aura and seizure type, interictal EEG, ictal EEG, findings of invasive monitoring, extent and type of surgical resection, and pathology.

Eleven children (65%) were seizure free after 1 year follow up. Analysis of these children revealed that the presence of a lesion was significant for seizure free outcome. All 9 patients with lesions were seizure free after surgical resection, while only 2 (25%) of 8 children without lesions were seizure-free (p < 0.05). In addition to the analysis of outcome, we will discuss in detail the clinical presentation, the preoperative evaluation, and the surgical technique for children with frontal lobe epilepsy. In doing so, we hope to further elucidate the complexity of frontal lobe epilepsy and provide additional data for predicting outcome in this form of epilepsy surgery for children.

PAPER #820  TUESDAY, 4:45-5:00 PM
Complications and Hazards With Invasive Electrodes in Pediatric Epilepsy Surgery

Steven J. Schiff
Steven L. Weinstein
Joan Conry
William D. Gaillard (Washington, DC)

Key Words: epilepsy surgery, electrode monitoring, children

To identify and categorize problems associated with depth and subdural electrode use in pediatric epilepsy patients the charts of 37 consecutive patients requiring 55 surgical procedures since 1992 were reviewed. Sixteen patients required depth and/or subdural electrode implantation for seizure localization and functional mapping. Ten patients had epilepsy (Engle) Class I or II outcomes (none or rare seizures), and there were no infections. Place-
Notes
ment of large grids in the very young child required relaxing incisions in the flat grids to match the increased curvature of the small brain.

One cortical contusion and 1 life-threatening subdural hematoma were observed in children aged 18 and 9 months, respectively. Two patients had breakage of fragile twisted wire depth electrodes from excessive movement during chronic monitoring, despite 1:1 nursing attendance. In 3 patients, electrode leads were cut by staff during dressing changes and one depth electrode fractured during electrode removal, in patients not generally cooperative with bedside procedures. Problems with incorrect placement of depth electrodes with newer flexible stylets were noted, and misplacement of a subdural bone flap for subdural grid placement occurred. Four patients had no focal resections offered, either because no seizures occurred during the 3 weeks maximum time permitted for subdural grid implantation or because the data collected indicated that resection was not feasible.

Invasive EEG monitoring has significant inherent risks, but certain hazards are accentuated in children and should be anticipated. Alternatives to chronic electrode monitoring should be considered in the very young.

PAPER #821 TUESDAY, 5:00-5:15 PM
Intraventricular Delivery for Gene Therapy in the Fatal Pediatric Neurogenetic Disorder, Canavan Disease
Matthew During (Auckland, New Zealand)∗
Andrew Freese (Haverford, PA)
Frank Sorgi (Philadelphia, PA)
Leaf Huang (Pittsburgh, PA)
Larissa Bilaniuk
Zhiyue Wang
Linda C. Mayes
Magretta R. Seashore
Edward Mee (Philadelphia, PA)
Paola Leone (New Haven, CT)

KEYWORDS: Canavan disease, leukodystrophy, gene therapy

The leukodystrophy, Canavan disease, is caused by mutations in the aspartoacylase (ASPA) gene. The subsequent loss of ASPA activity leads to an elevation in brain levels of N-acetyl-aspartate (NAA), resulting in spongiform degeneration of oligodendrocytes. Clinically, afflicted children demonstrate neurodevelopmental retardation and die at a young age.

We generated a liposome-encapsulated, polycation-condensed, plasmid DNA (LPD) complex expressing ASPA and delivered this LPD/ASPA complex intraventricularly in 2 children with progressive Canavan disease. They were followed with serial MRI, proton nuclear magnetic resonance spectroscopy (NMRS) of NAA, and psychometric testing. A control group of 5 children with Canavan disease was similarly followed.

The LPD/ASPA-treated children showed no significant adverse events. At 1 month after surgery, both children had normal levels of NAA in frontal and parietal regions. In 1 child, NAA levels remained in the normal range for 12 months. On repeated MRI, this child had a significantly improved myelin signal at 12 months, which was associated with improved neurodevelopmental scores and normalization of visual evoked potentials. The 5 untreated children with Canavan disease had stable, elevated NAA levels and no improvement in the MRI myelin signal.

Intraventricular delivery of ASPA/LPD complex normalized NAA levels and improved neurodevelopmental status. In 1 child, an observed change in the white matter MR signal indicated remyelination, which was not seen in any of the untreated children with Canavan disease. These results from the first gene therapy trial of a neurometabolic disorder indicate that direct in vivo gene therapy of Canavan disease is safe and may be associated with bio-
chemical, radiological, and clinical improvement. Expansion of this trial is underway to further establish safety and potential efficacy.

PAPER #822 TUESDAY, 5:15-5:30 PM
Anterior Circulation Strokes Following Nonaccidental Head Injuries in Children: A Proposed Mechanism

E. Christopher Troup (Macon, GA)
Robert Sanford
Deanna Ratliff (Memphis, TN)

Key Words: child abuse, anterior circulation infarct

Nonaccidental head injuries account for a significant morbidity and mortality on a pediatric neurosurgical service. We have encountered several cases in which children with a nonaccidental head injury (child abuse) developed unilateral anterior circulation infarcts. The infarcts also appeared ipsilateral to the most severe soft-tissue injury.

Three infants will be presented, all with nonaccidental head injuries, who developed unilateral anterior circulation infarcts. Each child had a small bruise on the neck, just below the mandible ipsilateral to the infarct, and soft-tissue injuries on the head and face. Two of these infants had left-sided injuries and one had right-sided injuries. The 2 infants with left-sided pathology had a right-handed abuser and the infant with the right-sided pathology had a left-handed abuser. The proposed mechanism involves transient carotid artery compression by the abuser’s nondominant thumb while the infant is held with the nondominant hand and struck with the dominant hand. In the case of the right-sided infarction the senior author predicted the left handedness of the person subsequently charged by authorities.

Clinical presentation, neuroimaging, and clinical management will be discussed.
Tuesday Afternoon

SECTION SESSION
AANS/CNS Section on Stereotactic and Functional Neurosurgery
2:45 PM – 5:30 PM (Room 113A/B/C)

SPECIAL SYMPOSIUM
AANS/CNS Section on Stereotactic and Functional Neurosurgery
2:45 PM – 3:45 PM (Room 113A/B/C)

ELECTRICAL STIMULATION OF THE CNS

Moderator:
Andres M. Lozano (Toronto, ON, Canada)
Douglas Kondziolka (Pittsburgh, PA)

Panelists:
Jean Siegfried, MD (Zurich, Switzerland)
Alim Benabid, MD (Grenoble, France)
Tuesday Afternoon

**SCIENTIFIC SESSION**

AANS/CNS Section on
Stereotactic and Functional Neurosurgery

3:45 PM – 5:30 PM  (Room 113 A/B/C)

**Moderators:** David W. Roberts, MD (Lebanon, NH)
Philip Gildenberg, MD (Houston, TX)
Dr. Emad N. Eskandar was born in Egypt but grew up in Omaha, Nebraska. He attended the University of Southern California School of Medicine where he was elected to Alpha Omega Alpha and graduated with the highest distinction in 1993. During medical school he spent two years at the National Institutes of Health, with the support of the Howard Hughes Research Scholars program, studying the role of the temporal cortex in the processing of information related to vision and memory.

Following medical school, Dr. Eskandar moved to Boston where he has been a resident on the Neurosurgical Service at the Massachusetts General Hospital. His research interests focus on understanding the processes underlying sensorimotor integration in the cortex and in the basal ganglia. Over the past two years, he has been working at Harvard Medical School where he has been studying the role of the parietal cortex in motion perception by recording the activity of single neurons in animals trained to perform visual guidance tasks. He has won several awards for his research including a fellowship from the National Eye Institute and the R. S. Morrison Fellowship from the Grass Foundation.

Dr. Eskandar enjoys golfing, fishing, hiking, scuba diving, and playing guitar.
The Role of Posterior Parietal Cortex in the Visual Guidance of Movement

Emad N. Eskandar
John A. Assad (Boston, MA)

KEY WORDS: visual localization, posterior parietal cortex, movement

A basic issue to be resolved in the study of the primate visual system is the question of how the brain uses visual information to guide movements. Numerous lines of evidence suggest that the posterior parietal association cortex (PP) plays an important role in the visual guidance of movements, although the nature of this role is unclear. The PP receives afferents from visual and somatosensory areas and projects to premotor areas. Lesions of PP produce a variety of debilitating deficits including hemi-neglect, problems in visual localization, and loss of spatial memory. Recently, a number of electrophysiological studies have revealed neuronal activity in PP that is not directly driven by visual stimulation and thus may reflect planning or guidance of movements. The experiments described here were designed to examine the relationship between these signals and hand movements.

We recorded from single neurons in the PP of a rhesus monkey trained to use a joystick to guide a small spot to a target on a computer monitor. In some of the trials the spot was invisible for most of its trajectory so that the animal had to “infer” or predict the direction of movement. Neurons responded selectively to particular directions of movement even when the stimulus was not visible. The directionality could have been due to the hand movement or to the animal inferring the direction of stimulus motion. To differentiate between the two, the animal was trained to perform the task with the direction of hand movement either the same as or opposite to the direction of stimulus motion. One hundred four cells were recorded from the intraparietal sulcus within the PP. Fifty cells (48%) were significantly modulated by the inferred direction of stimulus motion whereas 30 cells (29%) were significantly modulated by the direction of hand movement (two-way ANOVA, p < 0.05).

We conclude that many neurons in the posterior parietal cortex encode an abstract or predictive representation of moving stimuli that is distinct from motor planning. Such a signal would be useful to systems controlling hand or other types of movements.

Cognitive and Intellectual Dysfunctions Following Unilateral Posteroventral Pallidotomy in Parkinson’s Disease

Tetsuo Yokoyama
Yoko Imamura
Kenji Sugiyama
Shigeru Nishizawa
Kenichi Uemura (Hamamatsu, Japan)

KEY WORDS: Parkinson’s disease, pallidotomy, cognitive deficit

Pre- and postoperative cognitive functions were evaluated with the Hamamatsu Higher Brain Function Scale (HHBFS) in 15 patients with Parkinson’s disease (PD) who underwent unilateral posteroventral pallidotomy (PVP) to clarify its effect on cognitive functions. The HHBFS consists of Orientation, Digit Span (forward and backward), Digit Learning, 5-Minute Memory of 5 Words, Similarities, Serial 7s, Animal Name Listing, and Kana Pick-out Test, as well as the Mini Mental State (MMS). The patients consisted of seven men and eight women with a mean age of 66.8 ± 7.4 years (± SD). They were classified into two groups according to preoperative modified Hoehn & Yahr Staging (H & Y); 12 patients had a H & Y of less than 4.0 (not advanced PD) and three had a H & Y of more than 4.0 (advanced PD). Preoperative scores of the both tests were compared to those obtained from 12 healthy elderly subjects.
The MMS scores were normal (27.5 ± 1.9) in the 12 without advanced PD, but the HHBFS scores for Similarity, Serial 7s, Animal Naming, and Kana Pick-out Test (story) were significantly lower than those of control subjects (t-test, p < 0.05). In the 3 patients with advanced PD, the MMS scores showed dementia (13.3 ± 11.7), and all tasks of HHBFS except Orientation, Digit Span, and Digit Learning were impaired (t-test, p < 0.01). In the 7 patients without advanced PD who were operated on the left side, the evaluation at 1 month after operation showed impaired Animal Naming (p < 0.05), Kana Pick-out Test (story), and MMS (t-test, p < 0.01), but all of them improved to their pre-operative levels 3 months after the operation. No significant impairment of respective tasks was noted in patients who were operated on the right side. Contrary to these, Similarity significantly improved within 1 month after the operation in all 12 (p < 0.05). In advanced PD, Kana Pick-out Test (story) and Meaning of story were significantly impaired (p < 0.01), and these remained unchanged 3 months after the operation.

This is the first presentation to report that cognitive deficits were present temporarily for 1 or 2 months after PVP in the patients without advanced PD who were operated on the left side, but no remarkable deficits were noted in the patients who were operated on the right side. The patients with advanced PD have a risk of significant cognitive deficits after the operation.

PAPER #825  TUESDAY, 4:15-4:30 PM
Correlation Between Number of Transduced Cells and Behavioral Recovery in a Rat Parkinson’s Disease Model Following In Vivo Gene Therapy

Andrew Freese (Philadelphia, PA)
Daniel Young
David Silver
Charles Liu (Auckland, New Zealand)
Beverely Davidson (Iowa City, IA)
Michael J. O’Connor (Philadelphia, PA)
Paola Leone (New Haven, CT)
Matthew J. During (Auckland, New Zealand)

KEY WORDS: Parkinson’s disease, gene therapy, behavioral recovery

A number of gene therapy studies have focused on repleting lost dopaminergic function in animal models of Parkinson’s disease (PD) using methods to introduce genes encoding dopamine (DA) biosynthesis directly into the corpus striatum. The best established genetic target is tyrosine hydroxylase (TH), the rate limiting enzyme in DA biosynthesis, converting tyrosine into l-dopa. Until now, although encouraging results have been obtained, a quantitative evaluation of the correlation between number of transduced striatal cells and behavioral recovery has not been performed. Prior to consideration of human clinical application, such an analysis would be necessary.

An adenovirus vector expressing human TH (form II; AdTH) and controls (either an adenovirus vector expressing E. coli β-galactosidase [AdLacZ] or phosphate-buffered saline [PBS]) were used. The AdTH was stereotactically injected intrastratally into three groups of 6-hydroxydopamine-treated rats: 1) a single site, 2) 4 sites, and 3) 8 sites. Apomorphine-induced rotational behavior was assessed prior to vector or control injection, and for 6 weeks at weekly intervals thereafter. At the end of the experiments, the animals were sacrificed and TH immunoreactivity was assessed.

A direct correlation was observed between the number of TH immunoreactive cells and behavioral recovery in the 6-hydroxydopamine rotational model of PD following human TH gene transfer using an adenovirus vector, when compared to controls. In the subset of rats that received 4 or 8 injections, up to 100% behavioral recovery was observed. A minimum threshold of transduced cells following a single injection in some rats was observed; below this, behavioral recovery was not seen.
These results indicate that behavioral recovery in this model of PD following human TH gene transfer is directly proportional to the number of transduced cells, suggesting the importance of adequate and efficient gene delivery for consideration of eventual application to patients with PD. Further evaluation of gene transfer systems, their efficiency, and extent of striatal transfection is underway.

PAPER #826 TUESDAY, 4:30-4:45 PM
Intraoperative Hippocampal Electrocorticography Predicts the Extent of Hippocampal Resection in Temporal Lobe Epilepsy Surgery

Guy M. McKhann
Julie Schoenfeld
Donald E. Born
George A. Ojemann (Seattle, WA)

KEY WORDS: epilepsy, temporal lobe, hippocampus resection

Despite the many surgical procedures utilized in the treatment of mesial temporal lobe epilepsy, no consensus exists regarding how much hippocampus should be removed. Whether all patients require a maximal hippocampal resection has not been determined. At the University of Washington, all temporal lobe epilepsy operations are tailored by intraoperative electrocorticography (ECoG). The amount of hippocampal resection is based on the extent of interictal epileptiform abnormalities, resulting in a resection that is individualized for each patient. Using this approach, we prospectively followed 141 consecutive patients operated on for mesial temporal lobe epilepsy with pathological diagnoses of varying degrees of hippocampal sclerosis or gliosis to determine whether the extent of hippocampal resection correlates with outcome when a tailored approach is used. Additionally, we analyzed whether residual interictal epileptiform activity following temporal resection predicts poorer seizure control.

With at least 1 year of clinical follow up, 67% of the patients were seizure free or had a single postoperative seizure. There was no correlation between hippocampal resection size and seizure control ($r = -0.09, p = 0.3$). Using an intraoperatively tailored strategy, individuals with a larger hippocampal resection (> 2.0 cm) were not more likely to have a seizure-free outcome than patients with smaller resections ($p = 0.7$). Additionally, patients with residual postresection epileptiform hippocampal, but not cortical or parahippocampal, interictal ECoG activity had a significantly worse seizure outcome ($p < 0.0001$). We conclude that intraoperative hippocampal ECoG can predict how much hippocampus should be removed to maximize seizure-free outcome, allowing for sparing of possibly functionally important hippocampus.

PAPER #827 TUESDAY, 4:45-5:00 PM
Comparison of Stereotactic Open Radiofrequency Thalamotomy/ Pallidotomy With Gamma Knife Radiosurgical Lesions for Medically Intractable Movement Disorders

H. Warren Goldman
Stephen Gollomp (Philadelphia, PA)
Anthony Alta (Wynnewood, PA)
Janet Scanlon
Karen McGlinn (Philadelphia, PA)

KEY WORDS: radiosurgery, thalamotomy, pallidotomy, movement disorder

Renewed interest in the surgical management of certain movement disorders in recent years has led to increasing numbers of patients seeking surgical options when they reach medical intractability. A subgroup of this population may not be optimal candidates for conventional stereotactic surgery because of concomitant use of anticoagulants, medical infirmity, or advanced
age. In an effort to assess the relative safety and efficacy of radiosurgical thalamotomy/pallidotomy, we have reviewed the outcome of this procedure in patients deemed suitable candidates for surgery on neurological grounds but who are poor medical risks.

Sixteen patients underwent either radiosurgical Vim thalamotomy or GPi pallidotomy. Magnetic resonance imaging-based stereotactic localization was used as the sole imaging technique for target identification in conjunction with the Schaltenbrand and Wharen Atlas. Thirteen patients were treated for tremor (9 for essential tremor and 4 for parkinsonian tremor), 2 patients had enlargement of previously successful pallidotomies, and 1 patient had a sensory thalamotomy for thalamic pain syndrome. The clinical outcome and accuracy of lesion placement in this group was compared with historical controls from our surgical program.

All patients were significantly improved with major reduction in tremor in 84% of the group who underwent thalamotomy and excellent resolution of the dyskinesia and rigidity in the pallidotomy group. Modest though significant benefit was observed in the case of thalamic pain syndrome. Two patients with PD who had Vim lesions had larger than expected lesions with T2-weighted MRI signal changes extending beyond the limits of the intended targets. The appearance on postoperative MRI was inconsistent with the more benign clinical course. Although both patients received a regimen of intravenous high-dose dexamethasone, reversal of the MRI changes as well as clinical decline was achieved without permanent neurological sequelae. Placement of the intended lesions as seen on 3-month follow-up MRI was precisely as predicted. Overall efficacy was indistinguishable from that seen in patients who were treated with conventional radiofrequency lesions.

Gamma knife radiosurgical treatment for medically intractable movement disorders appears to compare favorably with open radiofrequency techniques without the risks of electrode placement.

**PAPER #828 TUESDAY, 5:00-5:15 PM**

**Effects of Apomorphine on Globus Pallidus Neurons in Parkinsonian Patients**

Andres M. Lozano*
William D. Hutchison
Ronald Levy
Jonathan O. Dostrovsky
Anthony E. Lang (Toronto, ON, Canada)

**KEY WORDS:** apomorphine, globus pallidus, Parkinson’s disease

Current hypotheses of basal ganglion dysfunction in Parkinson’s disease (PD) propose that a loss of dopaminergic nigrostriatal neurons gives rise to hyperactivity in the output nuclei, the globus pallidus internus (GPi), and the substantia nigra pars reticulata (SNpr), which results in the cardinal symptoms of PD. In order to directly test this theory, the nonselective D1, D2 dopamine receptor agonist apomorphine (APO, 30–100 µg/kg) was administered subcutaneously in 14 PD patients who were off medication (OFF state) while recording neurons in the GP.

For 15 neurons, it was possible to continuously monitor the firing rate as the effect of APO was observed. The average firing rates of many different neurons were determined from single unit histograms before (n = 312) and after (n = 196) injection of the drug. The average (± SD) firing rates before APO were: GPe, 45 ± 15 Hz (n = 85); GPi,e, 67 ± 14 Hz (n = 125); and GPi,i, 85 ± 19 Hz (n = 75). At 25 to 35 minutes after APO, the mean firing rate of GPe neurons had increased to 72 ± 18 Hz (n = 7), the rate of GPi,e neurons had decreased to 39 ± 15 Hz (n = 15), and in GPi,i the mean rate decreased to 34 ± 22 Hz (n = 18). After about 80 minutes post-APO the mean firing rates returned to values similar to those determined before APO.
This study supports current models of basal ganglion dysfunction in PD, which propose that GPe neurons are hypoactive and the output of GPi is excessive. The therapeutic effect of APO appears to result from a normalization of the imbalance of neuronal activity in the direct and indirect pathways of the basal ganglia.

PAPER #829 TUESDAY, 5:15-5:30 PM
A New Animal Model of Spasticity

Lynn Fitzgerald
Richard Simpson (Houston, TX)

KEY WORDS: spasticity, animal model, spinal cord, baclofen

Spasticity is a velocity-dependent increase in muscle tone combined with exaggerated tendon jerks. Most studies of spasticity are performed in paraplegic animals, which are difficult to maintain. Animal spasticity is usually measured with electromyography, which requires anesthesia. Our goal was to produce spasticity with less disability and quantify spasticity in an awake animal. To measure spasticity, the animal’s hindlimb rests on a transducer, and the limb is passively flexed. The difference in the force exerted by the limb in extension and flexion is measured. Preoperative baseline values (n = 10) are obtained on both legs (102 ± 2.2 g vs 103 ± 2.1 g).

To produce spasticity, T10-11 laminectomy and spinal cord hemisection is performed in anesthetized rats. The animals (n = 10) initially show ipsilateral (ipsi) hindlimb hypotonia (Day 1: ipsi 61 ± 7.2 g, contra 91 ± 6.0 g). Motor function recovers after 1 to 4 days. Ipsilateral spasticity develops gradually (Day 7: ipsi 119 ± 8.0 g, contra 85 ± 4.9 g) and plateaus after 13 days (Day 13: ipsi 159 ± 9.9 g, contra 102 ± 7.8 g, p < 0.05; Day 21: ipsi 178 ± 11 g, contra 100 ± 2.9 g p < 0.05).

Intrathecal catheters are placed in spastic animals. Intrathecal saline (20 µl) has no effect on limb tone. Baclofen (2 µg, n = 6) produces a significant decrease in the tone of the spastic limb (pre 155 ± 17 g, post 78 ± 9.0 g, p < 0.05) and a smaller decrease in tone of the contralateral limb (pre 79 ± 9.2 g, post 66 ± 8.3 g, p < 0.01).

Spinal cord hemisection in the rat allows preservation of bladder function, recovery of motor function, and a control (contralateral) leg. Measurement with a force transducer during passive flexion allows quantification of spasticity. The measured decrease in tone after intrathecal baclofen suggests that the model mimics human spasticity.
Wednesday, April 29, 1998

BREAKFAST SEMINARS
6:45 AM – 9:30 AM

Breakfast—Grand Hall
6:45 AM – 7:30 AM

Seminars
7:30 AM – 9:30 AM

#301 ANTERIOR CIRCULATION ANEURYSMS
Room 111B

Moderator:
Steven Giannotta, MD (Los Angeles, CA)*

Panelists:
H. Hunt Batjer, MD (Chicago, IL)
Arthur L. Day, MD (Gainesville, FL)
Jacques J. Morcos, MD (Miami, FL)
Takanori Fukushima, MD (Pittsburgh, PA)

#302 HOW I DO IT: HIGH RISK CAROTID PATIENTS
Room 202B

Moderator:
Donald O. Quest, MD (New York, NY)

Panelists:
Christopher Loftus, MD (Iowa City, IA)*
L.N. Hopkins, MD (Buffalo, NY)*
Issam Awad, MD (New Haven, CT)
Robert Spetzler, MD (Phoenix, AZ)
#303 PERIOPERATIVE MANAGEMENT OF SUBARACHNOID HEMORRHAGE
Room 202A

Moderator:
Ralph Dacey, MD (Saint Louis, MO)

Panelists:
Neil Martin, MD (Los Angeles, CA)
Neal Kassell, MD (Charlottesville, VA)
Christopher Ogilvy, MD (Boston, MA)
Philip Stieg, MD (Boston, MA)

#304 CONTEMPORARY PERCUTANEOUS TECHNIQUES FOR TRIGEMINAL NEURALGIA
Room 105A

Moderator:
Jeffrey Brown, MD (Toledo, OH)

Panelists:
Ronald Apfelbaum, MD (Salt Lake City, UT)
Douglas Kondziolka, MD (Pittsburgh, PA)
John (Sean) Mullan, MD (Chicago, IL)*
G. Robert Nugent, MD (Morgantown, WV)

#305 THE TETHERED CORD: CURRENT MANAGEMENT
Room 110B

Moderator:
David Gordon McLone, MD (Chicago, IL)

Panelists:
Dachling Pang, MD (Sacramento, CA)
Marion L. Walker, MD (Salt Lake City, UT)
W. Jerry Oakes, MD (Birmingham, AL)
Mark Dias, MD (Buffalo, NY)

#306 PEDIATRIC VASCULAR DISORDERS
Room 110A

Moderator:
Michael Edwards, MD (San Francisco, CA)

Panelists:
Michael Levy, MD (Los Angeles, CA)
R. Michael Scott, MD (Boston, MA)
Charles Teo, MD (Little Rock, AR)
Roger Hudgins, MD (Atlanta, GA)
FUNCTIONAL MAPPING OF CEREBRAL CORTEX
Room 112A

Moderator:
Charles J. Hodge, Jr., MD (Syracuse, NY)

Panelists:
George Ojemann, MD (Seattle, WA)
Mitchel Berger, MD (San Francisco, CA)*
Frederick Lenz, MD (Baltimore, MD)
Evandro DeOliveira, MD (Sao Paulo, Brazil)

MANAGEMENT OF VASCULAR MALFORMATIONS
Room 104B

Moderator:
Edward Oldfield, MD (Bethesda, MD)

Panelists:
Daniel Barrow, MD (Atlanta, GA)
Werner Hassler, MD (Duisburg, Germany)
Alex Berenstein, MD (New York, NY)
Joshua Bederson, MD (New York, NY)

SURGERY ON THE BRACHIAL PLEXUS:
INDICATIONS AND TECHNIQUES
Room 104A

Moderator:
Alan Hudson, MD (Toronto, ON)

Panelists:
David Kline, MD (New Orleans, LA)
Susan Tindall, MD (Atlanta, GA)

STEREOTACTIC SPINAL TECHNIQUES
Room 204B

Moderator:
Kevin T. Foley, MD (Memphis, TN)*

Panelists:
Iain Kalfas, MD (Cleveland, OH)*
Allan Hamilton, MD (Tucson, AZ)*
Mario Brock, MD (Berlin, Germany)
Stephen Papadopoulos, MD (Ann Arbor, MI)*
#311 MANAGEMENT OF RHEUMATOID AND CHRONIC INSTABILITY OF THE CRANIOVERTEBRAL JUNCTION AND CERVICAL SPINE
Room 203B

Moderator:
Volker K.H. Sonntag, MD (Phoenix, AZ)*

Panelists:
Curtis Dickman, MD (Phoenix, AZ)*
Richard G. Fessler, MD (Gainesville, FL)
Vincent C. Traynelis, MD (Iowa City, IA)*
Arnold Menezes, MD (Iowa City, IA)

#312 SURGERY OF THE ANTERIOR THORACIC AND LUMBAR SPINE: INDICATIONS AND CURRENT TECHNIQUES
Room 105B

Moderator:
Paul McCormick, MD (New York, NY)

Panelists:
R. Patrick Jacob, MD (Gainesville, FL)
J. Patrick Johnson, MD (Los Angeles, CA)*
Eric Woodard, MD (Boston, MA)
Narayan Sundaresan, MD (New York, NY)*

#313 INDICATIONS AND TECHNIQUES OF LUMBAR INTERBODY FUSION
Room 107B

Moderator:
Peter Klara, MD (Norfolk, VA)

Panelists:
Charles Ray, MD (Norfolk, VA)*
David Cahill, MD (Tampa, FL)
Charles Branch, Jr., MD (Winston-Salem, NC)*
Kent Grewe, MD (Portland, OR)
#314 CURRENT MANAGEMENT OF CEREBRAL TRAUMA
Room 112B

Moderator:
Harold F. Young, MD (Richmond, VA)

Panelists:
John Peter Gruen, MD (Los Angeles, CA)
Michael J. Rosner, MD (Birmingham, AL)
Kevin Gibbons, MD (Buffalo, NY)
Lawrence Marshall, MD (San Diego, CA)*

#315 GENE THERAPY OF CNS NEOPLASMS
Room 102A/B

Moderator:
Mark Rosenblum, MD (Detroit, MI)

Panelists:
Corey Raffel, MD (Rochester, MN)
Mitchel Berger, MD (San Francisco, CA)*
Michael L.J. Apuzzo, MD (Los Angeles, CA)
Zvi Ram, MD (Tel-Hashomer, Israel)*

#316 AVOIDANCE AND MANAGEMENT OF CRANIAL NERVE INJURY
Room 109A

Moderator:
Jon Robertson, MD (Memphis, TN)

Panelists:
Madjid Samii, MD (Hannover, Germany)
Thomas Lovely, MD (Pittsburgh, PA)
Donald Wright, MD (Washington, DC)
Bruce Pollock, MD (Pittsburgh, PA)

#317 NEUROSURGERY://ON-CALL® (COMPUTER DEMONSTRATION)
Room 203A

Moderator:
John Oro, MD (Columbia, MO)*

Panelists:
John McDonald, MD (Atlanta, GA)
John Popp, MD (Albany, NY)
Allison Casey (Park Ridge, IL)
Sidney Tolchin, MD (La Mesa, CA)
**#318 INTRACRANIAL ENDOSCOPY**

Room 204C

**Moderator:**
Alan Cohen, MD (Cleveland, OH)

**Panelists:**
Kim Manwaring, MD (Phoenix, AZ)
Hae Dong Jho, MD (Pittsburgh, PA)*
Robert Goodman, MD (New York, NY)
Axel Perneczky, MD (Mainz, Germany)

**#319 PREPARATION OF A SCIENTIFIC MANUSCRIPT FOR SUBMISSION TO THE JOURNAL OF NEUROSURGERY AND NEUROSURGICAL FOCUS: AN UPDATE**

Room 111A

**Moderator:**
Martin H. Weiss, MD (Los Angeles, CA)

**Panelists:**
Howard M. Eisenberg, MD (Baltimore, MD)
Julian T. Hoff, MD (Ann Arbor, MI)
Melba C. Christy, MD (Charlottesville, VA)
J. Keller Kaufman-Fox, MD (Charlottesville, VA)

**#320 CONSULTANTS CORNER: TUMOR**

Room 201C

**Moderator:**
William Chandler, MD (Ann Arbor, MI)

**Panelists:**
Rudolf Fahlbusch, MD (Erlangen, Germany)
Christer Lindquist, MD (Stockholm, Sweden)
Keith Black, MD (Los Angeles, CA)
Laligam Sekhar, MD (Washington, DC)

**#350 3-D VIDEO SEMINAR—ANTERIOR CIRCULATION ANEURYSMS**

Room 204A

**Moderator:**
Ken Smith, MD (Saint Louis, MO)

**Demonstrating Surgeon:**
Albert Rhoton, MD (Gainesville, FL)*
Wednesday Morning

**Poster Program**

9:00 AM – 4:30 PM

Exhibit Hall A/B/C

479 posters have been chosen for presentation. All will be on display Monday through Wednesday. Presentations will be given between 1:45 PM and 2:45 PM on Wednesday, April 29, 1998, for uninterrupted viewing.

(See Poster Program for complete listing of papers.)
Wednesday Morning

**SCIENTIFIC SESSION V**

9:45 AM – 11:15 AM (Room 103A/B/C)

**Moderators:** Roberto C. Heros, MD (Miami, FL)  
A. John Popp, MD (Albany, NY)

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**PAPER #752 WEDNESDAY, 9:45-10:00 AM**

Cauda Equina Syndrome Secondary to Lumbar Disc Herniation

Scott A. Shapiro  
Thomas B. Scully  
William E. Snyder  
Richard Chua  
David Fritz (Indianapolis, IN)

Discussant: W. Ben Blackett

**KEY WORDS:** cauda equina syndrome, intervertebral disc

Cauda equina syndrome from lumbar disc herniation occurs in 1% of all disc herniations. The majority of the literature supports surgery within 24 hours as improving the outcome. Medical-legal implications are significant with little precise knowledge. Therefore, an analysis of 44 cases of cauda equina syndrome was made both for neurological outcome and medical-legal outcome. This is a retrospective chi-square analysis of 44 cases operated on for lumbar disc herniation patients presenting with cauda equina syndrome.

Twenty cases were diagnosed and operated on within 48 hours of onset of the cauda equina syndrome, including 17 (85%) with surgery within 12 hours. The other 24 cases were operated on at least 48 hours after the onset of cauda equina syndrome with a mean delay of 9 days including 17 (71%) delayed by a mean of 3.7 days. Causes for delay were patient related in 4 (17%) and physician related in 20 (83%). Outcome for the early-surgery group was as follows: motor recovery went from 0-1/5 to 4/5 within a few days, followed by slower recovery to normal or near normal by 6 months in 100%. Bladder/continence recovery was rapid and complete by 6 months in 19 (95%) of these 20 patients; chronic severe sciatica was present in 3 (15%). There was no litigation in this group. Delayed surgery produced the following outcomes: 14 (60%) had persistent motor deficits of 2/5 or worse at 1 year with 2 wheelchair bound; 15 (62%) required chronic or intermittent bladder catheterization at 1 year with 9 (38%) having significant urinary/rectal sphincter incontinence. Chronic severe sciatica occurred in 17 (71%) with all 24 having some complaint of pain. All physician-imposed delay patients filed a lawsuit that was either settled out of court or was successful. Chi-square analysis showed a greater chance of persistent bladder/sphincter problem (p = 0.008), persistent severe motor deficit (p = 0.006), and persistent pain (p = 0.025) with delayed surgery. The chance of a successful lawsuit was greater with delayed surgery (p = 0.001).

The data strongly support treating this problem as a diagnostic and surgical emergency. The patient will successfully sue the physician/surgeon who does not treat this as a diagnostic and surgical emergency.
Modern neurosurgery has long had a strong laboratory foundation and much of this tradition can be traced to the Hunterian Neurosurgical Laboratory at the Johns Hopkins Hospital. Founded with the basic tenet “to investigate the causes and symptoms of disease” and establish the crucial role that the surgeon “may play, particularly in physiologic research,” the Hunterian Laboratory has adhered to this ideal despite the dramatic changes in neurosurgery over the last 100 years.

Named for the famous English surgeon John Hunter (1728–1793), the Hunterian Laboratory was conceived by William Welch and William Halsted as a special laboratory for experimental work in surgery and pathology. In 1904, Harvey Cushing was appointed by Halsted to head the lab. With the three primary goals of student education, veterinary surgery that stressed surgical technique, and meticulous operative and laboratory record keeping, the lab was quite productive, introducing the use of physiological saline, describing the anatomy and function of the pituitary gland, and establishing the field of endocrinology. In 1912, Cushing was succeeded by Walter Dandy whose work on experimental hydrocephalus and CSF circulation led to the development of pneumoencephalography. Just as the early days of neurosurgery evolved with close ties to general surgery, so did the Hunterian lab. In 1922, Ferdinand Lee, a general surgeon, became head of the lab. Between 1928 and 1942, over 150 original papers describing significant advances in surgery emanated from the Hunterian Laboratory including seminal preclinical work by Alfred Blalock and Vivian Thomas that led to the famous “blue baby” operation in 1944. With the introduction of the operating microscope in the 1950s, much of the focus in neurosurgical science shifted from the lab to the operating room. The old Hunterian building was demolished in 1956.

In 1987, the Hunterian Laboratory for surgical and pathological research was rebuilt on its original site and, in 1991, the Hunterian Neurosurgical Laboratory was reestablished, with a focus on novel treatments for brain tumors. The strong tradition of performing basic research with clinical relevance has continued.

Myelopathy secondary to disc disease and cervical spondylosis is a significantly disabling, sometimes life-threatening disease. The impact of medical and surgical treatment on the natural history of cervical myelopathy is poorly understood. The Cervical Spine Research Society (CSRS) Study is a multicenter study of a large heterogeneous group of patients who were referred for the evaluation and treatment of subacute or chronic recognized cervical spine pain problems. In this study, we focus on patients who have myelopathy as their predominant clinical presentation. The goals of the study are to characterize these patients, assess their outcome with respect to the treat-
ment received, treatment prescribed, and expected outcome, identify the most effective forms of treatment, and examine factors that influence outcome.

A total of 503 patients were enrolled in the study. Of these, 62 (12%) presented with myelopathy as the predominant complaint. Forty-one CSRS surgeons participated in the study. Each study patient completed a CSRS questionnaire on initial presentation and at follow-up appointment after receiving a treatment. The physician then completed a form. Statistical analysis was performed by a blinded third party using the SAS program.

Of 62 patients with myelopathy, 43 (69%) completed both questionnaires. The prescribed treatment was medical treatment for 15 patients (35%), surgical treatment for 22 (51%), and no treatment for 6 (14%). In actuality, 19 (44%) received medical treatment and 24 (55%) received surgical treatment. Overall, patients who were prescribed surgical treatment and received it fared best, with 19 (79%) reporting improvement in symptoms. Complete data on medical and surgical treatments, pain scales, social activity, work status, litigation, workers’ compensation, and patient satisfaction will be presented. Factors that influence outcomes in all patients will be discussed in detail. Surgery appears to be an effective means of therapy for most patients with cervical myelopathy. This is the first prospective, multisurgeon study that examines this important issue and correlates with present clinical opinion.

PAPER #755  WEDNESDAY, 10:30-10:45 AM
Do Outcomes After Radiosurgery Differ for Different Metastatic Brain Tumors?
Katrina Firlik
Yoshimasa Mori
Young Soo Kim
Douglas Kondziolka
John Flickinger
Ann Maitz
L. Dade Lunsford (Pittsburgh, PA)
Discussant: Raymond Sawaya

KEY WORDS: radiosurgery, metastasis

The goal was to evaluate differences in survival and tumor control rates for different metastatic brain tumors treated with the gamma knife. Patients who had undergone gamma knife stereotactic radiosurgery for the following tumor types metastatic to brain were included: breast, renal cell, non-small cell lung, and malignant melanoma. Patient records were retrospectively reviewed. Median survival after gamma knife radiosurgery was analyzed using the Kaplan-Meier method. For tumors with follow-up imaging, tumor control rates were obtained.

A total of 202 consecutive patients (343 tumors) were treated. Thirty patients had breast cancer, 35 had renal cell cancer, 77 had lung cancer, and 60 had malignant melanoma. Median survival was: 15 months for breast cancer, 11 months for renal cell, 10 months for lung, and 7 months for malignant melanoma. Local tumor control rates were: 86% for breast (41 tumors imaged), 90% for renal cell (39 tumors imaged), 85% for lung (91 tumors imaged), and 90% for malignant melanoma (72 tumors imaged). Specific tumor responses (gone, decreased, unchanged, or increased) were, respectively: breast (19%, 36%, 31%, 14%), renal cell (21%, 44%, 26%, 10%), lung (17%, 45%, 25%, 13%), and malignant melanoma (11%, 44%, 35%, 10%).

Although the tumor control rates for the four tumor types were similar, survival was longer for patients with breast metastases and shorter for patients with malignant melanoma metastases. Radiosurgery provides an effective tumor response for common brain metastases with survival limited by the efficacy of systemic management.
Variations of the Extreme-Lateral Approach: Anatomical Study and Clinical Analysis of 50 Patients

Eduardo Salas
Laligam N. Sekhar
Ibrahim M. Ziyal
Donald C. Wright (Washington, DC)

Discussant: Takanori Fukushima

Key Words: surgical approach, anatomical study, clivus, atlanto-occipital junction

The extreme-lateral approach is performed for various lesions of the lower clivus and the craniocervical junction. Several variations of this approach are described according to the nature and location of the targeted lesion. We analyzed our experience to describe the variations of the extreme-lateral approach and their specific indications.

Fifty patients underwent an extreme-lateral approach between June, 1993, and June, 1997. The indications were: 1) transfacetal approach for lesions below the atlanto-occipital joint (3 patients); 2) retrocondylar approach for laterally placed lesions above the atlanto-occipital joint (7 patients); 3) partial transcondylar approach for midline intradural lesions located above the atlanto-occipital joint (17 patients, mostly meningiomas); 4) transcondylar approach for extradural lesions involving the occipital condyle (12 patients, mostly chordomas and chondrosarcomas); 5) transjugular approach for extensive lesions involving the jugular foramen (8 patients, including glomus jugulare tumors, lower cranial nerve schwannomas, and meningiomas); and 6) transtubercular approach (jugular tubercle) for giant VA aneurysms (3 patients). We performed a bilateral extreme-lateral approach in all its variations in 6 adult specimens and correlated the anatomical findings with the clinical experience.

Occipitocervical fusion was required in 2 patients with transfacetal approach (Group 1), in 10 patients with transcondylar approach (Group 4), and in 1 patient with a partial transcondylar approach (Group 3) in whom the instability was not related to the partial condylectomy. Nine patients suffered CSF leakage. They were mostly related to recurrent tumors. In 6 of these patients the leak resolved with lumbar drainage. The other complications were extraxial hematoma (2 patients) and hydrocephalus (1 patient). The cranial nerves most frequently involved were the 9th, 10th, 11th, and 12th. The 9th, 10th and 11th cranial nerves improved in 3 patients because of the decompression, remained unchanged in 11, and worsened in 1. Two patients presented a new 12th nerve deficit and in 19 it remained unchanged. The mean Karnofsky Performance Score preoperatively was 77.6 (range 100–40) and postoperatively 72.7 (range 100–40). Total removal of the lesion was performed in 37 patients and subtotal in 10. In 3 patients with aneurysms trapping or clipping was achieved.

In lesions of the atlanto-occipital junction and lower clivus, the appropriate variation of the extreme-lateral approach should be chosen according to the exact location and nature of the pathology. That choice helps to achieve a complete removal with minimal morbidity and complications.
The open configuration intraoperative MR imager (GE Medical Systems) is a revolutionary system that allows surgeons to perform procedures under direct MR guidance. It allows real-time monitoring of parenchymal changes during tumor resection as well as accurate localization of margins and surrounding structures. Seventy patients underwent a craniotomy for tumor in the intraoperative MRI at the Brigham and Woman’s Hospital (Boston, MA). Patients with intracranial tumors difficult to target or resect due to their location near eloquent cortex or the deep white matter, or in whom previous surgery had not achieved full resection, were selected. Twenty patients who had tumors in or near motor/speech cortex underwent an awake resection. Thirty-five low-grade gliomas, 25 high-grade gliomas, and 10 metastatic lesions were resected. The intraoperative MRI is a major development in the surgical management of brain tumors, providing real-time radiographic visualization during the procedure. This eliminates the errors that can occur when relying on preoperatively obtained images which cannot accommodate for intraoperative changes in the anatomical position of neural structures and lesions.

Advantages of this system include: 1) absolutely accurate localization; 2) ability to establish motor and sensory areas structurally; 3) adaptation to intraoperative structural changes; 4) verification of complete resection; 5) immediate recognition of intraoperative complications such as hemorrhage/clot formation during closure.
Wednesday Morning

Scientific Session VI

9:45 AM – 11:15 AM     (Room 108A/B)

Moderators: R. Michael Scott, MD (Boston, MA)
             Robert B. Page, MD (Hershey, PA)

PAPER #758 WEDNESDAY, 9:45-10:00 AM

Outcome of 51 Consecutive Cases of Unilateral Locked Cervical Facets

Scott A. Shapiro
Richard Chua
William Snyder
David Fritz (Indianapolis, IN)

Discussant: Volker K.H. Sonntag

Key Words: locked facet, radiculopathy, closed reduction, outcome

An analysis of 51 consecutive cases with unilateral locked cervical facets treated between 1986 and 1996 was performed. Of these patients, 37 (73%) presented with radiculopathy, 9 (18%) with neck pain only, and 5 (10%) with cord injuries. Plain films showed subluxation only in 44 (86%). All patients underwent cervical CT and all with cord injuries had MRI. Fracture in addition to facet locking was seen in 24 (47%). Disc disruption with cord compression was seen in 5 (10%).

Based on CT, closed reduction was believed to be contraindicated in 11 (22%); of the remaining 40, 13 (33%) were closed reduced. Two patients with closed reductions were placed in a halo but resubluxed. Thus, all patients underwent surgery. Forty-six underwent posterior reduction and/or internal fixation alone (24 spinous process wire fixation and facet wiring to struts of iliac crest and 22 lateral mass plating and interspinous braided cable). Initial surgery regardless of technique was successful in 45 cases (98%). One case resubluxed and was reoperated with anterior cervical fusion/plating. The 5 cord-injured patients underwent emergency combined anterior/posterior decompression and internal fixation. Overall, there was no neurological worsening or mortality and three wound infections. At 1 year, all deficits had improved. Of 37 radiculopathies, 3 (8%) had persistent 4/5 weakness and the rest were normal including 4 delayed diagnosis cases. All 5 cord injuries improved immediately after surgery. Persistent neck pain was seen in 9 cases (17%). Though the lateral mass plates/interspinous cable are stronger and easier, chi-square analysis demonstrated no difference in outcome as compared to wire and iliac crest.

Cervical CT/MRI provides information aiding diagnosis and management. Our experience strongly suggests that reduction and internal fixation/bone fusion will be most successful for this injury.
A wide variety of surgical adjuvants to the standard bone decompression have been advocated in the treatment of the Chiari I malformation, especially when the tonsillar herniation is associated with hydrosyringomyelia. These include various shunting procedures, duroplasty, obex plugging, and resection of the cerebellar tonsils. Our practice has been to avoid these adjuvants and to perform a simple occipital craniectomy, C-1 laminectomy, and dural opening. The dura mater is left open and overlaid with oxidized cellulose. To evaluate the efficacy of this more limited procedure, a retrospective review was performed of the medical records of 31 consecutive patients treated over a 6-year period. Twenty-six (84%) of these patients had an associated spinal cord syrinx; all underwent the same procedure.

Follow up ranged from 6 to 72 months, with all patients having at least one postoperative MRI. Twenty-three (88.5%) of the 26 patients who presented with a syrinx had significant resolution of the syrinx on follow-up images with concomitant improvement of presenting signs and symptoms. Of the remaining 3 patients, 1 had progressive hydrocephalus and received a ventriculoperitoneal shunt, with symptom resolution. In the other 2 patients the syrinx did not diminish, although they remained clinically stable; one received a syringopleural shunt, and the other has been followed for only 6 months without change. Postoperative morbidity includes a 26% incidence of headaches, of which half resolved within 5 days and only one persisted beyond 2 weeks. Nausea and vomiting occurred in 16%. Neither of these figures significantly exceeds those of other large surgical series in which the dura mater was closed with a patch graft. Three patients (9.7%) did have a postoperative CSF leak; all responded to bedside suturing without further sequelae.

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

The surgical outcome for adults (>18 years old) who present with the tethered cord syndrome is generally believed to be worse than that of children. We report the results after microsurgical untethering in adults. From 1993 to 1997, there were 21 patients ranging from 20 to 58 years of age who presented for the first time with symptoms of the tethered cord syndrome. There were 15 females and 6 males. The most common presenting complaints were progressive sensorimotor dysfunction (84%), back and lower-extremity pain (80%), and bladder dysfunction (63%). All patients were evaluated with total spine MRI, plain spine radiography, and urodynamic testing when indicated. Surgical treatment consisted
of an aggressive microsurgical untethering and repair of the congenital defect. Intraoperative EMG monitoring was used as a surgical adjunct in 19 cases.

At follow up (1 month-3 years), all 21 patients showed improvement in their symptoms. The most significant improvements were in bladder function (90%) and motor strength (75%). Most patients (58%) had improvement in lower-extremity pain, but only 20% showed improvement in their back pain. All 19 patients so tested demonstrated improvement of the intraoperative EMG signals during surgery. Complications included two superficial wound infections and one patient with a temporary monoparesis. No patient died or exhibited long-term neurological deficits as a result of surgery. We conclude that aggressive microsurgical treatment of the tethered cord syndrome in adults can result in excellent to good outcomes in most patients.

PAPER #761 WEDNESDAY, 10:30-10:45 AM
Motor Evoked Potential Monitoring for Spinal Cord Tumor Surgery
Karl Kothbauer
Vedran Deletis
Fred J. Epstein (New York, NY)
Discussant: David Cooper

KEY WORDS: spinal cord tumor, evoked potential, intraoperative monitoring

The safety, feasibility, and clinical correlation of intraoperative motor (MEP) and sensory (SEP) evoked potential monitoring was retrospectively analyzed in 88 operations of intramedullary (n = 77) and extramedullary (n = 11) spinal cord tumors. The MEPs elicited by transcranial electrical stimulation were recorded epidurally and from limb muscles. Before surgery, 79 patients (89.8%) had normal motor status and 9 (10.2%) were severely impaired. Epidural MEPs could be monitored in 61.8% of the patients without severe deficits, muscle MEPs in 93.7%, and SEPs in 57%. Epidural MEPs were absent in all severely impaired patients. Muscle MEPs were absent in every plegic extremity. The SEPs were recordable in 4 of 9 severely paretic patients. Intraoperatively, MEPs, mostly recorded without averaging, provided instant feedback on the functional integrity of the motor system. No complications of transcranial stimulation and MEP recording occurred.

Postoperatively, 22 patients (25%) had new motor deficits. The sensitivity of combined epidural and muscle MEP monitoring for these was 100%, the specificity 80% (no false negatives). The sensitivity of SEPs, if recordable, was 88%, their specificity 41% (one false negative). Gross-total resection was achieved in 67% of the procedures, compared to 48.5% in a pre-MEP series (n = 72). We found that intraoperative monitoring of spinal cord surgery with epidural and muscle MEPs is safe, feasible, and superior to monitoring with SEPs. It is highly sensitive to intraoperative damage of the motor system. It provides rapid feedback to the surgeon. Apparently it has aided in resecting spinal cord lesions more radically.

PAPER #762 WEDNESDAY, 10:45-11:00 AM
Clinical Outcome of Patients With Subarachnoid Hemorrhage With Vasospasm Is the Same as in Patients Without Vasospasm Using Aggressive ICU Management
Christopher Ogilvy
Oscar Szentirmai
Deidre Buckley
Nicholas Zervas (Boston, MA)
Discussant: Neil Kassell

KEY WORDS: subarachnoid hemorrhage, vasospasm, outcome

Following treatment of an intracranial aneurysm after SAH, there is potential for significant morbidity and mortality as a result of cerebral vasos-
We reviewed 411 patients with SAH admitted to Massachusetts General Hospital between 1992 and 1997 and compared outcome in patients with and without clinical vasospasm. The patients' clinical conditions at time of treatment were as follows: Hunt and Hess (HH) Grade 1, 120 patients (30%); HH Grade 2, 38 patients (9.5%); HH Grade 3, 147 patients (37%); HH Grade 4, 73 patients (18%); and HH Grade 5, 22 patients (5.5%). Patients treated ranged in age from 7 to 95 years. The majority of aneurysms were obliterated within 24 to 48 hours of initial ictus. Within the total group of patients, clinical vasospasm developed in 177 patients. Vasospasm was managed with hypertensive, hemodilutional, and hypervolemic therapy in an ICU setting, with endovascular treatment used in 39 patients. Outcome was evaluated from 3 months to 5 years after treatment (average follow up of 2.2 years).

Outcome was assigned as Excellent: normal neurological function; Good: slight neurological deficit with return to work; Fair: unable to return to previous level of employment; Poor: full-time nursing care. Of the group of 177 patients with vasospasm, 128 (72%) had excellent or good outcome, 19 (11%) had fair outcome, and 6 (3%) had poor outcome, with 24 deaths (14%). In 234 patients without spasm, 181 (77%) had an excellent or good outcome, 8 (3%) fair, 4 (2%) poor, and 44 (19%) died. There was no significant difference in outcome between the two groups. Therefore, patients with vasospasm managed aggressively in a neurological ICU do as well as patients without vasospasm after SAH.

Intraoperative Ultrasound Localization of Pituitary Adenomas in Patients With Cushing’s Disease and Negative Pituitary MR Imaging

Joe Watson
Thomas Shawtier
John Doppman
Edward Oldfield (Bethesda, MD)

Discussant: William F. Chandler

Key Words: pituitary adenoma, Cushing’s disease, ultrasound

Pituitary surgery produces remission of Cushing’s disease (CD) with preservation of pituitary function in only 60 to 70% of patients. The inability to identify a small adenoma accounts for most of the failed sellar explorations. Most negative explorations are in patients with a negative MRI, which occurs in about 35 to 45% of patients with CD. The usefulness of intraoperative ultrasound (IOUS) for identifying an adenoma in patients with a negative MRI is unknown. We reviewed the IOUS findings of 67 consecutive patients with CD and a negative (n = 58) or equivocal (n = 9) MRI. Intraoperative ultrasound localized a tumor in 46 (73%) of 63 patients with surgically confirmed adenomas. The size of adenomas detected with IOUS versus those not detected (6.8 ± 3.4 mm vs 6.1 ± 2.8 mm [mean ± SD]) did not differ (p = 0.5). Intraoperative ultrasound revealed 21 tumors of < 5 mm, including 4 tumors of 3 mm or less, the smallest being 1.5 mm. Intraoperative ultrasound was useful in pediatric patients (n = 6) and in patients with previous pituitary surgery (n = 11). In 4 patients, no adenoma was found at surgery or in the pathological specimen (true negative). In 9 patients, abnormalities suspicious for an adenoma detected by IOUS were negative on exploration (false positive). Intraoperative ultrasound is a sensitive (73%) and specific (84%) method for detecting pituitary adenomas in patients with CD and negative MRI. Of the 67 patients with a negative MRI (90%), 60 had a surgical cure of CD. Improved ability to detect and localize these small tumors intraoperatively appears to have a positive effect on the surgical outcome in these patients.
PAPER #764 WEDNESDAY, 9:45-10:00 AM

The Fiberoptic Intraparenchymal Cerebral Pressure Monitor in 420 Patients

Scott A. Shapiro
Richard Chua
William Snyder
David Fritz (Indianapolis, IN)

Discussant: Jack Wilberger

KEY WORDS: intraparenchymal pressure, fiberoptic monitor

The fiberoptic intraparenchymal cerebral pressure (ICP) monitor has been shown to provide reliable data in patients monitored for a short period of time. An analysis of a large population including patients monitored for a prolonged period such as weeks has never been performed. We conducted a retrospective study in which we evaluated the fiberoptic ICP monitor for complications and accuracy.

The fiberoptic ICP monitor has been used in 420 consecutive patients since 1988. Pathology included trauma in 294, intracerebral hemorrhage (ICH) in 31, aneurysmal SAH in 33, AVM in 19, cerebral edema in 20, tumor in 12, and shunt evaluation in 6. The mean length of monitoring was 6.4 days with 113 (29%) monitored 1 to 2 days. One hundred forty-eight (37%) were monitored for 3 to 6 days, 73 for 7 to 10 days (18%), and 63 (16%) for 11 to 24 days; 122 also had ventriculostomies and there was strong correlation with the ICP monitor (p < 0.001). Complications from insertion were 3 (0.6%) ICHs, all with hepatic dysfunction and 1 acute subdural hemorrhage. In the group monitored > 6 days, 40 (10%) required at least one catheter change due to upward drift of the ICP. Fiberoptic breakage requiring replacement was documented in 51 (13%). An insertion site skin infection occurred in 1 case monitored for 23 days. Two bone flaps with monitor placement became infected. There were no cases of bacterial meningitis or cerebral abscess.

The monitor is easy, safe, and reliable to use with a very low rate of infection. Intracerebral hemorrhage was only a risk in coagulopathic patients.
Degradation of accuracy during frameless stereotactic neuronavigation due to brain or lesion shift in intracerebral rather than in skull base tumors is one of the major drawbacks of image-guided surgery. A mobile CT scanner system (Philips Tomoscan N) developed for the operating room was connected with a pointer device navigation system for image-guided surgery (Philips Easy Guide System). During the last 12 of 120 image-guided procedures, the advantages of this combination were evaluated.

The installation of the scanner into the operating room setup was successfully performed in the study cases of 10 meningiomas (6 cranial base and 4 convexity), 1 craniopharyngioma, and 1 large cerebral glioblastoma. The patients were positioned on a specially adapted scanner table which allowed movement to scanning position and back to the operating position at any time during surgery. The CT scanner remained in a fixed position and the scanner table as operating table was moved with the patient through the gantry. In all cases contrast-enhanced, preoperative CT scans after positioning and draping were of high quality, as a radiolucent head fixation was used. In all 8 meningiomas and the craniopharyngioma patient, the intraoperative scans without and with enhancement demonstrated complete resection. In two meningiomas, intraoperative CT showed a significantly large residual tumor, which was subsequently identified by the connected neuronavigation system and removed.

The combination of an intraoperative CT scanner with the pointer device neuronavigation system permits not only intraoperative resection control of brain tumors but also identification of invisible residual tumor tissue by intraoperative update of the neuronavigation data set. Additionally, image update solves the problem of intraoperative brain or tumors shift during image-guided resection.
resection by postoperative MRI, SRS, BTY) and employed a log-rank comparison. Tests of independence were done by Cox methodology.

There were 180 patients (97 men, 83 women), with an average age of 57.2 years. Pathology was glioblastoma multiforme in 136 and anaplastic astrocytoma in 44. Resection was complete in 15 and subtotal in 83; the balance underwent stereotactic biopsy alone. Eighteen patients had $^{192}$Ir BTY and 20 LINAC SRS (median survival 20 months for both). Based on a univariate log-rank technique, age, extent of resection, pathology, KPS, SRS, and BTY were significantly related to survival. With the Cox multivariate analysis, however, only age, extent of resection, pathology, and SRS remained independently significant, ranked in that order.

These data support the argument that SRS is an effective treatment modality which is independently related to prolonged survival in patients with malignant glioma. Despite an identical median survival, the superior performance of BTY patients appears explained by selection factors.

**PAPER #767 WEDNESDAY, 10:30-10:45 AM**

**Reoperation for Recurrent Acoustic Neuromas**

Gordon Tang (Barstow, CA)
Steven L. Giannotta (Los Angeles, CA)

Discussant: David Andrews

Key Words: acoustic neuroma, internal auditory canal, surgical approach, facial nerve preservation

Acoustic neuromas infrequently recur following appropriate surgical therapy. The increasing frequency of referrals for recurrent acoustic neuromas and consideration for radiosurgical management have prompted us to review our series of reoperations for recurrent acoustic neuromas to identify management strategies, study clinical features, and determine outcome. We reviewed 16 patients with recurrent acoustic neuromas who underwent reoperation. Follow up averaged 6.2 years. Patients with neurofibromatosis-2 were excluded.

There were 5 females and 11 males with a mean age of 46 years. Tumor size at recurrence ranged from 1.5 to 6 cm with a mean of 3.3 cm. All but one patient had some facial nerve compromise at recurrence. Increasing dizziness, unsteadiness, or radiographic recurrence were the most common modes of presentation. All prior resections were through a suboccipital approach. In all but one case, recurrence originated from the lateral internal auditory canal (IAC). Reoperations were performed by the translabyrinthine approach. Despite the increased difficulty of facial nerve dissection, only one patient had worsening of preoperative facial nerve function from House Grade IV to V. Cerebrospinal otorrhea with meningitis was present in one case and two patients had new postoperative ataxia. Total resection was achieved in all cases and, to date, no repeat recurrences have occurred. Early imaging may demonstrate enhancing tissue, falsely suggesting residual tumor.

In experienced hands, the suboccipital approach offers good results for a range of tumors and allows for hearing preservation. However, efforts to preserve the labyrinth may reduce exposure to the lateral one-third to half of the IAC, necessitating blind dissection. Retained tumor in this region appeared to be the most common cause for recurrence in our series. The translabyrinthine approach offers advantages for reoperation of acoustic neuromas by providing fresh tissue planes, exposing the entire IAC, and providing early identification of the facial nerve. Although complication rates appear to be higher, reoperation for recurrent acoustic neuromas can be accomplished with facial nerve preservation and a low repeat recurrence.

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Endovascular Coil Obliteration of Posterior Circulation Cerebral Aneurysms

Jeffrey E. Thomas
Patricia M. Grannon
Robert H. Rosenwasser (Philadelphia, PA)

Discussant: Duke Samson

Key Words: endovascular surgery, detachable coil, aneurysm, posterior circulation

This was a study to evaluate the effectiveness of endovascular coil occlusion as a treatment modality for cerebral aneurysms of the posterior circulation. Nineteen patients presenting with 19 posterior circulation aneurysms underwent endovascular occlusion of their lesions with the Guglielmi detachable coil. Thirteen patients presented with SAH (3 Grade IV, 3 Grade III, 3 Grade II, and 4 Grade I); 6 patients were Grade 0. Indications for endovascular treatment were advanced age, poor medical condition, and difficult surgical anatomy as evidenced by angiogram. All patients presenting with SAH were treated within 48 hours of the ictus. Following coil occlusion of the aneurysm, all patients were treated with volume expansion and systemic heparinization for 48 hours, then dextran for an additional 24 hours.

Of the 19 aneurysms, 17 were successfully obliterated by coiling. Coiling could not be performed in 1 giant basilar apex aneurysm because of a wide orifice and in 1 vertebral-PICA aneurysm because of extremely tortuous arterial anatomy. No intraoperative complications occurred. Procedure-related postoperative complications included 2 cases of transient ischemic attack which was resolved with anticoagulation therapy and 1 case of partial oculomotor paresis related to thrombosis of the aneurysm which responded to steroid treatment. Long-term follow up ranged from 3 to 16 months. No delayed postoperative complications were observed. Follow-up angiography was performed in 15 patients, between 6 and 15 months after coil embolization, and in 14 demonstrated persistent total aneurysm occlusion. One patient demonstrated significant reopening at the base of a giant PCA aneurysm, requiring repeated coiling which resulted in obliteration of the lesion.

Endovascular coil occlusion is an effective treatment alternative for cerebral aneurysms of the posterior circulation. The large majority of properly selected lesions are amenable to coiling, in contrast to aneurysms of the anterior circulation. Vessel tortuosity and wide orifice were associated features of posterior aneurysms which could not be occluded by coil. Systemic heparinization and volume expansion are important adjuncts to endovascular coil occlusion.

Intraoperative Localization of Brain Tumors and Surrounding Functional Cortex With Infrared Imaging

Alexander Gorbach
John Heiss
Conrad Kufta
Edward Oldfield (Bethesda, MD)

Discussant: Michael L.J. Apuzzo

Key Words: infrared imaging, tumor localization, intraoperative monitoring, cortex mapping

On-line functional imaging of brain tumors was examined using intraoperative infrared (IR) imaging. The same technique was also examined for mapping adjacent cortex during appropriate physiological stimulation. An IR camera with sensitivity of 0.02°C at 3 to 5 μm measured temperature distribution over the exposed cortical surface in 11 patients with intrinsic brain tumors. The cam-
era was placed 10 to 30 cm above the exposed brain, and images were displayed on an operating room monitor within 30 seconds of data acquisition.

The regions of all 11 tumors were clearly identified by low temperature (10 gliomas) or high temperature (1 cavernous angioma). In all patients, IR localization was consistent with the results of preoperative MRI, intraoperative ultrasound, and surgical findings. Although the region of tumor involvement could be distinguished clearly from the distant, normal brain, temperature heterogeneity occurred within the borders of individual lesions. Normal and tumor-related surface vessels greater than 0.5 mm in diameter and their dynamic temperature profiles reflecting focal pulsatile perfusion were visible on IR images.

Functional localization of eloquent cortex was performed during median, tibial, and trigeminal nerve stimulation, repetitive motor task performance, and language testing. These stimuli produced reproducible, rapid latency (0.5–7 seconds), topographically restricted temperature gradients (0.04–0.08°C) in the exposed cortex. Activated cortical sites with spatial resolution of up to 150 µm were displayed on-line. The localization of these sites was consistent with localization by intraoperative electrophysiology. Intraoperative IR imaging may enhance the accuracy and safety of tumor resections near eloquent cortex.
Wednesday Morning

Scientific Session VIII

9:45 AM – 11:15 AM  (Room 114)

Moderators: Robert A. Ratcheson, MD (Cleveland, OH)
John A. Kusske, MD (Laguna Hills, CA)

PAPER #770 WEDNESDAY, 9:45-10:00 AM
Role of Vascular Endothelial Growth Factor in the Pathogenesis of Venous Thromboembolic Disease in Patients With Brain Tumors

Gregory Licholai
Patrick Wen
Rona Carroll
Peter McL. Black (Boston, MA)

Discussant: Raymond Sawaya

Key Words: vascular endothelial growth factor, brain tumor, thromboembolism

Venous thromboembolic disease is a common problem in brain tumors patients. Vascular endothelial growth factor (VEGF) is a mitogen secreted by tumor cells which causes angiogenesis and peritumoral edema. We correlated VEGF mRNA expression in meningiomas to the presence of postoperative deep venous thrombosis (DVT). The RNA was isolated from 31 meningioma resections, analyzed by electrophoresis, and hybridized to labeled probes. Blots were autoradiographed and laser band densities recorded in arbitrary densitometry units (ADU). The RNA amount was internally standardized using β-actin. Statistical Wilcoxon rank-sum and Fisher exact tests were used.

Five (16%) of 31 patients with meningiomas experienced postoperative DVTs. The median VEGF RNA for DVT patients was 37,739 ADU (range 33,366–46,145 ADU). For the patients who did not have DVTs the median VEGF was 8650 ADU (range 0–40,131 ADU). This was statistically significant (p < 0.001), and there was also a significant correlation between a frontal location of the tumor (p = 0.005) and the presence of edema (p = 0.048) and the development of a DVT. There was no correlation between the development of DVT and clinical features such as patientís age, sex, KPS, steroid use, postoperative hemiparesis, or laboratory parameters such as PT, PTT, INR, and platelet count.

This study shows a correlation between VEGF mRNA expression in meningiomas and the development of DVTs and suggests that VEGF may contribute to the hypercoagulable state, possibly by increasing von Willebrand factor release from endothelial cells, augmenting platelet aggregation and adherence, enhancing tissue factor activity, or activating the extrinsic pathway of coagulation.
Transplantation of Neural Precursors in Neurodegenerative Diseases

Vivian Tabar (Worcester, MA)*
Lorenz P. Studer
Ronald D. McKay (Bethesda, MD)

Discussant: Zvi Ram

Keywords: neural precursor transplantation, Parkinson’s disease, Huntington’s disease

Neurotransplantation has been recently introduced as an experimental treatment for neurodegenerative diseases. Clinical trials have been limited by the paucity of donor cells and poor graft survival. We present a novel transplantation strategy for Parkinson’s (PD) and Huntington’s (HD) disease. Donor cells are derived from rat embryonic precursor cells obtained from early mesencephalic (VME, E11-12) or striatal (LG, E14-15) tissue, respectively. The cells are expanded as monolayer cultures in defined medium supplemented with 10 ng/ml bFGF over 1 to 3 passages, followed by a 7-day period of differentiation as 3-D macroaggregates (600–1200 µm) for VME or microaggregates (50–300 µm) for LG cultures. Macro- and microspheres develop a high degree of neuronal differentiation after prolonged in vitro periods (2–12 weeks) as evidenced by TuJ1 and MAP2ab immunohistochemistry. BUdR incorporation studies in VME and LG cultures revealed double labeling in 10 to 90% of TH+ and 80 to 95% of GABA+ cells, depending on developmental stage and BUdR exposure. Intrastriatal transplantation of VME aggregates in unilaterally 6-OHDA-lesioned rats resulted in complete recovery of rotational asymmetry in 50% of the animals up to 80 days posttransplantation. Surviving TH+ cells could be detected in all animals (1200 ± 325 cells/graft). Intrastriatal transplantation of LG microaggregates is carried out in rats with unilateral striatal ibotenic acid lesions. Behavioral outcome and graft survival studies are pending.

This work demonstrates the feasibility of utilizing in vitro expanded neural precursors as reconstructive tools in the CNS. The capacity of stem cells for ordered self-renewal, multipotentiality, and plasticity in response to local cues and growth factors might further widen the horizon of neural transplantation.

Myofascial Closure for Myelomeningocele: A 20-Year Experience

E. Christopher Troup (Macon, GA)
Robert Sanford
Michael Muhlbauer
Angela Steinman
Lela Ostby (Memphis, TN)

Discussant: Mark Dias

Keywords: myelomeningocele, myofascial flap, ventriculomegaly

Closure of myelomeningocele in the newborn is a common pediatric neurosurgical procedure involving reconstruction of the neural tube, dural closure, and fascial and skin closure. Most of the different strategies in the literature deal with skin closure. A myofascial closure developed by the senior author was presented to the Pediatric Section in 1980 following the first 84 cases. This report is a retrospective series of 160 consecutive children whose defects were closed utilizing a myofascial flap.

Five infants developed CSF leak requiring reoperation. cerebrospinal fluid leakage has been reported as high as 29% in some series. Because CSF leak in our series was low and not dependent on timing of shunting, we were able to document progressive ventriculomegaly prior to shunting. Shunting
was often delayed days to weeks after primary closure, with a shunt infection rate no higher than in our non-myelomeningocele hydrocephalic patients (approximately 4.5%). We believe that this management strategy results in fewer infants being shunted and reduced infection. The anatomy, technique of flap development, and surgical repair will be described in detail.

PAPER #773 WEDNESDAY, 10:30-10:45 AM
A Proposed Grading System to Predict Full Resection in Cranial Base Meningiomas

Zachary T. Levine
Russell I. Buchanan
Laligam N. Sekhar
Charles L. Rosen
Donald C. Wright (Washington, DC)

Discussant: Chandranath Sen

KEY WORDS: grading system, meningioma resection, cranial base

We propose a scheme to predict resectability of cranial base meningiomas based on preoperative criteria obtained from history, radiographic data, and patient examination. In addition, we found a significant correlation between full resection (Simpson Grade I/II) and higher postoperative Karnofsky score.

A total of 239 consecutive patients with cranial base meningiomas were operated on during the period 1993 to 1997. Preoperative, hospital course, and postoperative long-term data were tabulated. The cohort included 56 males and 183 females ranging in age from 10 to 83 years (mean 51 years). Statistical analysis of the data included chi-square tests and logistical regression to examine correlation of predictors with outcome. The threshold for significance was p < 0.05. The scoring system to predict tumor resection using preoperative predictors is being developed using logistical regression.

Upon examination of the data acquired from each patient, we found eight variables in three categories (history, physical, radiology) that highly correlated with resectability. The absence of prior treatment (surgery, radiation, or both) correlated well with full resection (p < 0.011). Intact function of the 3rd through 5th cranial nerves independently correlated with full resection (p < 0.000 each). Smaller tumor size as measured on MRI and lack of vessel encasement both correlated with full resection (p < 0.003, p < 0.000 respectively). The fewer fossae (anterior, middle, posterior) involved correlated with increasing resectability (p < 0.000). Tumor resection was also associated with postoperative outcome as measured by postoperative Karnofsky score, length of stay in a critical care unit, and total length of stay in the hospital. This association was investigated by the two-sample t-test, and p value was < 0.050 in each case.

Using prospectively acquired data we were able to construct a grading system to predict resectability based on preoperative criteria: prior treatment, cranial nerve dysfunction, tumor size, vessel encasement, and extent of tumor along the cranial base. In addition, patients with tumors that were fully resected had significantly higher postoperative Karnofsky scores than those patients with partial resection; however, we cannot say that full resection in all skull base meningiomas correlates with better outcome. Our data do not describe a causal relationship between full resection and good outcome. Further research should examine tumor biology and its effect on total resection. The resection grading system will allow comparison of treatment options, comparison between various clinical series, and guide individual surgeons in decision making.
The purpose of this study was to define the overall dimensions of the C1-2 transarticular pathway, assess risk to adjacent visceral and neurovascular structures, and establish linear and angular parameters for safe screw placement. Axial CT scans with 1-mm slice thickness and 14-cm fields of view were obtained on 14 patients with normal atlantoaxial articulations. The StealthStation workstation was used to reformat the axial images into sagittal, coronal, and 3-D views of the C1-2 anatomy. A “virtual” 4.0-mm diameter screw was constructed so that it was contained within the bony confines of the transarticular pathway while maximizing screw purchase in the C-1 lateral mass. The linear and angular dimensions of the idealized transarticular pathway were measured, as were distances from the virtual screw to the carotid sheath, hypopharynx, spinal canal, and vertebral artery.

The diameter of the isthmus of the C-2 pars interarticularis, which was typically pyramidal in shape, averaged 6.66 mm in the sagittal plane and 6.80 mm in the axial plane. Distance from the virtual screw edge to the vertebral artery averaged 2.26 mm and to the spinal canal 1.86 mm. Distance from the virtual screw to the carotid sheath and hypopharynx averaged 15.26 mm and 9.20 mm, respectively. No absolute linear and angular parameters for ideal transarticular screw placement could be established. The screw starting point averaged 3.28 mm cephalad to the C2-3 facet joint but with a range of 1.00 mm to 6.56 mm. The starting point averaged 5.56 mm lateral to the junction of the C-2 lamina and inferior articular process but with a range of 3.20 mm to 7.80 mm. The mean axial screw trajectory angle was 4.5° medial but ranged from 29° medial to 14° lateral.

The anatomical constraint of the atlantoaxial transarticular pathway is the isthmus of the C-2 interarticularis pars. The variability of C1-2 transarticular anatomy between patients is significant and precludes the establishment of absolute parameters for safe screw placement. Image-guided surgical technology allows for precise definition of individual anatomy and customized planning of transarticular screw trajectory to fit the individual anatomical constraints.
We performed a retrospective blinded review of 83 preoperative CT scans using the original Fisher scale and the proposed modified Fisher scale. The modified grading scale is as follows: Grade 0 — No blood or intraventricular hemorrhage alone or intraparenchymal hemorrhage alone; Grade 1 — Only basal cistern blood; Grade 2 — Only peripheral fissure blood; Grade 3 — Diffuse SAH with intraparenchymal hematoma; Grade 4 — Dense blood in basal cisterns and peripheral fissures. The CT grade was then correlated to the presence and severity of vasospasm diagnosed with transcranial Doppler ultrasound (TCD) postoperatively. Using the modified grading system, the incidence of vasospasm was found to increase with each grade: Grade 0: 1/10 patients (10%) had TCD vasospasm; Grade 1: 1/7 (14%); Grade 2: 3/8 (38%); Grade 3: 14/28 (50%); and Grade 4: 18/30 (60%). The results using the original Fisher scale were as follows: Grade 1: 2/11 (18.2%) patients had TCD vasospasm; in Grade 2: 10/24 (41.6%); Grade 3: 21/34 (61.7%); and Grade 4: 4/11 (36.4%).

The results obtained using the modified system are linear, with a correlation coefficient of $r = 0.98$, while the original Fisher scale was nonlinear ($r = 0.75$). There is a significant difference in the probability of developing vasospasm between grades using the new system ($p = 0.0032$ between each grade). Using this modified Fisher system, the chance of developing vasospasm can be more accurately predicted.
Wednesday Morning

**SPECIAL PRESENTATION**
**THE HISTORY OF PHILADELPHIA NEUROSURGERY**

11:15 AM – 11:25 AM (Ballroom A/B)

William Buchheit, MD (Philadelphia, PA)

**SPECIAL LECTURE III**

11:25 AM – 11:45 AM (Ballroom A/B)

**THE FUTURE OF MINIMALLY INVASIVE ENDOSCOPIC NEUROSURGERY**

Axel Perneczky, MD (Mainz, Germany)
(To be introduced by L.N. Hopkins, MD)
Wednesday Afternoon

SPECIAL SYMPOSIUM

11:45 AM – 12:45 PM   (Ballroom A/B)

DRAMATIC SOCIOECONOMIC CHANGES AFFECTING ALL NEUROSURGEONS

Moderator: Stanley Pelofsky, MD (Oklahoma City, OK)

Panelists:
Arthur L. Day, MD (Gainesville, FL)
Katie O. Orrico (Washington, DC)
John A. Kusske, MD (Laguna Hills, CA)

YOUNG NEUROSURGEONS SESSION

1:45 PM – 2:45 PM   (Room 204B)

HOW TO BECOME POLITICALLY ACTIVE WITHIN YOUR OWN PRACTICE

Russell Travis, MD (Lexington, KY)

By ticket only.

POSTER VIEW SESSION

1:45 PM – 2:45 PM   (Exhibit Hall A/B/C)

This time has been designated for uninterrupted viewing of the poster presentations. Authors will be present at their posters for questions and discussions.
Wednesday Afternoon

SECTION SESSIONS

2:45 PM – 5:30 PM

AANS/CNS Section on Pain
(Room 108A/B)

AANS Section on Disorders of the Spine and Peripheral Nerves
(Room 113A/B/C)

AANS/CNS Section on Neurotrauma and Critical Care
(Room 103A/B/C)

AANS/CNS Section on Tumors
(Room 114)
Wednesday Afternoon

SECTION SESSION
AANS/CNS Section on Pain
2:45 PM – 5:30 PM (Room 108A/B)

SPECIAL LECTURE
AANS/CNS Section on Pain
2:45 PM – 3:15 PM (Room 108A/B)
SPINAL CORD STIMULATION FOR ISCHEMIC PERIPHERAL VASCULAR DISEASE
Mario Meglio, MD (Rome, Italy)

SPECIAL SYMPOSIUM
AANS/CNS Section on Pain
3:15 PM – 4:00 PM (Room 108A/B)
CHRONIC INTRATHECAL MORPHINE FOR FAILED LAMINECTOMY SYNDROME: POINT/COUNTERPOINT
Richard Penn, MD (Chicago, IL)
John Loeser, MD (Seattle, WA)
Wednesday Afternoon

SCIENTIFIC SESSION

AANS/CNS Section on Pain

4:00 PM – 5:30 PM  (Room 108A/B)

Moderators: Kenneth Follett, MD, PhD (Iowa City, IA)
Dr. Rezai received a Bachelor of Science degree in Biology from the University of California, Los Angeles in 1986. He obtained his medical degree with honors from the University of Southern California in 1990. Subsequently, he completed his neurosurgery residency in June 1997 at New York University under Patrick J. Kelly. Dr. Rezai received the CNS clinical fellowship and the Bottrell fellowship awards in 1997.

Dr. Rezai is currently a clinical Fellow in Stereotactic and Functional Neurosurgery at the University of Toronto. Under the direction of Dr.'s Ronald R. Tasker and Andres M. Lozano, his fellowship focuses on the neurosurgical management of patients with movement disorders and chronic pain. Dr. Rezai's research interests includes the use of functional brain mapping such as Magnetoencephalography (MEG) and functional MRI (fMRI) and their application in integration into stereotactic techniques. He is currently using fMRI and MEG to investigate the mechanism involved in pain and tremor generation in patients undergoing chronic Deep Brain Stimulation (DBS).
Deep Brain Stimulation for Intractable Neuropathic Pain:
Contemporary Management and Outcome in 80 Patients

Ali R. Rezai
Alexandre M. Francisco (Toronto, ON, Canada)
Oswaldo V. Vilela Filho (Curitiba, Brazil)
Andres M. Lozano
Ronald R. Tasker (Toronto, ON, Canada)

KEY WORDS: brain stimulation, neuropathic pain, pain relief

From 1979 to 1997 at the Toronto Hospital, 80 consecutive patients with intractable chronic pain underwent deep brain stimulation (DBS) trials. In contrast to previous studies, this series did not include patients with nociceptive pain such as the failed back syndrome. All 80 patients had failed previous medical and surgical interventions. The mean age was 55 years; 42 were males. The mean duration of pain was 78 months. The pain was secondary to lesions in the central (40 patients) and peripheral (40 patients) nervous systems. Diagnoses included post-stroke pain (23), anesthesia dolorosa (20), spinal cord lesion/injury (17), and other peripheral nervous system lesions (20 patients). Electrode placement was guided by microelectrode recording and stimulation. The targets chosen were the somatosensory thalamus (VC) in 25 patients, medial lemniscus (ML) in 5, VC and periventricular gray simultaneously in 43, VC and ML in 5, and VC and internal capsule in 1 patient.

Pain relief was classified as excellent (> 2/3 pain reduction), good (1/3 to 2/3), fair (up to 1/3), and failure (no relief). Of 80 patients operated on, 71 proceeded to the test stimulation stage. Forty-eight of these 71 had satisfactory pain relief (> 50%) and received a permanent subcutaneous stimulator at a second procedure. At last follow-up review (mean 59 months, range 5–131 months), outcomes were excellent in 14 patients, good in 9, fair in 5, and no relief in 20. Technical or equipment-related complications were seen in 38% (earlier patients most commonly), and 20% had neurosurgical complications (2.5% permanent deficits).

In patients with intractable chronic neuropathic pain, DBS provides long-term pain relief in 35% of those undergoing initial trials or in 58% of those with permanent implantation. With continued advances in DBS technology and improved understanding of the pain pathophysiology, we can improve outcomes while minimizing complications.

Functional MRI: A Tool for the Evaluation of Spinal Cord Stimulation

Elaine Kiriakopoulos
Ronald Tasker
Andres Lozano
David Mikulis (Toronto, ON, Canada)

KEY WORDS: functional imaging, spinal cord stimulation, chronic pain

The management of chronic pain of spinal origin continues to represent a challenge for neurosurgeons. Spinal cord stimulation for chronic intractable pain is an effective therapy in approximately 50% of patients. The present study utilizes a novel imaging approach, functional magnetic resonance imaging (fMRI), to examine the central effects of spinal cord stimulation.

Three patients with a history of chronic intractable pain were treated at the Toronto Hospital with a trial of dorsal column stimulation (DCS). Functional MRI on a 1.5-Tesla conventional MRI system was used to study the effects of DCS in these patients. Images were collected while the stimulator was turned “off” and “on.”
In all cases a significant improvement in pain symptoms was achieved with DCS. Functional MRI mapping revealed regions of selective activation in the sensory cortex and cingulate gyrus. A graded response to stimulation was reported subjectively and correlated to an increase in the number of activated pixels on the fMRI mapping.

This report is the first to describe the cerebral effects of exogenous spinal cord stimulation with fMRI. fMRI allows for the objective examination of the effects of DCS and may provide an objective means of evaluating the efficacy of DCS as a therapy for intractable pain of spinal origin.

PAPER #832  WEDNESDAY, 4:30-4:45 PM  
Novel Delivery of Nucleosides and Antisense Oligonucleotides to the CNS

Robert M. Levy*  
Sherman Ward  
Kurt Schalgeter  
Dennis Groothuis (Chicago, IL)

Key Words: nucleoside, antisense oligonucleotide, convection-enhanced delivery

Nucleosides and antisense oligonucleotides are important agents for the treatment of CNS viral infection and neoplasms. They cross the blood-brain barrier (BBB) poorly; less than 1% of an intravenous dose will enter the brain. Convection-enhanced delivery (CED) circumvents the BBB, delivering 100% of the drug to the brain. We compared intravenous (IV), intraventricular (IVT), and CED of cytosine arabinoside (Ara-C), AZT, and a 20-mer nonsense phosphorothioate oligonucleotide (P-ODN) to the brain.

Sprague-Dawley rats were used for Ara-C and AZT and Swiss ICN mice for P-ODN. For IV delivery, 50 mCi 14C-Ara-C or 14C-AZT or 10 mCi of 35S-P-ODN were used. Timed plasma samples were obtained and the animals were killed 30 to 120 minutes later. For IVT delivery, a cannula was stereotactically placed in the lateral ventricle and an osmotic pump was used to deliver 1 to 5 mCi of drug. For CED, a cannula was stereotactically placed in the caudate nucleus and an osmotic pump used to deliver 1 to 5 mCi of drug at 0.5 to 10 ml/hr for 1 to 7 days. The animals were then sacrificed, the brains removed, and 20-mm cryostat sections were used for quantitative autoradiographic analysis.

Delivery patterns were similar for all three drugs. After IV administration, tissue concentrations reached maximum levels of less than 1 mCi/g. After IVT administration, the brain concentration was highest at the ventricular surface and declined exponentially; cortical drug concentration was negligible. Drug distribution after CED displayed a central component with very high drug concentrations and a peripheral component in which tissue concentrations declined exponentially. The volume of the central component was dependent on the rate and duration of the infusion. At 7 days, radiolabeled drug had reached the entire ipsilateral hemisphere and extended into the contralateral hemisphere.

In contrast to limited penetration by IV or IVT routes, CED of small amounts of drug produces very high tissue concentrations. Distribution is determined largely by infusion rate and duration and, by adjusting these parameters, large volumes of brain can be reached. Preliminary studies have not shown toxicity associated with CED infusions. Thus, CED appears to be safe and effective and may be of significance for the treatment of CNS disease in humans.
PAPER #833  WEDNESDAY, 4:45-5:00 PM
Percutaneous Balloon Compression for Trigeminal Neuralgia: Results in 183 Consecutive Patients

Jeffrey Brown
Jan J. Gouda
Devedutta Sangvai (Toledo, OH)

KEY WORDS: trigeminal neuralgia, balloon compression, outcome

Balloon compression has been an established treatment for 15 years for classical trigeminal neuralgia or neuralgia associated with multiple sclerosis. This series reviews the results of treatment in 183 consecutive patients. The technique has varied but is now standardized. Modified submental, anterioposterior, and lateral radiographic imaging is used. Autonomic changes and anatomical localization of the compressive site are sought. A blunt cannula system with guiding stylets has been designed, and compression pressure is monitored. Compression is limited to 1 minute at 1100 to 1300 mm Hg using a latex balloon catheter, so that the retrogasserian myelinated fibers are compressed at the entrance to Meckel's cave.

Mean patient age is 64 years (range 27–97 years). Mean follow up is 55 months (range 2 months–13 years). 30% had previous destructive procedures and 7% had multiple sclerosis. 37% had first division pain. Initial pain relief achieved is 93%. Pain-free Kaplan-Meier survival rate is 70% at 7 1/2 years. Recurrence rate is 25%. One patient has an absent corneal reflex. 19% had masseter weakness, which was temporary and recovered in a maximum of 1 year. The 6% of patients who describe their numbness as severe were treated early in the series before pressure monitoring was done; 80% think it to be mild. Other morbidity includes: diplopia (one patient), aseptic meningitis (2%), external carotid fistula (one patient). Pain-free survival rate in patients with first division pain is the same as that for other divisions. Anesthesia dolorosa has not occurred.

Balloon compression is a simple, cost-effective outpatient treatment. It injures the large myelinated fibers which trigger the pain and is especially useful in multidivision or first division trigeminal neuralgia.

PAPER #834  WEDNESDAY, 5:00-5:15 PM
NGF and Anti-NGF Treatment of Chronic Pain Following Spinal Cord Injury

Marc D. Christensen
Claire Hulssebosch (Syracuse, NY)

KEY WORDS: spinal cord injury, chronic pain, nerve growth factor

Spinal cord injury (SCI) frequently results in chronic pain and dysesthesias. We have developed a model of chronic pain following traumatic SCI. Furthermore, the mechanisms responsible for the induction and maintenance of chronic pain have been investigated and treatment modalities tested. Male Sprague-Dawley rats were hemisected at T-12 and were tested preoperatively and postoperatively in response to mechanical and thermal stimuli using paw withdrawal and supraspinal responses to von Frey hair (vFh) and a radiant heat source. Prior to spinal hemisection in controls (n = 10), the rats did not withdraw their paws or vocalize to vFh bending force of 4.41 mN. Following SCI (n = 10), an increased frequency of paw withdrawal to 4.41 mN by 70% and decreased latency of withdrawal in response to a radiant heat source occurred (p < 0.05). Mechanisms responsible for the development of these dysesthesias after SCI include anatomical and molecular plasticity of pain fibers in response to endogenous neurotrophins such as nerve growth factor (NGF). Spinal cords and dorsal root ganglia (DRG) of intrathecal NGF (35 ng/hr)-treated animals (n = 5) had increased levels of the nociceptive peptide calcitonin gene-related (CGRP) protein and mRNA when compared to controls (p < 0.05).The NGF-treated rats also displayed marked exacerbation of
mechanical and thermal allodynia as assayed by vFh testing, thermal withdrawal latencies, and supraspinal nociceptive behavior such as vocalization (p < 0.05). Finally, animals treated (n = 10) with intrathecal antibodies to NGF (anti-NGF) had attenuated quantities of CGRP protein and mRNA in the spinal cord and DRG (p < 0.05). Also, intrathecal anti-NGF prevented the development of mechanical and thermal allodynia and supraspinal nociceptive behaviors following SCI (p < 0.05).

PAPER #835  WEDNESDAY, 5:15-5:30 PM
Long-term Outcome of Spinal Cord Stimulation for Chronic Pain Management

Giancarlo Barolat*
Beth Ketcik (Philadelphia, PA)

Key Words: chronic pain, spinal cord stimulation, electrode implantation

This is a retrospective study on 102 patients subjected to implantation of a spinal cord stimulation system for nonmalignant chronic pain management. The study was conducted through an extensive questionnaire and telephone interview by a disinterested third party. All the patients were implanted with a complete spinal cord stimulation system without a preliminary trial with a temporarily implanted electrode. Diagnostic categories were: neuropathic pain, failed back syndrome, spinal cord injury pain, and miscellaneous. Average follow-up review was 3.8 years (6 months–8 years). Patients were divided in two groups: all the implanted patients in the survey (Group A) and the implanted patients who experienced some degree of pain relief with the stimulation (Group B). Group B (80 patients) closely matches previously published series where an initial temporary screening was performed.

Twenty-one percent of the patients never experienced any pain relief. Of the remaining 80 patients, 75% were still using the stimulator at follow up: 51% of the 80 patients were experiencing good to excellent results and 20% moderate results. There was no reduction over time in the amount of pain relief in patients who initially had at least 75% pain relief. Patients with initial pain relief between 50 and 74% observed a moderate reduction in their pain relief after 2 years. Patients who initially experienced less than 50% pain relief observed a dramatic reduction in their results in the long-term follow up. Psychological screening contributed to the success of the procedure.

With proper medical and psychological screening and with demonstrated initial pain relief, spinal cord stimulation remains one of the most effective modalities in the long-term management of chronic pain.
Wednesday Afternoon

SECTION SESSION
AANS/CNS Section on Disorders of the Spine and Peripheral Nerves
2:45 PM – 5:30 PM (Room 113A/B/C)

SPECIAL SYMPOSIUM
AANS/CNS Section on Disorders of the Spine and Peripheral Nerves
2:45 PM – 3:45 PM (Room 113A/B/C)

ABC’S OF PERIPHERAL NERVE INJURY MANAGEMENT
Allan J. Belzberg, MD (Baltimore, MD)

ABC’S OF EMGS
Robert L. Tiel, MD (Mandeville, LA)
Wednesday Afternoon

Scientific Session

AANS/CNS Section on Disorders of the Spine and Peripheral Nerves

3:45 PM – 5:30 PM (Room 113A/B/C)

Moderators: Richard G. Fessler, MD (Gainesville, FL)
Kevin T. Foley, MD (Los Angeles, CA)

Paper #836 Wednesday, 3:45-4:00 PM
Inhibition of Postoperative Peridural Fibrosis and Reduction of Fibrosis-Related Symptoms With a Bioresorbable Carbohydrate Gel: ADCON-L

Joseph Maroon (Pittsburgh, PA)*

Key Words: fibrosis, pain, carbohydrate gel, lumbar discectomy

Peridural scar and adhesions, which develop as a result of the natural healing process after lumbar discectomy, can result in recurrence of clinical symptoms due to dural or nerve root compression and tethering. This randomized, controlled, double-blind, multicenter clinical investigation evaluated the safety and effectiveness of a novel bioresorbable gel (ADCON-L) for the inhibition of postoperative peridural fibrosis and reduction of fibrosis-related symptoms.

Adult patients undergoing first-time L4-5 or L5-S1 discectomies were entered into the study. Immediately prior to closure, patients were randomly assigned to receive ADCON-L gel or no treatment (control). Peridural fibrosis was quantified 6 months postoperatively by MRI using the method of Ross, et al. (Neurosurgery, 38: 855-861, 1996). MR images were obtained with and without gadolinium enhancement and were scored by a single neuroradiologist blinded to patient treatment. Patient outcome was assessed using the Roland Morris Activities Performance (RMAP) scale and by measuring the incidence of pain upon specific activities of daily living. For this protocol-specified interim analysis, all statistical tests on the outcome measures were one-tailed with a Type I error of 0.05.

One hundred sixty-five patients were available for evaluation from 14 clinical sites in the United States at the time of this analysis. There were no significant differences in preoperative patient demographics or clinical examination findings. Patients receiving ADCON-L gel had significantly less peridural scar as measured by MRI than the control patients 6 months postoperatively (p = 0.016). Furthermore, there was a significant reduction in the incidence of extensive peridural scar (> 75% of MRI field containing scar) in the patients receiving the gel (p = 0.003). Patients receiving ADCON-L also had significantly better scores on the RMAP scale compared to control patients (p = 0.016) and reported less activity-related pain. There were no statistically significant differences between the groups in the types and frequencies of adverse or medical events.

Interim results of this prospective, multicenter, randomized clinical investigation indicate that ADCON-L gel safely inhibits peridural scar in patients undergoing first-time spinal root decompression and that these patients have better postoperative outcome.
Risks, Benefits, and Alternatives to Atlantoaxial Arthrodesis With Posterior Transarticular Facet Screws

Dan M. Lieberman
Bruce McCormack
Eldon Eichbaum
Kathleen Lamborn (San Francisco, CA)

KEYWORDS: facet screw, arthrodesis, spinal injury, rheumatoid arthritis

Clinical series and biomechanical studies have indicated the overall superiority of posterior transarticular facet screws (PTFS) to techniques using wire or clamps for atlantoaxial arthrodesis. However, there is considerable reported variation in efficiency of arthrodesis and complications achieved using Magerlís technique between cohorts, and precise indications for PTFS for specific groups have not been widely discussed. Here we report our own series from UCSF of 37 cases of atlantoaxial arthrodesis using PTFS. In addition, to clarify the risks and benefits of the procedure to traditional practice, we collated all reported cases of atlantoaxial arthrodesis since 1978 by etiology and compared their outcome.

Among all such patients with rheumatoid arthritis (n = 242), nonunion (n = 26, 11%) occurred more frequently using Halifax clamps (n = 4, 22%) or wiring techniques (n = 15, 13%) than with PTFS (n = 7, 6%). In this group, there were 10 perioperative deaths (4%). Following trauma (n = 273), there were no reported cases of non-union using PTFS in contrast with traditional techniques (n = 7, 5%), and 5 deaths (2%). By contrast, in the treatment of os odontoideum (n = 42) non-union was more common following PTFS (n = 1, 20%), compared to traditional techniques (n = 3, 12%).

PTFS are associated with higher rates of fusion and similar morbidity when compared with traditional techniques for patients following trauma, or with rheumatoid arthritis. For patients with os odontoideum, however, the additional rigidity achieved using PTFS may not ultimately translate into a higher rate of fusion.

The Fusion Rate for an Anterior Cervical Fusion (ACF) in Patients With a Prior History of a Successful ACF at a Different Level

Bryan Wellman
Vincent Traynelis (Iowa City, IA)

KEYWORDS: spine fusion, cervical spondylosis

It is known that the fusion rate for a single-level anterior cervical fusion (ACF) is 95%, while a two-level ACF carries an approximate fusion rate of 85%. However, it is not known what the fusion rate is for a single-level ACF in a patient with a prior history of an ACF at another level. Is the fusion rate in this group similar to a single-level fusion or a two-level fusion?

Over the last 10-year period a total of 492 ACFs were performed for cervical spine spondylosis by the Division of Neurosurgery at The University of Iowa. Of these 492 patients, 23 (4.7%) had a history of a prior ACF at a different level. In this select group of 23 patients, the medical records and flexion/extension cervical spine radiographs were evaluated to establish fusion rates and risk factors for fusion failures. Three patients did not have flexion/extension radiographs and were eliminated from further analysis. Of the remaining 20 patients, 17 had successful fusions resulting in an 85% fusion rate. All 3 patients with fusion failures were cigarette smokers. With regard to graft material, a similar fusion rate was seen with autologous and banked bone (88% for autologous and 83% for banked bone).
In summary, the fusion rate for an ACF in patients with a history of a prior ACF at a different level is similar to the fusion rate for performing a two-level fusion. Also, as in other fusion series, smoking is a major risk factor for fusion failure.

PAPER #839 WEDNESDAY, 4:30-4:45 PM
Fractures of the Lower Thoracic and Lumbar Spine: Treatment With the Internal Fixator

Wulf-Rüdiger Niendorf
Henry Schroeder
Stefan Berger
Michael Gaab (Greifswald, Germany)

KEY WORDS: spine fracture, internal fixator, instrumentation

The goals of treating unstable spine injuries—operative decompression, stabilization, and early mobilization—are available in most cases with the internal fixator alone and in one low-risk operation. This kind of posterior fixation has become the preferred method of treatment for unstable thoracolumbar fractures. Since 1989, a total of 122 patients underwent posterior instrumentation of unstable thoracolumbar fractures with the internal fixator in our department. Types used were the fixators by Kluger, DSF, and Weber. Clinical data were recorded prospectively with respect of the type of fracture, neurological symptoms, details of instrumentation, operative complications, correction of spinal deformity, and long-term outcome. Clinical investigations and evaluations of CT, MRI, and plain x-ray films were made at admission, immediately postoperatively, at removal of the implants (9-12 months after the accident), and 1 and 2 years after removal of the internal fixator.

The follow-up study was achieved in over 93% of the patients, with a mean postoperative observation of 3 years. Up to now a fusion rate of 98% has been obtained; in only two cases was the posterior instrumentation not adequate and an anterior procedure was necessary. We recorded and evaluated factors for the relapse of the kyphotic deformity after stabilization (e.g., decompression of the spinal canal via extensive laminectomy). Major postoperative complications were 3 isolated nerve root deficits and 5 infections (2 deep, 3 superficial). The instrumentation was removed only in deep infections. There was no intraoperative death and no injury of prevertebral vessels or inner organs. Instrument failures, such as broken screws and rods, developed in 7% of cases. No patient with a complete transversal syndrome but 95% of the patients with incomplete neurological lesions improved.

We recommend posterior stabilization with the internal fixator as a standard treatment of unstable fractures of the thoracolumbar spine. This method allows an early mobilization of the patients, a satisfactory reconstruction, and stabilization of the fractured vertebral body.

PAPER #840 WEDNESDAY, 4:45-5:00 PM
Anterior Fusion: The Wrong Operation for Discogenic Cervical Radiculopathy

Frederick Simeone (Philadelphia, PA)

KEY WORDS: spinal fusion, surgical approach, radiculopathy, foraminotomy

When operative correction of cervical nerve root compression secondary to acute or chronic disc herniation is required, most neurosurgeons and virtually all orthopedic spine surgeons choose an anterior approach. The operation of posterior foraminotomy and discectomy, with or without the operating microscope, has emerged as an equally effective yet safer operation, with fewer long-term complications, less time off from work for the patient, and a more cost-effective procedure.
The author has performed microsurgical posterior foraminotomy for many years, and the results of the last 5 years’ clinical experience (780 cases) have been analyzed. By their own assessment, good to excellent results were achieved in 98% of patients. Nine patients were involved in litigation or had compensable injuries preoperatively.

Transient postoperative weakness was seen in a small number of patients, usually in a cerebrospinal nerve root distribution. Four patients had permanent weakness postoperatively which was not present preoperatively. The recovery of preoperative neurological deficit was variable and could not be determined accurately from this retrospective review. As one would expect, certain complications of anterior surgery were not seen in posterior microforaminotomy patients. These include hoarseness, difficulty with swallowing, Homer’s syndrome, fusion non-union, and donor site complications. Transient complications related to the pin fixation on cranium head holders were seen in the posterior microforaminotomy patients and were not seen in patients with anterior surgery. The overall infection rate in patients with posterior microforaminotomy was 0.25%. Patients with posterior microforaminotomy were discharged on the day following surgery, although same-day discharge is possible. The mandatory use of a cervical collar was not required or recommended. The postoperative time off from work averaged 3 weeks, which is shorter than that reported for anterior fusion surgery. Because mandatory collar use following anterior fusion is frequently required, time off from work is extended in patients who could not drive with a cervical collar in place because of comfort or legal restrictions. There were no instances of late subluxation following unilateral cervical foraminotomy.

With equal or superior results to anterior surgery (depending on the series reviewed), microforaminotomy offers distinct long-term advantages. Accelerated disc degeneration at adjacent levels above or below an anterior cervical fusion occurs frequently. Instability or, more frequently, symptomatic herniation may require more surgery, although this process of fusion-generated adjacent stress may take years to develop. Nevertheless, symptomatic adjacent disc herniations are common, although no series accurately determines this incidence. Another distinct advantage of posterior microforaminotomy is that more than one level can be explored when the history, examination, and radiographs cannot differentiate between two symptomatic levels. This dilemma is frequently seen when C5-6 and C6-7 herniations occur concurrently. To explore two or more levels anteriorly requires fusion at each explored level.

Neurosurgeons should learn to master the annoying technical difficulties encountered during the learning curve of this procedure so as to offer their patients a less complicated, more efficient cure for discogenic cervical radiculopathy.

PAPER #841 WEDNESDAY, 5:00-5:15 PM
The Use of Pedicle or Pars Screws in Long-Segment Subaxial Posterior Cervical Fusion
David W. Cahill (Tampa, FL)

KEY WORDS: spinal fusion, pedicle screw, laminectomy

Since 1991, we have used laminectomy and posterior cervical fusion with lateral mass plates for the surgical management of spondylotic or rheumatoid myelopathy when cord compression is present for more than 3 motion segments. We also employed these procedures for selected tumor and deformity cases. Prior to 1995, we plated and fused only the laminectomized segments. Using this technique, the incidence of caudal segmental screw pull-out was unacceptably high (8/27). In an effort to prevent this complication, we began to employ pedicle screws at the caudal end of any construct extending across 5 or more vertebrae (4 segments) in 1995. Long screws are routinely used in T-1 and T-2 and not infrequently in C-7. Routine lateral mass screws
are employed in C3-6. In constructs extending 6 or more cervical levels, long C-2 pars screws are used to prevent rostral screw migration.

In the 30 months since these techniques have been employed, there have been no cases of screw pull-out at either end of long constructs in 37 consecutive cases. There have been no vascular, radicular, or cord injuries and no infections. There have been 2 fractured T-1 pedicle screws, both 3.5-mm screws and both unilateral. No 4-mm screws have fractured. Due to the extreme movements applied to long lordotic lateral mass plates during neck flexion, the incidence of end screw pull-out in plates fixed only with short lateral mass screws may be as high as 30% in constructs involving more than 4 vertebrae, especially in the face of osteopenia, multicolumn instability, deformity, or kyphosis. The incorporation of long cervicothoracic pedicle screws or axial pars screws into such constructs appears to greatly increase fixation strength and decrease screw failure and risk of pseudarthrosis.

PAPER #842  WEDNESDAY, 5:15-5:30 PM
Circumferential Surgery for the Management of Cervical Ossification of the Posterior Longitudinal Ligament

Nancy Epstein (New Hyde Park, NY)

Key Words: spinal fusion, ossification of posterior longitudinal ligament, spinal stenosis

Can simultaneous anterior and posterior (circumferential) surgery in patients with ossification of the posterior longitudinal ligament (OPLL) and spinal stenosis of the cervical spine, achieving both decompression and stabilization, be accomplished with acceptable risk? Between 1989 and 1996, 22 circumferential operative procedures were performed including an average of 2.5-level corpectomy with 5-level posterior wiring and fusion. These patients were severely myelopathic (average Nurick Grade 3.5) and were followed for a mean interval of 22 months (range 4–52 months).

Circumferential procedures required an average of 9.8 hours and 3.5 units of blood transfused. Postoperatively, patients improved approximately + 3.0 Nurick grades. Simultaneous circumferential surgery for OPLL and cervical spinal stenosis may be successfully performed in under 10 hours with limited blood loss and acceptable risk.
Wednesday Afternoon

SECTION SESSION

AANS/CNS Section on Neurotrauma and Critical Care

2:45 PM – 5:30 PM     (Room 103A/B/C)

SPECIAL LECTURE

AANS/CNS Section on Neurotrauma and Critical Care

2:45 PM – 3:15 PM     (Room 103A/B/C)

ANTIBIOTIC-RESISTANT ORGANISMS:
WHAT EVERY NEUROSURGEON SHOULD KNOW

Robert Muder, MD (Pittsburg, PA)
(To be introduced by Brian T. Andrews, MD)
Wednesday Afternoon

SPECIAL SYMPOSIUM

AANS/CNS Section on
Neurotrauma and Critical Care

3:15 PM – 3:45 PM (Room 103A/B/C)

UPDATE ON NEUROTRAUMA:
CURRENT STATE OF THE ART
Moderator: Brian T. Andrews, MD (San Francisco, CA)

SPINAL CORD INJURY:
CURRENT STATE OF THE ART
Michael Fehlings, MD (Toronto, ON, Canada)

HEAD INJURY:
CURRENT STATE OF THE ART
M. Ross Bullock, MD (Richmond, VA)
Wednesday Afternoon

**SCIENTIFIC SESSION**

3:45 PM – 5:30 PM     (Room 103A/B/C)

**Moderator:** Brian T. Andrews (San Francisco, CA)

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**PAPER #843 WEDNESDAY, 3:45-4:00 PM**

**Risk of Early Closed Reduction in Cervical Spine Trauma**

Gerald Grant
Sean M. Grady
Sohail Mirza
Jens Mirza
David Newell (Seattle, WA)

**KEY WORDS:** spine injury, closed reduction, intervertebral disc herniation

Controversy continues regarding the proper treatment of acute traumatic cervical spine injuries. We reviewed 82 patients with traumatic cervical injury to determine the risk of neurological deterioration following immediate closed reduction.

The medical records and imaging studies (CT and MRI) of 82 patients (63 male [77%] and 19 female [23%], mean age 42 years) with bilateral (15, 18%) and unilateral (26, 32%) locked facet dislocations, burst fractures (21, 26%), extension injuries (9, 11%), or miscellaneous cervical fractures with subluxation (11, 13%) were reviewed. Disc injury was defined by MRI as herniation or disruption: a herniation was described as deforming the thecal sac or nerve roots and a disruption was defined as a disc with high T2-weighted MRI signal characteristics in a widened disc space.

Of the 82 patients, 58% presented with complete (25, 30%) or incomplete (21, 26%) spinal cord injuries. Eleven patients (13%) presented with a cervical radiculopathy, 18 patients (22%) were intact, and 7 (9%) had only transient neurological deficits in the field. Eighty patients (97.6%) were reduced with early, rapid closed reduction using serial plain radiographs and Gardner-Wells craniocervical traction. Two patients failed closed reduction and underwent emergency open surgical reduction. The average time for closed reduction was 2.1 ± 0.24 hours (SEM). The incidence of disc herniation and disruption in the 76 patients who underwent a postreduction MRI was 22% (17 patients) and 23.6% (18 patients), respectively. The incidence of disc herniation in 15 patients with bilateral facet dislocations was 13.3% (2 patients). The incidence of disc herniation in 26 patients with unilateral facet dislocations was 23.1% (6 patients). The presence of disc herniation or disruption had no effect on the degree of neurological recovery measured by ASIA motor score and Frankel Scale following early closed reduction (p = 0.34, Pearson correlation coefficient). Only 1 (1.3%) of 80 patients deteriorated following closed reduction. This patient presented with a burst fracture with a subluxation type of injury, deteriorated (4/100 motor score points) beyond 6 hours postreduction, and was found to have a lateral disc herniation on MRI. Another patient underwent a prereduction MRI revealing a herniated disc which then resolved upon repeated imaging following closed reduction.
Although disc herniation and disruption can occur following all types of traumatic cervical fracture subluxations, the incidence of neurological deterioration following closed reduction in these patients is extremely rare (1.3%). We recommend immediate closed reduction in these patients without prior MR imaging.

PAPER #844  
WEDNESDAY, 4:00-4:15 PM  
Surgical Treatment of Acute Spinal Cord Injury Study (STASCIS): Results of a Multicenter Retrospective Pilot Study in 585 Patients  

Charles Tator  
Michael Fehlings (Toronto, ON, Canada)  

Key Words: spinal cord injury, cord decompression, timing of surgery  

The timing and role of decompression in acute spinal cord injury (SCI) remain controversial. To obtain pilot data in support of a planned prospective, multicenter trial of decompression by surgery or traction in SCI (STASCIS), we conducted a multicenter retrospective study to document the current surgical management of patients with SCI. All patients between the ages of 16 and 75 years with SCI or traumatic lesions of the cauda equina admitted between August, 1994, and April, 1995, at 36 North American centers were included in the study. Patients with associated severe head injuries (n = 18), intoxication (n = 7), or ankylosing spondylitis (n = 2) were excluded. A total of 585 cases were analyzed, of which 42.2% were complete (ASIA A) and 57.8% were incomplete (ASIA B-D). The anatomical distribution of injuries was: 64.5% cervical, 18.7% thoracic, 11.0% lumbar, 5.8% lumbosacral. Imaging modalities used included CT in 100% of cases, CT/myelography in 6%, and MRI in 54%. Traction was used in 47% of cervical injuries and resulted in successful cord decompression in 42% of those in whom it was used; traction was associated with neurological deterioration in 14 cases (8.1%). Surgery was performed in 65.4% of cases. The timing of surgery varied widely: < 24 hours, 23.5%; 25–48 hours, 15.8%; 48-96 hours, 19%; and > 5 days, 41.7%. These data indicate that, while surgery is commonly used in patients with SCI, there is very little agreement on the optimal timing of surgical treatment. The pilot study confirms the need for a randomized controlled trial of the optimal timing of surgical decompression in SCI.

PAPER #845  
WEDNESDAY, 4:15-4:30 PM  
Effect of Cerebrospinal Fluid Drainage on Cerebral Perfusion and Oxygenation  

Mary Kerr  
Donald W. Marion  
Patricia Orndoff  
Barb Weber  
Susan Sereika  
Rick Henker (Pittsburgh, PA)  

Key Words: cerebrospinal fluid drainage, cerebral perfusion, oxygenation  

Cerebrospinal fluid drainage is known to decrease the ICP; however, there is little evidence to indicate whether the intervention improves cerebral perfusion and/or oxygenation. The purpose of this investigation was to document improvement in cerebral perfusion and/or oxygenation post-CSF drainage. The study involved 36 severely head-injured adults (GCS score < 8). ICP, cerebral perfusion pressure (CPP), cerebral blood flow velocity (V_mca), jugular venous oxygen tension, and regional cerebral oxygenation (rSO_2) were recorded before, during, and after a CSF drainage protocol. Data were collected at 70 Hz using BiopPAC, a continuous multimodal computerized bedside data acquisition system. All subjects underwent 3 CSF drainage protocols with the volume amount (1, 2, and 3 ml) removed in random order. Analysis involved a within-subject RM-ANOVA aggregated across drainage protocols.
Following drainage, there was a 17% reduction in ICP (25.4–21.0 mm Hg). In 9 subjects (25%), the mean ICP returned to elevated baseline values within 7 minutes after drainage. There was minimal improvement in CPP (4.2%), and by 1 minute postdrainage, the CPP returned to baseline values. There were no significant changes in $V_{mca}$, jugular venous oxygen tension, or $rSO_2$ following drainage.

CSF drainage provides a transient decrease in ICP in the majority of patients. However, there is no evidence this decrease in ICP improves cerebral perfusion or oxygenation.

PAPER #846 WEDNESDAY, 4:30-4:45 PM
Rapid Correction of Warfarin Coagulopathy in Intracranial Hemorrhage

Nicholas M. Boulis
Miroslav P. Bobek
Alvin Schmaier
Julian T. Hoff (Ann Arbor, MI)

KEY WORDS: coagulopathy, warfarin, intracranial hemorrhage

Indications for the use of warfarin anticoagulation in the prevention of thrombotic and embolic disease have expanded. However, warfarin increases the risk of intracranial hemorrhage. Such bleeding lasts longer, creating larger hemorrhages. Therapeutic anticoagulation impedes surgical evacuation. Nonetheless, there are no guidelines for the emergency correction of warfarin-induced coagulopathy with intracranial hemorrhage. One retrospective study found that prothrombin complex speeds correction.

We randomly assigned patients to Group 1 or 2 when a prothrombin time (PT) > 17 was demonstrated and intracranial hemorrhage was documented on CT. The groups had similar mean ages (62 and 65 years) and both underwent expedient vitamin K and rapid fresh-frozen plasma (FFP) administration. Group 1 received factor IX complex (Konyne 80: Bayer) dosed according to calculated factor deficits. Group 2 underwent internal jugular central venous pressure monitoring to prevent fluid overload during rapid FFP administration. Prothrombin time and international normalized ratio (INR) measurements were taken at 2-hour intervals from the time of randomization. In group 1, PT was also measured before and after Konyne administration. Time to correction (INR = 1.3) was 3.75 hours in Group 1 (n = 5) and 8.75 hours in Group 2 (n = 4) (p < 0.017). Rate of correction was 3.04 PT/hr in Group 1 and 0.94 PT/hr in Group 2 (p < 0.027). The rate correction during Konyne administration was 49.2 $\pm$ 23.1 PT/hr. Group 1 required less colloid volume for correction (615 vs 1952 cc, p < 0.04). This study encourages further investigation of Konyne 80 as a standard treatment of warfarin-related intracranial hemorrhage.

PAPER #847 WEDNESDAY, 4:45-5:00 PM
Using Transgenic Technology to Explore the Function of Single Genes in the Pathophysiology of Traumatic Brain Injury

Kathryn E. Saatman
Ramesh Raghupathi
Michio Nakamura
Uwe Scherbel
Eugene S. Flamm (Philadelphia, PA)

KEY WORDS: brain injury, neurofilament, transgenic technology, gene function

Transgenic mice provide the opportunity to investigate the effects of single gene manipulations on nervous system function. In two recent studies, we utilized this approach to examine the roles for bcl-2, a proto-oncogene involved in inhibi-
tion of both necrotic and apoptotic cell death, and neurofilament, a key component of the neuronal cytoskeleton, in the pathophysiology of traumatic brain injury.

In the first study, transgenic mice overexpressing human bcl-2 (TG, n = 21) and their wild-type littermates (n = 18) were subjected to controlled cortical impact brain injury (CCI). Overexpression of bcl-2 significantly attenuated injury-induced cortical cell loss at 1 week postinjury, with only limited improvement in neurological motor function, suggesting that bcl-2 may play a protective role during the injury-induced neurodegeneration cascade. In the second study, transgenic mice overexpressing human high molecular weight neurofilament fused to beta-galactosidase (NFH-LacZ TG, n = 19) and their wild-type littermates (n = 17) received CCI. The NFH-LacZ TG mice, which display neurofilament-rich perikaryal inclusions and reduced endogenous neurofilament content prior to brain injury, exhibited a marked exacerbation of both injury-induced neurological motor dysfunction and cell loss over 4 weeks postinjury.

These data suggest that abnormal neurofilament composition may adversely affect the initial response to brain injury and the progression of cell death and functional recovery. Our studies highlight the manner in which transgenic technology is being exploited to elucidate the roles of single genes in the behavioral and histopathological sequelae in traumatic brain injury. (Supported, in part, by NINDS P01-NS08803, R01-NS26818, NIGMS R01-GM34690, and a VA Merit Review grant.)

PAPER #848 WEDNESDAY, 5:00-5:15 PM
S-100 and NSE Serum Measurements After Severe Head Injury

Chris Woertgen
Ralf Dirk Rothoerl
Matthias Holzschuh
Christopher Metz
Alexander Brawanski (Regensburg, Germany)

KEY WORDS: head injury, S-100, neuron-specific enolase

Neuron-specific enolase (NSE) and S-100 protein are known indicators of brain damage. We investigated the time course of NSE and S-100 protein after severe head injury in correlation to outcome. We included 30 patients (GCS score < 9), who had been admitted within 5 hours after injury, in a prospective study. Blood samples had been taken on admission, 6, 12, and 24 hours, and every 24 hours up to the 5th day after injury. The outcome was estimated at discharge using the Glasgow Outcome Scale (GOS).

Of the 30 patients, 70% reached a good outcome (GOS score 3-5). All concentrations of NSE and 83% of the S-100 samples were elevated with the first probe (mean: 30.2 mg/L NSE and 2.6 mg/L S-100). Patients with a bad outcome had an NSE concentration of 38 mg/L (mean) compared to 26.9 mg/L (mean) in patients with a good outcome. Patients with a bad outcome had an S-100 concentration of 4.9 mg/L (mean) compared to 1.7 mg/L (mean) in patients with a good outcome (p < 0.05; Fisher PLSD). The mean values of NSE and S-100 decreased during the first 5 days. Four patients with an increasing ICP showed a quickly increasing concentration of NSE; the S-100 level showed a slower rise in two patients. The NSE and S-100 serum levels did not correlate to ICP values (NSE-ICP: \( R^2 = 0.33 \); S-100-ICP: \( R^2 = 0.56 \)).

Our results show that the first serum concentration of S-100 seems to be predictive for outcome after severe head injury.
Novel Mechanisms of Calcium-Mediated Axonal Injury After Spinal Cord Trauma

Michael Fehlings
Sandeep Agrawal (Toronto, ON, Canada)

**KEY WORDS:** axonal injury, spinal cord injury, calcium channel

Although increases in intracellular calcium are linked to the pathophysiology of CNS injury, the mechanisms of calcium-induced white matter injury are unclear given the apparent lack of calcium channels on axons. To clarify this, we have examined mechanisms of calcium-dependent axonal injury in an in vitro model of spinal cord injury (SCI). Compound action potentials were recorded from isolated rat dorsal column segments. Injury was accomplished by compressing the dorsal column strip with a 2-g clip for 15 seconds. Confocal immunocytochemistry for L, N and P/Q-type calcium channels was performed with double labeling for GFAP.

Removal of extracellular calcium promoted significantly greater recovery of CAP amplitude to 86.3% ± 7.6% of control (71.0% ± 2.0%). Blockade of voltage-gated calcium channels with cobalt (20 mM) also conferred neuroprotection (CAP amplitude 82.8% ± 1.2% of control). Pretreatment with the Sodium/Calcium exchanger blockers benzamil or bepridil did not confer neuroprotection. In contrast, AMPA/kainate receptor blockers NBQX and CNQX (10 mM) enhanced the recovery of CAP amplitude to 85.6% ± 2.7% and 86.5% ± 3.9% of control values. Blockade of L-type voltage-gated calcium channels with diltiazam (50 mM) also conferred neuroprotection (CAP amplitude 86.6% ± 3.3% of control). With immunocytochemistry we identified the presence of L-type immunopositive periaxonal astrocytes (double labeled with GFAP).

The injurious effects of calcium in traumatically injured CNS white matter appear to be related to voltage-gated calcium channels and calcium-permeable AMPA/kainate receptors lacking the GluR-B subunit. These results may allow the development of novel neuroprotective approaches for CNS injury.

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**BUSINESS MEETING**

AANS/CNS Section on Neurotrauma and Critical Care

5:30 PM – 6:00 PM  (Room 103A/B/C)

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**Wednesday Afternoon**

**SECTION SESSION**

AANS/CNS Section on Tumors

2:45 PM – 5:30 PM (Room 114)

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**SPECIAL SYMPOSIUM**

AANS/CNS Section on Tumors

2:45 PM – 3:45 PM (Room 114)

INNOVATIVE STRATEGIES FOR TREATMENT OF GLIOMAS

Moderator: Kevin O. Lillehei, MD (Denver, CO)

ADVANCES IN IMMUNOTHERAPY/ TUMOR VACCINES

Drew Pardol, MD (Baltimore, MD)

ANTI-ANGIOGENIC APPROACHES TO GLIOMA THERAPY

Judah Folkman, MD

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**SCIENTIFIC SESSION**

AANS/CNS Section on Tumors

3:45 PM – 5:10 PM (Room 114)

Moderators: Mark Bernstein, MD (Toronto, ON, Canada)

Joseph Peipmeier, MD (New Haven, CT)
Wednesday Afternoon

PRUESS RESIDENT AWARD PRESENTATION

AANS/CNS Section on Tumors

3:45 PM – 4:00 PM  (Room 114)

Matthias M. Feldkamp, MD (Toronto, ON, Canada)
(To be introduced by Mark Bernstein, MD)

Matthias Feldkamp was born in Toronto, Ontario, Canada, and was raised in Toronto and Saskatoon, Saskatchewan, Canada. He attended medical school at the University of Saskatchewan in Saskatoon, obtaining his MD degree in 1991. He subsequently interned at Mount Sinai Hospital in Toronto, and began his neurosurgical training at the University of Toronto in 1992. Following three years of clinical training, he transferred into the Surgical Scientist Program at the University of Toronto, where he has been pursuing a PhD degree since 1995. His research interest is in molecular neuro-oncology, and his focus has been on the role of Ras pathway activation in astrocytomas. His research is being undertaken at the Samuel Lunenfeld Research Institute in Toronto, under the supervision of Dr. Abhijit Guha. He plans to complete his PhD degree requirements by 1999, and to complete his neurosurgical training. His career plans encompass both an interest in basic laboratory neuro-oncology research as well as a clinical neurosurgery practice with an emphasis on neuro-oncology.
Expression of Growth Factor Receptors in Glioblastoma Multiforme Cell Lines and Tumor Specimens: Results in Ras Activation and Ras-Dependent Tumor Proliferation

Matthias M. Feldkamp
Nelson Lau
Abhjit Guha (Toronto, ON, Canada)

Key Words: growth factor receptor, tumor proliferation, glioblastoma multiforme

Glioblastoma multiformes (GBM) express high levels of growth factor receptors, particularly the epidermal growth factor receptor (EGF-R). We have proposed that these receptors activate the Ras pathway in these cells, resulting in Ras-dependent proliferation. The status of EGF-R and PDGF-R was evaluated in five established astrocytoma cell lines (U87, U118, U138, U343, and U373). Levels of activated Ras GTP were measured in these cell lines using a 32P loading assay. Twenty operative GBM specimens were evaluated for levels of activated Ras GTP using a novel luciferase-based assay. The relevance of Ras activation in cell lines was evaluated using genetic (dominant negative Ras-N17) and pharmacological means (treatment with the farnesyl transferase inhibitor L-739, 749). Activation of downstream mitogenic pathways (MAPK) was evaluated using a myelin basic protein (MBP) kinase assay.

The mean level of Ras GTP in the operative tumor specimens was 1.62 ± 0.62 fmol/5 g DNA, compared to levels of only 0.08 and 0.04 fmol/5 g DNA in two nonneoplastic head injury specimens used as controls. Levels of Ras GTP in the five astrocytoma cell lines were similar to levels in v-H-Ras-transformed murine fibroblasts (RT8 cells), with approximately 30% of Ras being in the activated (GTP-bound) form in these cells. Nontransformed human astrocytes also demonstrated lower levels of Ras GTP (11.2% ± 1.1% of Ras in GTP-bound form). Direct sequencing of H-Ras and K-Ras in these five astrocytoma cell lines confirmed the absence of oncogenic Ras mutations to explain the high levels of Ras GTP observed. Expression of the truncated EGF-R p140-EGF-R in U118 cells resulted in higher levels of constitutive Ras GTP, correlating with a twofold proliferative advantage following 14 days in tissue culture. When Ras activity was inhibited using Ras-N17, proliferation and colony formation in soft agar were reduced to less than 50% of control cells, correlating with 50% reductions in Ras GTP levels and 50% reductions in MAPK activity. Pharmacological Ras inhibition in U87 cells using the farnesyl transferase inhibitor L-739, 749 resulted in 48% reduction in proliferation (p = 0.0063 by paired t-test), even at low doses (10^{-6} M) over short periods (12 days treatment).

These experiments confirm the relevance of Ras-mediated signaling in the molecular pathogenesis of GBMs. Those GBMs expressing the truncated constitutively activated EGF-R p140-EGF-R demonstrate even higher levels of Ras activation. Activation of the Ras pathway is critical in the proliferation of these tumors, as evidenced by genetic manipulation (using Ras-N17) as well as pharmacological inhibition (with L-739, 749). While oncogenic mutations of Ras are not present in astrocytomas, the overexpression of surface receptors results in functionally important activation of the Ras pathway, making such tumors potentially amenable to novel pharmacological agents which specifically target the Ras pathway.
We report our long-term experience with endoscopic transsphenoidal resection of pituitary tumors in 172 patients followed for a mean of 3.1 years. Our technique makes use of 4-mm diameter nasal endoscopes (Nos. 00, 300, 700, 1200) to define a surgical approach which is through a 10-mm septal incision and is transnasal, transseptal (uniseptal dislocation), and transsphenoidal. Using video-endoscopy (3 chip-camera) and functional endoscopic sinus surgery instruments, we expose the sphenoid sinus including the sella turcica. Endoscopic surgery allows a well-illuminated, magnified panoramic view of the sphenoid cavity and sellar contents. Tumor removal, particularly in the suprasellar and lateral sellar/juxta cavernous sinus location, is carried out under direct vision using 2-mm endoscopes.

Radical tumor removal was possible in 85% of pituitary macroadenomas with suprasellar extension (101 of 172 patients). Normalization of hormonal levels was achieved long term in 92% of prolactinomas, 84% of growth hormone-producing microadenomas, and 83% of ACTH-producing microadenomas. Eight patients had transient diabetes insipidus, 2 had postoperative CSF rhinorrhea, and 2 required evacuation of postoperative intrasellar hematomas. The average postoperative stay was 2 days in 85% of patients. Endoscopic transsphenoidal resection of pituitary tumors is now our technique of choice in the treatment of pituitary tumors.
Michael Hsiao, DVM, PhD, is a graduate of National Taiwan University School of Veterinary Medicine. He did his graduate study in the Department of Pathology at the University of Southern California where he obtained his PhD degree in 1991. From 1991 to 1996, he worked with Martin Haas, PhD, in the Department of Tumor Biology at the University of California, San Diego, to study T-cell leukemia. In 1995, he joined the laboratory of Molecular Neurosurgery at Stanford as a postdoctoral fellow to work with Gerald Silverberg, MD, and Victor Tse, MD, PhD, on gene therapy in brain tumor.

Dr. Hsiao’s current interest is in vector design and development, and he is collaborating with Dr. Tse to study the molecular biology of brain tumor, particularly in the field of tumor establishment and angiogenesis.
The aim of this study was to explore the bystander effect of wt-p53 transfection of glioblastoma cell growth suppression. A p53-mediated bystander effect was described while we were investigating the functions of the p53 gene in suppressing rat RT2 glioblastoma tumor growth in situ. This bystander effect of the p53 gene showed promise compared to apoptosis and cell cycle arrest, particularly since high transfection or infection efficiency is very difficult to achieve using the vectors currently available.

An RT2 glioblastoma model was established. Subcutaneous RT2 tumors were intratumorally challenged with p53/liposome complexes and the pattern of vascular growth studied. The results showed a 51% reduction of tumor growth by the p53 gene compared to vector control. There was an 81% reduction in arterial branches to the p53 infected tumors and a 75% reduction in capillary density within the tumors. Further, there was a 95% reduction in lung metastases in the p53-treated group compared to controls. One hundred percent of the mice survived more than 50 days after p53/liposome injections. In comparison, all of the mice challenged with vector/liposome died within 15 days. p53 transgene expression was found in only about 20% of the tumor cells. However, significant necrosis was found at the center of the tumors and no apoptosis detected after injecting p53/liposome complexes.

Our results demonstrated that the p53 transfection/expression by the intratumoral injections of p53/liposome complexes resulted in significant tumor suppression and prolonged animal survival. We show in these experiments that inhibition of angiogenesis triggers the bystander effects leading to massive necrosis, reduced growth, and inhibition of metastasis.
This study aimed to determine the morbidity and mortality in patients with skull base meningiomas treated with an approach that combines aggressive surgery with radiosurgery or confocal radiotherapy for residual tumor. Treatment results were analyzed in 100 consecutive patients with skull base meningiomas managed by one surgeon with a median follow-up time of 5 years. Treatment principles were: to observe an asymptomatic tumor; to operate if the tumor was growing or becoming symptomatic unless surgery was medically contraindicated or refused by the patient; to make surgery as aggressive as possible but with the goal of preserving full function of the patient; and to use radiosurgery or confocal radiation therapy if residual tumor was demonstrated. Preoperative, postoperative, and observational data were prospectively accumulated and stored in a database system. Median follow-up time was 5 years, with a range from 2 to 10 years. The presentation was subtle in these tumors, emphasizing the importance of low-risk management. The most frequent presenting symptoms were headache (44%) and changes in visual acuity (29%). Cranial nerve deficits (31%) and cerebellar signs (23%) were the most common physical findings. Seventy-one patients had surgical resection. In these, 93% had greater than 50% resection and 47% had radiographically complete resection. There
were no perioperative deaths and 5 surgical complications for a rate of 7%. Complications included hemiparesis (2.8%), new cranial nerve palsy (2.8%), and indolent osteomyelitis (1.4%). Nineteen patients had observation only; none progressed. Ten patients had radiation only, primarily because of patient preference or medical contraindications to surgery in the setting of substantial symptoms. There were no complications of this therapy. With a median 5-year follow up, only one patient (1%) demonstrated tumor progression using the treatment paradigm outlined here. These results demonstrate that skull base meningiomas which require treatment can be managed with a combination of aggressive surgery and confocal radiation with an acceptable functional status in 99% of cases. This is strikingly better than the published results of radical surgery for these lesions.

PAPER #854 WEDNESDAY, 4:40-4:50 PM
Effects of Combined GM-CSF and IL-2 in the Treatment of Rat 9L Glioma
Walter Jean
Stephen Spellman
Florian Merkle
Christine Flores
Lance Dela Barre
Michael Garwood
Walter Hall
Walter C. Low (Minneapolis, MN)

KEY WORDS: gliosarcoma, immunological therapy, lymphocyte

Previous data from our laboratory suggested that granulocyte-macrophage colony-stimulating factors (GM-CSF), in the presence of irradiated tumor cells, may increase the survival of rats with intracerebral 9L glioma. In the present study, we postulated that a treatment using combined GM-CSF and IL-2 would increase antitumor efficacy by stimulating both the “helper” and “cytotoxic” arms of the immune system.

One million (10^6) 9L gliosarcoma cells were stereotactically implanted in the right striatum of syngeneic Fischer 344 rats. At the same time, osmotic minipumps (Alzet) were implanted in the flank, delivering either saline, GM-CSF (10 ng/day), or GM-CSF and IL-2 (105 IU/day) for 28 days (n = 6 per group). Irradiated 9L cells (6000 rads 137Cs) were injected subcutaneously on Days 0, 3, 7, 14, and 21. Cured animals were rechallenged 4 months later, receiving 10^6 9L cells in the left striatum. Specific anti-9L tumor memory was also assessed by delayed-type hypersensitivity assay, in which 10^6 irradiated 9L cells were injected into the right pinna. The subsequent swelling was compared to that in the left pinna, which was injected with saline. A separate cohort of animals received intracerebral 9L tumors, saline, or combination GM-CSF/IL-2 treatment, and irradiated tumor cell injections as above (n = 10 per group). Intracerebral tumor growth was monitored by MRI at routine intervals. Selected animals were then sacrificed for the histological assessment of brain tumor infiltration by immune system cells.

Half of the animals treated with GM-CSF and IL-2 survived more than 5 months, whereas all control animals died by Day 28 (Fisher exact test, p < 0.001). Those treated with GM-CSF alone had an intermediate survival rate. Cured animals survived intracerebral tumor rechallenge, whereas naive control animals (n = 3) died by Day 20 (Fisher exact test, p < 0.01). In GM-CSF/IL-2 cured rats, delayed-type hypersensitivity swelling measured at 13.4 ± 0.94 × 10^4 inches ± SEM, compared with 0.97 ± 1.26 × 10^4 inches in controls (p < 0.005). Brain MRI was utilized to correlate antitumor response and histological data. OX-62+ dendritic cells were found within the shrinking tumors of GM-CSF/IL-2 treated animals, whereas they were completely absent from tumors in controls and treatment nonresponders. The CD8+ cytotoxic lymphocyte infiltration into the tumors of responders was markedly enhanced in comparison to expanding tumors. CD4+ lymphocytic and macrophage infiltrations were also more enhanced in shrinking tumors.
These results suggest that, in the presence of irradiated tumor cells, combination treatment with GM-CSF and IL-2 has greater antitumor efficacy than GM-CSF alone. This immunological antitumor response is long lasting, specific, and correlates with intratumoral presence of antigen-presenting dendritic cells.

PAPER #855  WEDNESDAY, 4:50-5:00 PM
TrkA Induction in Glioma Exerts Unique Effects of Tyrosine Kinase Modulation by Downregulating Metalloproteinases and Decreasing Invasion

N. Scott Litofsky
Nilesh Kotecha (Worcester, MA)
Mahesh Lachyanker (Providence, RI)
Alonzo Ross (Shrewsbury, MA)
Lawrence Recht (Worcester, MA)

Key Words: nerve growth factor receptor, metalloproteinases, tyrosine kinase modulation

We have previously established that inducing expression of high-affinity nerve growth factor receptor trkA in C6 rat glioma cells results in less in vivo invasion. Since other growth factor receptors signaling through tyrosine kinase pathways—epidermal growth factor receptor (EGFr) and platelet-derived growth factor receptor (PDGFr)—increase aggressiveness, we compared effects on in vitro invasion and the production of gelatinases A and B, two matrix metalloproteinases (MMP) important in glioma invasion. We hypothesized that trkA induction exerts unique effects of tyrosine kinase modulation by reducing tumor invasion and downregulating MMP activity.

C6 cells were separately transfected with genetic sequence of trkA (C6-trkA), EGFr (C6-EGFr), and PDGFr (C6-PDGFr). MMP activity was assayed by zymography and quantified by densitometry. Tumor invasion was assayed by infiltration through Matrigel in Neuroprobe chambers. C6-trkA was significantly less invasive than all other cell lines (p < 0.003, ANOVA). MMP-9 activity for C6-trkA was comparable to other lines. C6-trkA had significantly less activity for MMP-2 compared to C6 (p < 0.006, ANOVA) but not less than other lines. The ratio of MMP-9 to MMP-2, however, was significantly less for C6-trkA compared to all other cell lines, indicating relative downregulation of MMP-9 in C6-trkA.

We conclude that C6-trkA glioma cells are less invasive than C6 and have relative downregulation of MMP-9. The more invasive nature and lack of MMP-9 downregulation in C6-EGFr and C6-PDGFr indicate that the more benign effects are specific to the trkA receptor, in contrast to usual tyrosine kinase receptor modulation.

PAPER #856  WEDNESDAY, 5:00-5:10 PM
Gene Rearrangements in Malignant Gliomas

John Cowell
Olga Chernova
David Miller
Gene Barnett (Cleveland, OH)

Key Words: tumor suppressor gene, chromosome, gene translocation, glioma

Extensive loss of heterozygosity (LOH) analysis has demonstrated that partial or complete loss of chromosomes 10q and 19q occurs in the vast majority of malignant gliomas. These observations indicate that these regions contain glioma-associated tumor suppressor genes. However, identification of these genes is complicated by the large size of the deleted region. Another important mechanism resulting in gene inactivation is through chromosome
translocation, which disrupts the genes located at the breakpoints. Thus, these translocation breakpoints pinpoint the position of the critical tumor suppressor gene within the region of LOH.

We have identified glioblastoma cells that carry a reciprocal t(10;19)(q24;q13) translocation with breakpoints in both critical regions. Using FISH analysis, yeast artificial chromosomes have been positioned across the breakpoints on both rearranged chromosomes. The exact position of the translocation breakpoints have now been identified using somatic cell hybrids, and bacterial artificial chromosomes which cross the chromosome 10 breakpoint have been isolated. A gene we have called “GBM1” has been isolated from this BAC. Expression studies show that this gene is normally only expressed in human brain and muscle and, while expressed in most low-grade brain tumors, is inactivated in many glioblastomas. Following cytogenetic analysis of primary tumor cells, another tumor has been identified where the GBM1 gene is translocated into chromosome 11. As a result of this translocation, the GBM1 gene is inactivated. This highly specific pattern of inactivation implies a role for this gene in the development of advanced-stage brain tumors.

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**THE FARBER LECTURE**

AANS/CNS Section on Tumors

5:10 PM – 5:30 PM (Room 114)

**A MOLECULAR BASIS FOR THE “INTRINSIC RADIRESISTANCE” OF ASTROCYTIC TUMORS**

Mark Israel, MD (San Francisco, CA)
(To be introduced by Peter McL. Black, MD, PhD)

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**BUSINESS MEETING**

AANS/CNS Section on Tumors

5:30 PM – 6:00 PM (Room 114)
Thursday, April 30, 1998

BREAKFAST SEMINARS
6:45–9:30 AM

Breakfast—Grand Hall
6:45–7:30 AM

Seminars
7:30–9:30 AM

#401 POSTERIOR CIRCULATION ANEURYSMS
Room 105B

Moderator:
Neal Kassell, MD (Charlottesville, VA)

Panelists:
Duke Samson, MD (Dallas, TX)
Neil Martin, MD (Los Angeles, CA)*
Michael Horowitz, MD (Dallas, TX)
Jacques J. Marcos, MD (Miami, FL)

#402 TECHNIQUES FOR CEREBRAL REVASCULARIZATION
Room 107B

Moderator:
Laligam Sekhar, MD (Washington, DC)

Panelists:
Fernando G. Diaz, MD (Detroit, MI)
R. Michael Scott, MD (Boston, MA)
Takanori Fukushima, MD (Pittsburgh, PA)
Philip Stieg, MD, PhD (Boston, MA)
#403 MANAGEMENT OF AVMs
Room 111B

Moderator:
H. Hunt Batjer, MD (Chicago, IL)

Panelists:
Adam Lewis, MD (Jackson, MI)
L. N. Hopkins, MD (Buffalo, NY)*
Neil Martin, MD (Los Angeles, CA)
L. Dade Lunsford, MD (Pittsburgh, PA)*

#404 PEDIATRIC BRAIN TUMORS
Room 105A

Moderator:
Harold Hoffman, MD (Toronto, ON)

Panelists:
Karin Muraszko, MD (Ann Arbor, MI)*
Ian Pollack, MD (Pittsburgh, PA)
Maurice Choux, MD (Marseilles, France)

#405 INTRACRANIAL FRAMELESS STEREOTAXIS:
PRINCIPLES AND TECHNIQUES
Room 202B

Moderator:
David Roberts, MD (Lebanon, NH)*

Panelists:
Richard Bucholz, MD (Saint Louis, MO)*
Robert Maciunas, MD (Nashville, TN)
Gene Barnett, MD (Cleveland, OH)*
Robert Goodman, MD (New York, NY)

#406 PALLIDOTOMY FOR PARKINSON’S DISEASE
Room 110B

Moderator:
Roy A.E. Bakay, MD (Atlanta, GA)*

Panelists:
Ronald R. Tasker, MD (Toronto, ON)
Robert P. Iacono, MD (Loma Linda, CA)
Marwan Hariz, MD (Umea, Sweden)
#407  LUMBAR STENOSIS: CURRENT TREATMENT
AND LONG TERM OUTCOME
Room 102A/B

Moderator:
Russell Travis, MD (Lexington, KY)

Panelists:
John Jane, MD (Charlottesville, VA)
Fred Simeone, MD (Philadelphia, PA)
Philip Weinstein, MD (San Francisco, CA)
Nancy Epstein, MD (New York, NY)

#408  MANAGEMENT OF CERVICAL DISC DISEASE:
STATE OF THE ART
Room 203B

Moderator:
Charles Fager, MD (Burlington, MA)

Panelists:
Dennis Vollmer, MD (San Antonio, TX)
W. Michael Vise, MD (Jackson, MS)
Scott Shapiro, MD (Indianapolis, IN)
Hae-Dong Jho, MD (Pittsburgh, PA)*

#409  DIAGNOSIS AND MANAGEMENT OF FORAMINAL
AND FAR LATERAL LUMBAR DISC HERNIATIONS
Room 203A

Moderator:
Robert Hood, MD (Salt Lake City, UT)

Panelists:
Joseph Maroon, MD (Pittsburgh, PA)
W. Robert Hudgins, MD (Dallas, TX)
Maurice Smith, MD (Memphis, TN)*
Nicolas DeTribolet, MD (Lausanne, Switzerland)
#410 EVALUATION AND MANAGEMENT OF PENETRATING INJURIES TO THE CNS
Room 110A

Moderator:
Barth Green, MD (Miami, FL)

Panelists:
Robert Heary, MD (Upper Montclair, NJ)
Michael Gerson, MD
Brian Andrews, MD (San Francisco, CA)

#411 CRANIAL NERVE PRESERVATION IN ACOUSTIC TUMOR SURGERY
Room 112B

Moderator:
Peter Jannetta, MD (Pittsburgh, PA)

Panelists:
Madjid Samii, MD (Hannover, Germany)
Kalmon Post, MD (New York, NY)
Stephen Haines, MD (Charleston, SC)
William Buchheit, MD (Philadelphia, PA)

#412 TUMORS OF THE CLIVUS AND FORAMEN MAGNUM
Room 112A

Moderator:
Jon H. Robertson, MD (Memphis, TN)

Panelists:
Ossama Al-Mefty, MD (Little Rock, AR)
Chandranath Sen, MD (New York, NY)
Jeffrey Bruce, MD (New York, NY)
Paul Chapman, MD (Boston, MA)
#413 CURRENT AND NOVEL TREATMENT OF MALIGNANT GLIOMAS
Room 104B

Moderator:
Henry Brem, MD (Baltimore, MD)*

Panelists:
Edward Oldfield, MD (Bethesda, MD)
Andrew Kaye, MD (Melbourne, Australia)
Ivan Ciric, MD (Evanston, IL)
Douglas Laske, MD (Bala Cynwyd, PA)*

#414 THIRD VENTRICULAR TUMORS
Room 202A

Moderator:
Michael L.J. Apuzzo, MD (Los Angeles, CA)

Panelists:
Ernst Grote, MD (Tubingen, Germany)
Alan Cohen, MD (Cleveland, OH)
Gazi Yasargil, MD (Little Rock, AR)
Joao Lobo Antunes, MD (Lisbon, Portugal)

#415 ABNS BOARD PREPARATION
Room 104A

Moderator:
Donald Becker, MD (Los Angeles, CA)

Panelists:
Fremont P. Wirth, MD (Savannah, GA)
Robert Wilkins, MD (Durham, NC)
John Van Gilder, MD (Iowa City, IA)
Thursday Morning

SPECIAL COURSE I

9:45 AM – 12:00 NOON  (Room 113A/B/C)

COMPUTER TECHNOLOGY FOR NEUROSURGEONS

Moderator: Robert E. Harbaugh, MD (Lebanon, NH)

NEUROSURGERY://ON-CALL®, Past, Present, and Future
John Oro, MD (Columbia, MO)

Telemedicine
Julian Bailes, Jr., MD (Maitland, FL)

Computer Assisted Surgery
Richard Bucholz, MD (St. Louis, MO)*

The Virtual Meeting
Joel MacDonald, MD

Virtual Reality for Surgical Training
Joseph Rosen, MD (Hanover, NH)

Microprocessors and Other Possibilities
Grant Shumaker, MD (Lebanon, NH)

On-line Outcome Studies
Robert E. Harbaugh, MD (Lebanon, NH)

What Computers Can’t Do
Robert E. Harbaugh, MD (Lebanon, NH)
Thursday Morning

**SPECIAL COURSE II**

9:45 AM – 12:00 NOON   (Room 103A/B/C)

**NEUROENDOSCOPY: INNOVATIONS AND CONTROVERSIES**

**Moderator:** Alan R. Cohen, MD (Cleveland, OH)

**Introduction**  
Alan R. Cohen, MD (Cleveland, OH)

**Endoscopic Treatment of Ventricular Cysts**  
Enrique Ferrer, MD (Barcelona, Spain)

**What’s New in Hydrocephalus**  
Alan R. Cohen, MD (Cleveland, OH)

**Advances in Endoscopic Treatment of Ventricular Tumors**  
Michael Gaab, MD (Hanover, Germany)*

**Endoscopic Pituitary Surgery Update**  
Carl B. Heilman, MD (Boston, MA)

**Endoscope-Assisted Microneurosurgery**  
Axel Perneczky, MD (Mainz, Germany)

**Overview of Endoscopic Spinal Surgery**  
Curtis Dickman, MD (Phoenix, AZ)*
Thursday Morning

**SPECIAL COURSE III**

9:45 AM – 12:00 NOON     (Room 108A/B)

**SURGICAL NEURO-ONCOLOGY—HOW I DO IT**

**Moderator:** James T. Rutka, MD (Toronto, ON, Canada)

**Awake Craniotomy for Brain Tumors**  
Mark Bernstein, MD (Toronto, ON, Canada)

**Frameless Stereotactic Resection of Thalamic Tumors**  
Patrick Kelly, MD (New York, NY)

**Surgical Approaches to Ventricular Tumors**  
Joseph Piepmeier, MD (New Haven, CT)

**Surgical Approaches to Brainstem Tumors**  
James T. Rutka, MD (Toronto, ON, Canada)

**Surgical Approaches for Difficult Meningiomas**  
Ossama Al-Mefty, MD (Little Rock, AR)

**Neurosurgical Strategies for Pituitary Tumors**  
Edward Oldfield, MD (Bethesda, MD)

**Surgery for Brain Metastases**  
Raymond Sawaya, MD (Houston, TX)

**HSV-TK/Ganciclovir Gene Therapy for Recurrent Glioblastoma: Results of an International, Multicenter Study**  
Mark Bernstein, MD (Toronto, ON, Canada)
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