THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

83RD ANNUAL MEETING

SEPTEMBER 22-25, 2021

American Association of Neurological Surgeons

Jointly Provided by the AANS

FUTURE MEETINGS

September 28 – October 1, 2022
Broadmoor Hotel
Colorado Springs, Colorado

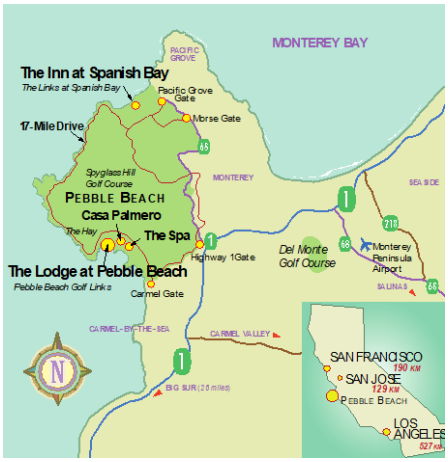
October 4-7, 2023
The Cloister at Sea Island
Sea Island, Georgia

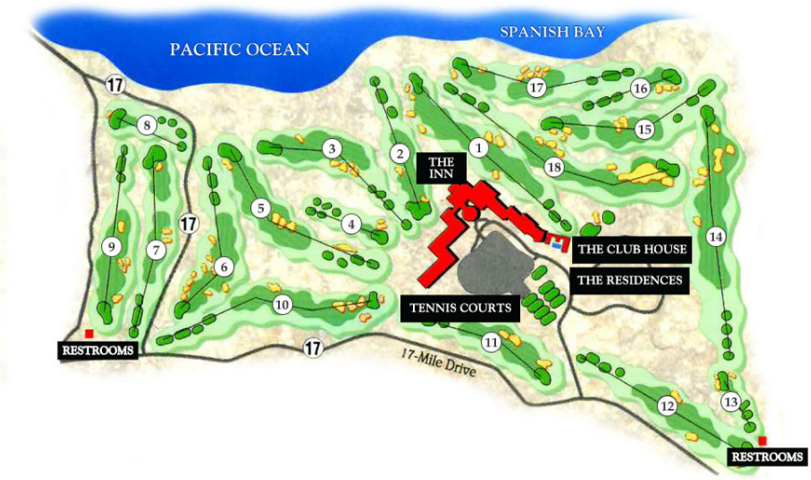
Mark your calendars now!

General Information

Hotel Information

THE INN AT SPANISH BAY
2700 17-Mile Drive, Pebble Beach, CA 93953
(831) 574-5605





Registration Location:	**www.americanacademyns.org**

Registration:

On-site Opens Wednesday, September 22, 2021

Complete form on website. Email inquiries directly to Eden@voilameetings.com

A Special Thanks to the following exhibitors supporting the

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
83RD ANNUAL SCIENTIFIC MEETING

Please take time to visit with them during the Break

- BrainLab/DePuySynthes
- Elekta
- Hyperfine
- Integra LifeSciences
- Leica Microsystems
- Synaptive
- Zap Surgical
- Carl Zeiss Meditec, US

WEDNESDAY, SEPTEMBER 22

1:00 – 6:30 pm	**Registration**	Group Hospitality Desk by Main Ballroom
3:30 – 5:00 pm	**Executive Committee Meeting**	Muirfield
6:30 – 8:30 pm	**Opening Reception**	Fire Pit Terrace

THURSDAY, SEPTEMBER 23

6:00 am – 4:00 pm	**Registration**	Group Hospitality Desk by Main Ballroom
6:30 – 7:30 am	**Members Breakfast & Business Meeting** (Voting Membership Only)	St. Andrews E/W
8:00 – 10:00 am	**Guest & Spouse/Partner Breakfast**	Peppoli Restaurant/Lawn
7:30 – 7:35 am	**Welcoming Remarks**	Main Ballroom
7:35 – 7:45 am	**Round Robin Roundup!**	Main Ballroom
7:45 – 8:50 am	**Peer Reviewed Abstract Session I:** Select Reports - Basic and Clinical	Main Ballroom
8:50 – 9:45 am	**Peer Reviewed Abstract Session II:** Brain Function and Restoration	Main Ballroom
9:45 – 10:00 am	Break	Expo Space
10:00 – 10:55 am	**Peer Reviewed Abstract Session III:** Spine Science	Main Ballroom
10:55 – 11:50 am	**Peer Reviewed Abstract Session IV:** Tumor Biology & Treatment	Main Ballroom
11:50 am – 12:05 pm	Break	Expo Space
12:05 – 12:45 pm	**Presidential Address**	Main Ballroom
12:30 – 1:30 pm	**Boxed Lunches for Golfers**	Spanish Bay Golf Course
1:30 – 4:30 pm	**Academy Spine Emerging Investigators' Program**	St. Andrews E/W

6:30 – 9:30 pm	**Dinner**	Beach Club

FRIDAY, SEPTEMBER 24

6:00 am – 4:00 pm	**Registration**	Group Hospitality Desk by Main Ballroom
6:30 – 7:30 am	**Members Breakfast & Business Meeting** (Voting Membership Only)	St. Andrews E/W
8:00 – 10:00 am	**Guest & Spouse/Partner Breakfast**	Peppoli Restaurant/Lawn
7:30 – 7:35 am	**Welcoming Remarks**	Main Ballroom
7:35 – 8:30 am	**Peer Reviewed Abstract Session V:** Neurosurgical Trials: From Concept to Completion	Main Ballroom
8:30 – 9:25 am	**Peer Reviewed Abstract Session VI:** Vascular Science	Main Ballroom
9:25 – 9:35 am	Break	Expo Space
9:35 – 10:00 am	**Past Presidential Address**	Main Ballroom
10:00 – 11:05 am	**Peer Reviewed Abstract Session VII:** Novel Technologies & Approaches	Main Ballroom
11:05 – 11:20 am	Break	Expo Space
11:20 am – 12:15 pm	**Peer Reviewed Abstract Session VIII:** Epilepsy & Functional	Main Ballroom
12:15 – 12:50 pm	**Peer Reviewed Abstract Session IX:** Vascular Practice	Main Ballroom
12:00 – 12:30 pm	**Boxed Lunches for Golfers**	Spanish Bay Golf Course
1:30 – 4:30 pm	**Joint Academy Emerging Investigators' Program**	St. Andrews E/W
6:00 – 6:30 pm	**Cocktail Reception**	Fairway Patio
6:30 – 9:30 pm	**Gala Dinner (Black Tie Optional)**	Main Ballroom & Fairway Patio

SATURDAY, SEPTEMBER 25

7:00 – 12 pm	**Registration**	Group Hospitality Desk by Main Ballroom
7:00 – 9:30 am	**Members & Guests Breakfast**	Peppoli Restaurant/Lawn

7:30 – 8:20 am	**Special Abstract Session:** The Oldfield Session	Main Ballroom
8:20 – 9:10 am	**Academy Award Presentation and Lecture**	Main Ballroom
9:10 – 9:55 am	**Peer Reviewed Abstract Session X:** First in Human	Main Ballroom
9:55 – 10:10 am	Break	Expo Space
10:10 – 11:15 am	**Peer Reviewed Abstract Session XI:** Skull Base and Vascular	Main Ballroom
11:15 am – 12:30 pm	**Peer Reviewed Abstract Session XII:** Trauma and Various Topics	Main Ballroom
12:30 pm	**Closing Remarks & Meeting Adjourn**	Main Ballroom

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

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Christopher Loftus, MD – Second Delegate

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Robert Gross, MD
Amy Heimberger, MD

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Spence Braden	1940
Joseph P. Evans	1941
Francis Murphey	1942
Frank H. Mayfield	1943
A. Earl Walker	1944
Barnes Woodhall	1946
William S. Keith	1947
Howard A. Brown	1948
John Raaf	1949
E. Harry Botterell	1950
Wallace B. Hamby	1951
Henry G. Schwartz	1952
J. Lawrence Pool	1953
Rupert B. Raney	1954
David L. Reeves	1955
Stuart N. Rowe	1956
Arthur R. Elvidge	1957
Jess D. Herrmann	1958
Edwin B. Boldrey	1959
George S. Baker	1960
C. Hunter Shelden	1961 - 62
Samuel R. Snodgrass	1963
Theodore Rasmussen	1964
Edmund J. Morrissey	1965
George Maltby	1966
Guy L. Odom	1967
James G. Galbraith	1968
Robert H. Pudenz	1969 - 70
William B. Scoville	1971
Robert L. McLaurin	1972
Lyle A. French	1973
Benjamin B. Whitcomb	1974
John R. Green	1975
William H. Feindel	1976
William H. Sweet	1977
Arthur A. Ward	1978
Robert B. King	1979
Eben Alexander, Jr.	1980
Joseph Ransohoff II	1981
Byron C. Pevehouse	1982
Sidney Goldring	1983
Russel H. Patterson, Jr	1984
Thomas Langfitt	1985
Phanor L. Perot, Jr	1986
Shelley N. Chou	1987
James T. Robertson	1988
Thoralf M. Sundt, Jr.	1989
Robert Ojemann	1990
Nicholas Zervas	1991
Henry Garretson	1992
George Tindall	1993
William A. Buchheit	1994
David L. Kelly, Jr	1995
John M. Tew, Jr	1996
Julian T. Hoff	1997
Edward Connolly	1998
J. Charles Rich	1999
George A. Ojemann	2000
Roberto C. Heros	2001
Donald O. Quest	2002
David G. Piepgras	2003
Volker K.H. Sonntag	2004
Martin B. Camins	2005
L. Nelson Hopkins	2006
Richard Morawetz	2007
Robert F. Spetzler	2008
Ralph G. Dacey, Jr.	2009
Steven Giannotta	2010
Robert A. Solomon	2011
James T. Rutka	2012
Griffith R. Harsh	2013
Fredric B. Meyer	2014
Mitchel S. Berger	2015
Mark N. Hadley	2016
William T. Couldwell	2017
Daniel L. Barrow	2018
E. Antonio Chiocca	2019
M. Sean Grady	2020

PAST VICE-PRESIDENTS

Francis Murphey	1941	Griffith R Harsh, III	1986
William S. Keith	1942	Ellis B Keener	1987
John Raaf	1943	Robert Grossman	1988
Rupert B. Raney	1944	Jim Story	1989
Arthur R. Elvidge	1946	John Jane, Sr.	1990
F. Keith Bradford	1949	Stewart Dunsker	1991
David L Reeves	1950	Burton M Onofrio	1992
Henry G. Schwartz	1951	Martin H Weiss	1993
J. Lawrence Pool	1952	John M. Tew, Jr.	1994
Rupert B. Raney	1953	John C. VanGilder	1995
David L. Reeves	1954	Edward Connolly	1996
Stuart N. Rowe	1955	George Ojemann	1997
Jess D. Hermann	1956	Charles H. Tator	1998
George S. Baker	1957	Donald O. Quest	1999
Samuel R. Snodgrass	1958	Howard M. Eisenberg	2000
C. Hunter Shelden	1959	Richard B. Morawetz	2001
Edmund Morrissey	1960	Martin B. Camins	2002
Donald F. Coburn	1961 - 62	Arthur L. Day	2003
Eben Alexander, Jr.	1963	William F. Chandler	2004
George L Maltby	1964	Steven L. Gianotta	2005
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Francis A. Echlin	1966	Griffith R. Harsh IV	2007
Benjamin Whitcomb	1967	Daniel L. Barrow	2008
Homer S. Swanson	1968	M. Sean Grady	2009
Augustus McCravey	1969 - 70	Warren Selman	2010
Edward W. Davis	1971	Jeffrey Bruce	2011
John R. Green	1972	James Drake	2012
George J. Hayes	1973	Corey Raffel	2013
Richard L. DeSaussure	1974	Alan R. Cohen	2014
Ernest W. Mack	1975	Michael T. Lawton	2015
Frank E. Nulsen	1976	James M. Markert, Jr.	2016
Robert S. Knighton	1977	Robert Harbaugh	2017
Robert G. Fisher	1978	Nelson M. Oyesiku	2018
H Thomas Ballantine, Jr.	1979	Mark Johnson	2019
George Ehni	1980	Matthew Howard III	2020
Courtland H. Davis, Jr.	1981		
John F. Mullan	1982		
Hugo V. Rizzoli	1983		
James W Correll	1984		
E. Bruce Hendrick	1985		

PAST SECRETARY-TREASURERS

Francis Murphey	1938 - 40
A. Earl Walker	1941 - 43
Theodore C. Erickson	1944 - 47
Wallace B. Hamby	1948 - 50
Theodore B. Rasmussen	1951 - 53
Eben Alexander	1954 - 57
Robert L. McLaurin	1958 - 62
Edward W. Davis	1963 - 65
Robert G. Fisher	1966 - 68
Byron C. Pevehouse	1969 - 72

PAST SECRETARIES

Byron C. Pevehouse	1973
Russel H. Patterson, Jr	1974 - 1976
Phanor L. Perot, Jr	1977 - 1980
John T. Garner	1981 - 1983
James T. Robertson	1984 - 1986
Nicholas T. Zervas	1987 - 1989
William A. Buchheit	1990 - 1992
Julian T. Hoff	1992 - 1995
Roberto C. Heros	1995 - 1998
David G. Piepgras	1999 - 2001
L. Nelson Hopkins	2002 - 2004
Ralph G. Dacey, Jr	2005 - 2007
James Rutka	2008 - 2010
Mitchel S. Berger	2011 - 2013
Daniel L. Barrow	2014 - 2017
James M. Markert, Jr.	2018 –2020

PAST TREASURERS

Russel H. Patterson, Jr.	1973
Phanor L. Perot, Jr	1974 - 1976
John T. Garner	1977 - 1980
James T. Robertson	1981 - 1983
Nicholas T. Zervas	1984 - 1986
William A. Buchheit	1987 - 1989
Julian T. Hoff	1990 - 1992
Roberto C. Heros	1992 - 1995
David G. Piepgras	1996 - 1998
L. Nelson Hopkins	1999 - 2001
Ralph G. Dacey, Jr.	2002 - 2004
James T. Rutka	2005 - 2007
Griffith Harsh	2008 - 2010
Daniel L. Barrow	2011 - 2013
E. Antonio Chiocca	2014 – 2017
Douglas Kondziolka	2018 – 2020

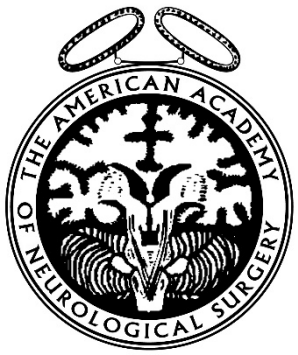
Oldfield Lecture

Russell Lonser	2018
Amy Heimberger	2019

Hotel Netherland Plaza, Cincinnati, Ohio	October 28 - 29, 1938
Roosevelt Hotel, New Orleans, Louisiana	October 27 - 29, 1939
Tudor Arms Hotel, Cleveland, Ohio	October 21 - 22, 1940
Mark Hopkins Hotel, San Francisco, California	November 11 - 15, 1941
Ambassador Hotel, Los Angeles, California	November 11 - 15, 1941
The Palmer House, Chicago, Illinois	October 16 - 17, 1942
Hart Hotel, Battle Creek, Michigan	September 17 - 18, 1943
Ashford General Hospital, White Sulphur Springs, West Virginia	September 7 - 9, 1944
The Homestead, Hot Springs, Virginia	September 9 - 11, 1946
Broadmoor Hotel, Colorado Springs, Colorado	October 9 - 11, 1947
Windsor Hotel, Montreal, Canada	September 20 - 22, 1948
Benson Hotel, Portland, Oregon	October 25 - 27, 1949
Mayo Clinic, Rochester, Minnesota	September 28 - 30, 1950
Shamrock Hotel, Houston, Texas	October 4 - 6, 1951
Waldorf-Astoria Hotel, New York City, New York	September 29 - October 1, 1952
Biltmore Hotel, Santa Barbara, California	October 12 - 14, 1953
Broadmoor Hotel, Colorado Springs, Colorado	October 21 - 23, 1954
The Homestead, Hot Springs, Virginia	October 27 - 29, 1955
Camelback Inn, Phoenix, Arizona	November 8 - 10, 1956
The Cloister, Sea Island, Georgia	November 11 - 13, 1957
The Royal York Hotel, Toronto, Canada	November 6 - 8, 1958
Del Monte Lodge, Pebble Beach, California	October 18 - 21, 1959
Copley Sheraton Plaza, Boston, Massachusetts	October 5 - 8, 1960
Royal Orleans, New Orleans, Louisiana	November 7 - 10, 1962
El Mirador, Palm Springs, California	October 23 - 26, 1963
The Key Biscayne, Miami, Florida	November 11 - 14, 1964
Terrace Hilton Hotel, Cincinnati, Ohio	October 14 - 16, 1965
Fairmont Hotel & Towers, San Francisco, California	October 17 - 19, 1966
The Key Biscayne, Miami, Florida	November 8 - 11, 1967
Broadmoor Hotel, Colorado Springs, Colorado	October 6 - 8, 1968
St. Regis Hotel, New York City	September 21, 1969

Camino Real, Mexico City, Mexico	November 18 - 21, 1970
Sahara-Tahoe Hotel, Stateline, Nevada	September 26 - 30, 1971
New College, Oxford, England	September 4 - 7, 1972
Huntington-Sheraton Hotel, Pasadena, California	November 14 - 17, 1973
Southampton Princess Hotel, Bermuda	November 6 - 9, 1974
The Wigwam (Litchfield Park), Phoenix, Arizona	November 5 - 8, 1975
Mills Hyatt House, Charleston, South Carolina	November 10 - 13, 1976
Mauna Kea Beach Hotel, Kamuela, Hawaii	November 2 - 5, 1977
Hotel Bayerischer Hof, Munich, Germany	October 22 - 25, 1978
Hyatt Regency, Memphis, Tennessee	November 7 - 10, 1979
Waldorf-Astoria Hotel, New York City, New York	October 1 - 4, 1980
Sheraton Plaza, Palm Springs, California	November 1 - 4, 1981
Ritz-Carlton Hotel, Boston, Massachusetts	October 10 - 13, 1982
The Lodge at Pebble Beach, California	October 23 - 26, 1983
The Homestead, Hot Springs, Virginia	October 17 - 20, 1984
The Lincoln Hotel Post Oak, Houston, Texas	October 27 - 30, 1985
The Cloister, Sea Island, Georgia	November 5 - 8, 1986
Hyatt Regency, San Antonio, Texas	October 7 - 10, 1987
Omni Netherland Plaza, Cincinnati, Ohio	September 13 - 17, 1988
Loews Ventana Canyon, Tucson, Arizona	September 27 - October 1, 1989
Amelia Island Plantation, Amelia Island, Florida	October 2 - 7, 1990
Salishan Lodge, Gleneden Beach, Oregon	September 22 - 26, 1991
Ritz-Carlton Hotel, Naples, Florida	October 21 - 25, 1992
The Wigwam, Phoenix, Arizona	October 27 - 30, 1993
The Cloister, Sea Island, Georgia	November 3 - 6, 1994
Loews Ventana Canyon Resort, Tucson, Arizona	November 1 - 5, 1995
The Greenbrier, White Sulphur Springs, WV	September 18 - 22, 1996
Rimrock Resort, Banff, Alberta, Canada	September 10 - 14, 1997
Four Seasons Biltmore, Santa Barbara, California	November 4 - 7, 1998
Ritz-Carlton, Amelia Island, Florida	November 10 - 13, 1999
The Broadmoor, Colorado Springs, Colorado	October 11 - 14, 2000
The Breakers, Palm Beach, Florida	November 14 - 17, 2001
The Phoenician, Scottsdale, Arizona	October 16 - 19, 2002

Colonial Williamsburg, Williamsburg, Virginia	October 29 - November 1, 2003
Four Seasons Berlin & Hotel Taschenbergpalais, Dresden, Germany	October 3 - 8, 2004
Ritz-Carlton, Half Moon Bay, California	September 21 - 24, 2005
Ritz-Carlton, Reynolds Plantation, Greensboro, GA	October 18 - 21, 2006
Ritz-Carlton, Lake Las Vegas, Nevada	October 31 - November 3, 2007
Barrow Neurological Institute Phoenix; Enchantment Resort, Sedona Arizona	September 10 - 13, 2008
The Breakers, Palm Beach, Florida	November 4 - 7, 2009
The Inn at Spanish Bay, Pebble Beach, California	November 3 - 6, 2010
The Fairmont Scottsdale Princess, Scottsdale, AZ	October 19 - 22, 2011
The Chatham Bars Inn, Chatham, Massachusetts	October 17 - 20, 2012
The Resort at Pelican Hill, Newport Coast, CA	September 25 - 28, 2013
WaterColor Inn & Resort, Santa Rosa Beach, FL	September 17 - 20, 2014
Hotel Europäischer Hof, Heidelberg, Germany	October 7 - 10, 2015
Four Seasons Resort, Jackson Hole, Wyoming	September 14 - 17, 2016
Four Seasons Santa Barbara, Santa Barbara, CA	September 13 – 16, 2017
The Breakers, Palm Beach, Florida	October 24 – 27, 2018
Rome Cavalieri Waldorf Astoria, Rome, Italy	September 18-21, 2019
Virtual	September 26, 2020



MISSION STATEMENT

The purpose of the Academy meeting shall be to promote scientific and social interaction among its members, to foster neurological surgery as a specialty of medicine, to encourage and sponsor basic and clinical research activity in the neurological sciences, and to promote the knowledge and skill of those who devote themselves to neurological surgery in accordance with the high ideals of the medical profession.

This activity will include live presentations from faculty to include case presentations and discussion, as well as time for questions and answers.

THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

American Association of Neurological Surgeons
Jointly Provided by the AANS

LEARNING OBJECTIVES

- Describe the implications of modern genomics for brain tumor diagnosis, vascular disorders, and errors of metabolism
- Discuss the evolution of spinal techniques for management of cervical and lumbar pathology based on randomized trials
- Identify novel applications for therapeutic devices in the nervous system, including intravascular therapy for stroke, hemorrhage, and hydrocephalus, and functional surgery applications beyond movement disorders.
- Define the impact of new imaging technology for both pre-operative and intra-operative management of neurosurgical disorders.

ACCREDITATION STATEMENT

This activity has been planned and implemented in accordance with the accreditation requirements and policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of the AANS and the American Academy of Neurological Surgery. The AANS is accredited by the ACCME to provide continuing medical education for physicians.

DESIGNATION STATEMENT

The AANS designates this live activity for a maximum of 15 *AMA PRA Category 1 Credits*™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Link for CME reporting will be sent to you via email following the meeting.

DISCLOSURE STATEMENT

Before the program, anyone in control of the educational content of this activity will disclose the existence of any financial interest and/or the relationship they or their significant other have with the manufacturer(s) of any commercial product(s) to be discussed during their presentation. Disclosures are included in the final program.

INTENDED AUDIENCE/BACKGROUND REQUIREMENT

The scientific program presented is intended for neurosurgeons either in training or in active practice.

AANS JOINT PROVIDERSHIP DISCLAIMER STATEMENT

The material presented at the 81st Annual Meeting of the American Academy of Neurological Surgery has been made available by the American Academy of Neurological Surgery and the AANS for educational purposes only. The material is not intended to represent the only, nor necessarily the best, method or procedure appropriate for the medical situations discussed, but rather it is intended to present an approach, view, statement, or opinion of the faculty, which may be helpful to others who face similar situations.

Neither the content (whether written or oral) of any course, seminar or other presentation in the program, nor the use of a specific product in conjunction therewith, nor the exhibition of any materials by any parties coincident with the program, should be construed as indicating endorsement or approval of the views presented, the products used, or the materials exhibited by the American Academy of Neurological Surgery and jointly provided by the AANS, or its Committees, Commissions, or Affiliates.

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

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Adam Arthur	Consultant	Balt
Nicholas Boulis	Consultant Contracted Research Ownership Interest – Future Stock Options Stockholder	Sio Gene Therapy, UCB Pharma, BlueRock Bio, Neurogene PTC CODA Bio, Nipro LLC, Renew Bio Braintrust Bio
Fady T. Charbel	Consultant	Transonic Inc., Stryker Inc.
E. Antonio Chiocca	Consultant Ownership Interest – Future Stock Options Patent Holder/Royalty	Voyager Therapeutics, DNAtrix, GSK, Biogen, Insightec, Candel Therapeutics Seneca Therapeutics, DNAtrix, Immunomic Therapeutics Candel Therapeutics
Kevin Cockroft	Contracted Research	IAC grant
William Curry	Received Fees for Non-CME/CE Services	IMRIS (Board of Directors)
Michael Fehlings	Ownership Interest – Future Stock Options	Inteligex Inc.
Richard Fessler	Consultant Ownership Interest – Own the Company	Orthofix, DePuy-Synthes In Queue Innovations

Name	Type of Disclosure	Entity/Company
Paul Gardner	Contracted Research Ownership Interest – Future Stock Options Royalty Stockholder	Gleolan SPIWay LLC Peter Lazic, US, Inc. Renerva
Zoher Ghogawala	Ownership Interest – Future Stock Options	NIDUS
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Brian Hoh	Research Support Stockholder	AstraZeneca, Janssen Proprio Vision, Progressive Neuro, Galaxy Therapeutics
Matthew Howard	Stockholder Patent Holder Research Support	Direct Spinal Therapeutics Inc. KLS Martin NIH
Douglas Kondziolka	Industry Grant Support Stockholder	Brainlab Chiefy
Peter Konrad	Stockholder	Neurotargeting LLC
Ilya Laufer	Consultant Royalty	Medtronic Globus, SpineWave
Michael Lawton	Consultant Royalty	Zeiss Mizuho
Kendall Lee	Consultant Ownership Interest – Own the Company	Medtronic Navinetics Inc.
Allan Levi	Speakers Bureau	AANS / Medtronic
L. Dade Lunsford	Consultant Stockholder	Insightec AB Elekta
Andre Machado	Consultant Patent Holder	Abbott, Enspire DBS Enspire DBS, Cardionomics
William Mack	Consultant Ownership Interest – Future Stock Options Stockholder	Imperative Care, Interga Stream Biomedical, Spartan Micro, Q'Apel Cerebrotech, Viseon, Truvic, Rebound Therapeutics, Endostream
Geoffrey Manley	Industry Grant Support Research Support	Abbott Laboratories NIH, DOD
James Markert	Ownership Interest – Own the Company	Treovir LLC, Aettis Inc.
J Mocco	Stockholder	Synchron

Name	Type of Disclosure	Entity/Company
Jacques Morcos	Consultant Stockholder	Leica Kogent
Peter Nakaji	Royalty Stockholder	OsteoMed GT Medical Technologies
Shahid Nimjee	Stockholder	Basking Biosciences Inc.
John O'Toole	Consultant	Globus Medical
Daniel Resnick	Ownership Interest – Future Stock Options	NIDUS
Howard Riina	Consultant Stockholder	Medtronic eClips Neuro
John Sampson	Consultant Patent Holder Stockholder	Medicenna, Annias Immunotherapeutics, Insera Health, Shorla, Creosalus, Annias Immunotherapeutics, Neuronium, Istari Oncology Annias Immunotherapeutics, Istari Oncology, Nueronium, Immunomic Therapuetics, Insera Health
Luis Savastano	Patent Holder/Stockholder	Endovascular Engineering
Daniel Sciubba	Consultant Ownership Interest – Future Stock Options Ownership Interest – Own the Company	Depuy-Synthes, Stryker, Baxter Augmedics BioPhy
Christopher Shaffrey	Consultant Patent Holder Stockholder	NuVasive, Medtronic, and SI Bone NuVasive, Medtronic, Zimmer Biomet, and SI Bone NuVasive
Khoi Than	Consultant Speakers Bureau	Bioventus, Integrity Implants, DePuy Synthes LifeNet Health, Globus
Nicholas Theodore	Consultant Royalty Stockholder	Globus Medical, Misonix Globus Medical, Depuy Synthes Globus Medical
Craig van Horne	Consultant	BrainLab, Boston Scientific
Michael Vogelbaum	Contracted Research Consultant Patent Holder	Celgene Inc., Olympus Inc., Oncosynergy Inc. Cellinta Inc., Plus Therapeutics Inc. Infuseon Therapeutics Inc.

Name	Type of Disclosure	Entity/Company
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	Patent Holder	DePuy-Synthes Spine
	Royalty	Depuy-Synthes
	Stockholder	Kinesiometrics, ISD, Medical Device Partners,

Those who have reported that they do not have any relationships with commercial interests:

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Ted Godfrey	Atlanta, GA	Elekta
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Rob Korn	Guilford, CT	Hyperfine
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Patrick Morse	San Carlos, CA	Zap
Richard Rosene	San Carlos, CA	Zap
James Carter	Dublin, CA	Zeiss
Chris Danko	Dublin, CA	Zeiss

WEDNESDAY, SEPTEMBER 22, 2021

REGISTRATION AND RECEPTION

THURSDAY, SEPTEMBER 23, 2021

7:30 – 7:35 WELCOMING REMARKS

Sepideh Amin-Hanjani, MD

7:35 – 7:45 Round Robin Roundup! The Academy Round Robin Letters, 1939-2021

Frederick G. Barker, MD

7:45 – 8:50 Peer Reviewed Abstract Session I: Select Reports – Basic and Clinical
Moderators: Sepideh Amin-Hanjani & James Rutka

7:45 – 7:55 The Potential Role of Trigeminal Nerve Stimulation in TBI and SAH

Raj K. Narayan, MD; Chunyan Li; Timothy G White, MD; Kevin Shah, MD; Keren Powell

Introduction
Hypotension is associated with substantially worse outcomes in patients with severe TBI and delayed cerebral ischemia (DCI) worsens outcomes after aneurysmal SAH. Pharmacological strategies to treat either have not been very effective. The trigeminal nerve richly innervates cerebral blood vessels via connections to brainstem nuclei. CGRP is a key vasodilatory molecule secreted by trigeminal nerve endings.
Objective
To assess the ability of percutaneous trigeminal nerve stimulation (TNS) to improve outcome measures in animal models of TBI and SAH.
Methods
In male Sprague Dawley rats, a controlled cortical impact (CCI) model of severe TBI was used, and animals were divided into 1) Sham. 2) Delayed fluid resuscitation 3) Immediate fluid resuscitation and 4) Low frequency oscillation at 0.1Hz.
In another set of experiments, an endovascular perforation model was used to induce SAH. Biphasic electrical pulses were delivered to the infraorbital nerve over a 60-minutes. 48 hours after SAH induction, cerebrospinal fluid was drawn for calcitonin gene-related peptide (CGRP) measurement.
Results
TNS induced CBF oscillations conferred significant protection to the pericontusional areas in TBI with reductions in hypoxic brain injury, neuroinflammation and lesion volume, leading to better neurological outcomes.

In the SAH model, TNS increased CGRP levels substantially and increased luminal diameters of the ICA, MCA, and ACA as compared to SAH-control and sham rats. SAH-control rats demonstrated a 4.9-fold increase in microthrombi, compared to sham rats, with a 2.5-fold decrease with TNS.
TNS induced CBF oscillations conferred significant protection to the pericontusional areas in TBI with reductions in hypoxic brain injury, neuroinflammation and lesion volume, leading to better neurological outcomes.
Conclusion
TNS may be a novel therapeutic approach to both TBI and aneurysmal SAH.

7:55 – 8:05 The International Tuberculum Sellae Meningioma Study Surgical Outcomes and Grading Scale Refinement: Final Results

Michael William McDermott, MD; Stephen Magill

Introduction
Tuberculum sellae meningiomas (TSM) surgical outcomes were studied in 40 participating centers. In addition, with a larger data set we refined and simplified an earlier grading system for predicting visual outcomes and EOR based on surgical approach.
Objectives
To report TSM management trends, peri-operative outcomes and recurrence rates after TCA or TSA approaches and refinements to a previously reported grading system.
Methods
We conducted a 40-site retrospective study on 947 patients with TSM using standard statistical methods to evaluate EEA or TCA approaches. Validation and refinement cohorts were used to simplify the grading system.
Results
Of 947 cases, a TCA was used in 629 (66.4%), a TSA in 318 (33.6%). Use of TSA has increased from 2003-2019 and is currently used in 50% of cases. Median follow up was 26 months (0-265 months), and 63 (6.6%) were WHO grade II/III. Gross total resection (GTR) was achieved in 70.0% of cases and did not differ between TSA (68.2%) and TCA (71.2%) (OR 1.2 for TCA, 95%CI 0.9-1.5, p=0.3667). On MVA, GTR was less likely in tumors with greater maximum diameter (OR 0.8 per cm, 95%CI 0.7-0.9) and those with pre-operative visual deficit (OR 0.6, 95%CI 0.4-0.9). Mortality was 0.5%. Complications occurred in 23.9%. New unilateral or bilateral blindness occurred in 3.3% and 0.4%, respectively. CSF leak rate was stable from 2007-2019, at 17.3% for TSA and 2.2% for TCA (OR 9.1, 95%CI 5.0-16.8). The previously published grading scale was significantly prognostic for visual outcome and EOR in the validation and refinement cohort ($p<0.001$). Data in the refinement cohort supported a simplified grading scale defined by: tumor-score 1 (< 17mm diameter) or 2 (>= 17mm diameter), canal-score 1 (no optic canal invasion) or 2 (any optic canal invasion), and artery-score 1 (abutting < 180 degrees) or 2 (encasing arteries > 180 degrees), which resulted in equivalent prognostic performance for vision (delta-AUC 0.02, p=0.08) and EOR (delta-AUC 0.005, p=0.55) compared to the published scale. The validation cohort validated the simplified grading scale for visual worsening (OR 1.69 per point increase, 95%CI 1.31-2.18, $p<0.001$) and EOR (OR 1.61 for STR per point, 95%CI 1.38-1.88, $p<0.001$). The simplified grading scale remained independently prognostic after adjusting for clinical covariates including age, size, WHO grade and approach.
Conclusion
The use of TSA for TSM is increasing and trends toward better visual outcomes and significantly decreased recurrence rates after GTR. We refined and validated a simplified grading scale for predicting visual outcome and EOR based on TSM pre-operative tumor characteristics.

8:05 – 8:15 Infantile Treatment with Erythropoietin Plus Melatonin Mitigates Gait Deficits in Adult Rats with Perinatal Brain Injury

Shenandoah Robinson, MD; Lauren Janzie

Introduction

Only ~half of children with cerebral palsy (CP) are diagnosed before 2 years of age, and thus many miss the neonatal window for treatment. Lack of effective interventions for CP drives families to seek unproven, potentially dangerous treatments. We tested whether infantile treatment with erythropoietin (EPO) and melatonin (MLT) mitigated deficits from perinatal brain injury (PBI).

Objectives

We hypothesized that infantile EPO+MLT treatment would restore gait in adult rats with PBI.

Methods

Pregnant dams underwent laparotomy on embryonic day 18 with transient uterine artery occlusion followed by lipopolysaccharide injection. Shams underwent same duration of anesthesia with laparotomy only. On postnatal day 1 (P1), PBI pups (both sexes) were randomized to EPO+MLT or vehicle treatment (P15-P20), and coded. Computerized, digital gait analyses were performed. Group differences were tested for normality, and compared with two-way ANOVA or Kruskal-Wallis with posthoc corrections; $p<0.05$ was considered significant.

Results

Vehicle-treated PBI rats (n=10) exhibited an abnormal gait reminiscent of CP, compared to shams (n=39) and EPO+MLT-treated PBI rats (n=7). Compared to sham or EPO+MLT-treated PBI rats, vehicle-treated PBI rats showed more stride length variation (both $p<0.003$), increased stride frequency (both $p<0.03$), reduced swing duration (shams, $p=0.003$; EPO+MLT-treated rats, $p=0.02$), and more ataxia (both $p<0.003$).

Conclusion

Improved gait after EPO+MLT treatment in late infancy suggests that pharmacotherapies can alter motor abilities well after the neonatal window. Intervention beyond the neonatal period using widely available repurposed medications may be beneficial in infants at risk for CP. These data emphasize early diagnosis and treatment considerations for infants at risk of lifelong neurological deficits.

8:15 – 8:25 Chronic Subdural Hematomas Following Middle Meningeal Artery Embolization: Factors Associated with Resolution

Felipe Albuquerque, MD; Joshua Catapano, MD; Andrew F. Ducruet, MD

Introduction

Middle meningeal artery (MMA) embolization for the treatment of Chronic subdural hematomas (cSDHs) is associated with a decrease in treatment failure compared to conventional therapies. However, literature remains scarce on variables associated with improved outcomes following MMA embolization.

Objectives

The present study analyses radiographic outcomes after MMA embolization for cSDHS and variables associated with hematoma resolution at 90-days post-embolization.

Methods

A single quaternary center's patients who underwent a MMA embolization for a cSDH from 1/1/2018 to 12/31/2020 were retrospectively analyzed. Patients with a 90-day follow-up scan were included in the study. Radiographic outcome at 90-days post-embolization was analyzed, with a complete and/or near complete resolution (<5 mm on axial CT head) as the primary outcome. A univariate analysis for factors associated with 90-day resolution was performed. A subsequent stepwise multivariable logistic regression analysis for

variables predictive of resolution at 90-days was performed for all factors with a p-value <0.2 on univariate analysis.

Results

Of 76 patients who underwent a MMA embolization, 58 patients (81%) were found to have a follow-up scan at 90-days post-embolization. In the 58 patients included in the study, a total of 72 cSDHs were embolized (14 patients with bilateral hemorrhages). There was one complication (1%) reported (a CVA in a type 3 arch that was performed via femoral access and early in the study) and 3 (4%) cSDHs required surgical rescue. At 90-days, 45 cSDHs (63%) were resolved and/or nearly resolved. On univariate analysis: distal penetration, combined anterior and posterior embolization, Charlson Comorbidty Index <5, and no pre-embolization anticoagulant/antiplatelet medications were factors associated with p-values <0.2 for complete and/or near complete resolution at 90-days post-embolization. On stepwise multivariable logistic regression analysis, only distal penetration was found to be associated with resolution at 90-days (OR 5.0, 95% CI 1.7-14.6, p=0.003).

Conclusion

MMA embolization for cSDHS is safe and effective. Distal embolic penetration appears to be associated with an increase frequency of resolution at 90-day follow-up.

8:25 – 8:35 Genomics of 785 Cases of Trigeminal Neuralgia Type 1

Kim J. Burchiel, MD; Ze've Seltzer; Olga Korczeniewska; Scott Diehl

Introduction

Trigeminal Neuralgia (TN) is a rare but very debilitating condition characterized by sudden onset of severe reoccurring orofacial pain in episodes that may last minutes or longer. Pain can be triggered by a light touch or wind to the face, tooth brushing or other mild stimuli that are not normally painful. Episodes can also occur spontaneously without an obvious trigger. Available treatments have limited effectiveness and recurrence is common.

Objectives

To identify common and/or rare genetic variants that affect risk of Trigeminal Neuralgia.

Methods

In this multicenter study, subjects were recruited at six locations in the U.S. and in the UK and Canada. The case-control GWAS study included 1,017 reference controls and 785 cases diagnosed as classical or idiopathic TN (with or without neurovascular compression of the trigeminal nerve, excluding patients with constant orofacial pain). The cross-sectional discovery (DNA sequencing) study included a subset of 100 TN patients from the larger GWAS sample. The primary outcome was association of TN risk with common DNA polymorphisms and rare variants predicted to damage protein function in biologically relevant pathways. A sub-analysis was performed focused on female patients with early onset at age 45 or younger because this subgroup less often presents with trigeminal nerve neurovascular compression compared to male patients of any age or older female patients, thus suggesting a potentially unique etiology.

Results

Genome-wide statistically significant associations were found at polymorphisms in KCNK10, LIG3, NFAT2 and XRCC4 genes in analyses of all patients (Figure 1) and the BDNF gene in female patients with early onset (Figure 2). DNA sequencing analyses revealed 349 rare, potentially causative protein-damaging mutations in 182 genes important for the nervous system or associated with human neurological disorders or pain. Six patients had mutations in TRPM4 and five patients had mutations in DOCK3, DYNC1H1 or the sodium channel genes SCN10A or SCNN1B. Female patients with onset by age 45 had significantly more mutated genes involving myelin or Charcot-Marie-Tooth disease (P=0.004) and ion channel genes (P=0.03) compared to older females or males regardless of age of onset (Figure 3).

Conclusion

Our data demonstrate that TN is a complex and heterogeneous disorder whose risk is influenced by both common polymorphisms and rare mutations. Female patients with early onset may constitute an etiologically distinct subtype of TN with unique genetic risk factors. The pathways identified suggest targets for development of improved analgesics and other therapeutic approaches for treatment of pain.

8:35 – 8:45 A Pro-oncogenic Lentiviral Swine Model of Spinal Cord Glioma

Nicholas M. Boulis, MD; Mohib Tora

Introduction

The current literature does not describe well-characterized topical large mammalian models of spinal cord glioma (SCG) for use in pre-clinical neurosurgical studies. Prior work has applied driver mutations targeting the RTK/RAS/PI3K and p53 pathways to induce the formation of high-grade gliomas in rodent models. The present study reports our efforts at modeling high-grade SCG in the minipig using lentiviral gene transfer.

Objectives

Despite significant morbidity and mortality, there is no consensus treatment strategy, especially for high-grade lesions. Currently, there is a weak level of evidence in the literature (Class IIb, level of evidence C) which largely relies on expert opinion and case series. While surgical debulking is the initial mainstay of treatment in supratentorial GBM, this proves incredibly difficult in the spinal cord where the entire bulk of the parenchyma is eloquent tissue. The compact architecture of the spinal cord and its associated tracts and nerves, and the infiltrative growth pattern of these tumors lead to the decision by most neurosurgeons to avoid aggressive resection of these tumors. We undertook the creation of a large animal model for spinal cord glioma to facilitate the development of novel immunotherapeutic and surgical strategies for treatment.

Methods

Six Gottingen Minipigs received thoracolumbar (T14-L1) lateral white matter injections of a combination of lentiviral vectors, expressing platelet-derived growth factor beta (PDGF-B), constitutive HRAS, and shRNA-p53. Animals underwent baseline and endpoint magnetic resonance imaging (MRI) and were evaluated daily for clinical deficits. Hematoxylin and eosin (H&E) and immunohistochemical (IHC) analysis was conducted and comparisons of the tumor core and leading edge. Data are presented using descriptive statistics including relative frequencies, mean, standard deviation. Statistical comparisons between tumor core and leading edge were conducted using two-way ANOVA and Tukey's Post-Hoc, where $P < 0.05$ considered statistically significant (Prism Graphpad 9, San Diego, CA).

Results

100% of animals (n = 6/6) developed quantifiable clinical motor deficits ipsilateral to the oncogenic lentiviral injections by a pre-determined 3-week endpoint. MRI scans demonstrated contrast enhancing mass-forming lesions at T-14-L1. Neuropathologic features demonstrate consistent and reproducible growth of a high-grade glioma with astrocytic morphology in all animals. Ki-67 index was highly immunopositive across all tumors, with a mean of 37.1% (SD: 14.2). The tumors were grossly immunopositive for SOX2, Olig2, and NG2, and were immunonegative for PDGFRA. We observed statistically significant differences in Ki-67, SOX2, Olig2, and NG2 ($P<0.001$) immunopositivity in comparing the tumor core and leading edge, but not PDGFRA. RNA-sequencing and gene-set enrichment analysis demonstrated statistically significant enrichment of mesenchymal and classical glioma subtypes using Verhaak, Neftel, and Suva gene sets ($P<0.05$) and several hallmark pathways.

Conclusion

Utilization of vector driven gene transfer offers a feasible pathway to glioma modeling in large mammalian models. The present minipig model is the first vector induced pig model of high-grade SCG and may potentially be used in pre-clinical neurosurgical development programs.

8:45 – 8:50	**Wrap-up/ Transition**

8:50 – 9:45	**Peer Reviewed Abstract Session II: Brain Function and Restoration** Moderators: Aviva Abosch & Matthew Howard

8:50 – 9:00	**Time Cells in the Human Hippocampus During Episodic Memory Processing**

Bradley Charles Lega, MD

Introduction

The representation of temporal information is a key feature of episodic memory, a form of mnemonic processing sensitive to aging, traumatic brain injury, and Alzheimer's Disease. A key insight from Eichenbaum, Buzsaki, and others is that the representation of time in the brain utilizes similar mechanisms as spatial association. This led to the discovery of 'time cells' in rodents, which fulfill a key role in episodic memory analogous to place cells during spatial navigation. However, it they have not previously been reported in humans.

Objective

Test for the presence of time cells, and a related population termed `ramping cells,' in the human MTL. Connect the activity of these cells with behavioral features of episodic memory processing.

Methods

We used microelectrode recordings from 27 surgical epilepsy patients who performed free recall, a standard assay of episodic memory. We isolated single units using the Combinato package, and analyzed the resulting spike matrices to test for two classes of time sensitive cells. We developed a novel metric to characterize the consistency of time cell firing.

Results

Using both non-parametric KW test and a general linear modeling approach, we identified time cells during both encoding and retrieval, and we linked the consistent firing of time cells to memory behavior. We also identified ramping cells following the methods of Moser.

Conclusion

Time cells and ramping cells support episodic representations in the human MTL. Our findings establish a mechanism predicted from rodent models and computational modeling.

9:00 – 9:10	**Initial Experience of a Transvascular Brain Machine Interface to Restore Function to Patients with Severe Paralysis**

J D. Mocco, MD; Nick Opie; Peter Yoo; James Bennett; Peter Mitchell; Christin Bird; Andrew Morokoff, MBBS, PhD; Thomas Oxley

Introduction

Advances in brain-machine interfaces (BMIs) have enabled people with severe paralysis to control external equipment with their thoughts. However, efforts requiring a craniotomy have been complicated by risks of infection and hemorrhage, as well as signal degeneration due to gliosis/encapsulation. A transvascular minimally invasive BMI may mitigate the above risks.

Objectives

To evaluate preliminary safety and efficacy experienced by the first four participants implanted with a novel transvascular BMI.

Methods

Four participants with severe paralysis were implanted over 16 months. The electrode array was placed within the superior sagittal sinus adjacent to the pre- and primary-motor cortex. A subcutaneous unit transmitted electrocorticographic activity to an external processor, enabling communication to a standard personal computer. Safety was evaluated by recording of all Serious Adverse Events (SAEs), as well as evaluation on CTV at three and twelve months for any evidence of device migration, stenosis, or thrombosis. Preliminary efficacy was evaluated by assessment of the patients' ability to independently use the technology at home (y/n) and an assessment of typing speed (correct characters per minute, CCPM) and accuracy at twelve months.

Results

All patients were successfully implanted. No serious adverse events occurred. CTV imaging at three and twelve months demonstrated no evidence of device migration, stenosis, or thrombosis. Independent home use of the system was achieved by all participants within 7-36 days following device activation. Using the device, all participants were able to enhance digital communication, perform online shopping, and engage with financial management tools. Two patients reached the twelve-month typing assessment, with typing speeds and accuracies of 13.81 CCPM at 92.6% and 20.1 CCPM at 93.2%.

Conclusion

Preliminary experience suggests that transvascular BMI placement is safe and may enable severely paralyzed participants to independently improve at-home functionality.

9:10 – 9:20 Deep Cerebellar Stimulation for Post-stroke Motor Rehabilitation: Human Translation

André Machado, MD PhD; Alexandria Wyant; Raghavan Gopalakrishnan, PhD MBA; Ela Plow; K Baker

Introduction

Our group has proposed DBS of the dentatothalamocortical pathway as a novel approach to promote post-stroke motor rehabilitation. We have shown in preclinical models that DBS of the dentate nucleus (DN) enhances perilesional cortical excitability, functional reorganization and synaptogenesis.

Objectives

We present the preliminary results of a prospective phase-I clinical trial that represents the first-in-man translation of this emerging therapy.

Methods

Patients with moderate to severe hemiparesis between one- and three-years post MCA stroke were enrolled. All patients underwent implantation of DBS in the DN contralateral to stroke. Physical therapy (PT) was administered for three months prior to turning DBS ON. Thereafter, DBS was programmed followed by 4 months of PT combined with DN-DBS. Response to treatment was defined as a post-DBS gain of 5-points in the Fugl-Meyer assessment of the upper extremity (FMA-UE). Event-related EEG and local field potentials (LFPs) were assessed perioperatively.

Results

Twelve patients (eight men) with FMA-UE < 30 have been implanted without serious complications. Ten patients have completed the study, seven of whom are classified as therapeutic responders. Among patients who presented with at least minimal distal motor function at baseline, the mean FMA-UE improvement was 10 points and associated with significant functional gains. We found significant movement-related modulation of DN LFPs in a topography-specific fashion. Finally, DN-DBS was associated with cortical area and frequency-specific modulation of movement-related EEG.

Conclusion

DN-DBS has been safe and feasible in this phase-I study. Preliminary outcomes and electrophysiological findings are encouraging and support a multicenter RCT to evaluate safety and efficacy.

9:20 – 9:30 How We Speak

Kiefer Forseth; **Nitin Tandon, MD**

Introduction
Production of even the simplest words rely on distributed brain networks. The translation of conceptual knowledge to an articulatory plan engages these theoretical networks but there is scant empirical evidence of the underlying neural mechanisms.
Objectives
Our objective is to fill a major gap in our understanding of aphasia and in the development of relevant therapeutics.
Methods
We studied word production at an unprecedented scale (134 patients; 25,810 electrodes) in epilepsy patients undergoing direct intracranial recordings. A surface-based mixed-effects multilevel analysis enabled the creation of a finely resolved spatiotemporal atlas of language cortex. We derived single-trial sequences of network state dynamics using an autoregressive hidden Markov model to distinguish cognitive states defined by causal interactional motifs. Next, we disrupted language via direct cortical stimulation to disrupt language to assess the criticality of nodes within the networks. Stimulation induced depolarization was transformed onto the pial surface via current spread models to generate subject-specific and then population maps.
Results
We produced the first comprehensive high-resolution 4D-representation of brain activity during word production and derived a grouped dynamical model to resolve five discrete neural states distinguished by unique patterns of distributed cortical interaction. In addition to the canonical language sites, the mid fusiform cortex and the superior frontal sulcus were critical to word production, as evidenced by direct stimulation. Middle-fusiform gyrus dominated information outflow during a conceptualization state while the superior-frontal sulcus contributed predominantly to a word formulation state.
Conclusion
This large-scale multimodal population-level analysis, expands the use of intracranial recordings from spatiotemporal descriptors of neural processes to the evaluation of the possible mechanistic foundations creating a new framework for the understanding of language disorders and possible interventions to address them.

9:30 – 9:40 A Speech Neuroprosthesis for Decoding Words in a Person with Severe Paralysis

Edward Chang MD

Introduction
Technology to restore communication for paralyzed patients who have lost the ability to speak has the potential to improve autonomy and quality of life. Decoding words and sentences directly from the neural activity of a paralyzed individual who cannot speak may be an improvement over existing methods for assisted communication.
Objectives
To decode words directly from the neural activity of a paralyzed individual who cannot speak.
Methods
We implanted a high-density, subdural multi-electrode array over the speech motor cortex of a person with anarthria, the loss of the ability to articulate speech, and spastic quadriparesis caused by brainstem stroke. Across 48 sessions, we recorded 22 hours of cortical activity while the participant attempted to say individual words from a 50-word vocabulary. Using deep learning, we created computational models to detect and classify words from patterns in the recorded cortical activity. We applied these models and a language model, which describes how frequently certain word sequences occur in natural language, to decode full sentences as he attempted to say them.

Results
We decoded sentences from the participant's cortical activity in real time at a median rate of 15 words per minute with a median word error rate of 26%. In post-hoc analyses, we detected 98% of individual word production attempts and classified words with 47% accuracy using cortical signals that were stable throughout the 81-week study period.
Conclusion
In a person with anarthria caused by brainstem stroke, we used machine learning and a natural language model to decode words and sentences directly from cortical activity as the person attempted to speak. We demonstrate successful real-time word and sentence decoding from the speech cortex of a paralyzed person using a clinically viable long-term neural interface.

9:40 – 9:45 Wrap-up/ Transition

9:45 – 10:00 Break

10:00 – 10:55 Peer Reviewed Abstract Session III: Spine Science
Moderators: Ziya Gokoslan & Daniel Resnick

10:00 – 10:10 Prediction of Outcome in Cervical Incomplete Spinal Cord Injury using Machine Learning Group-Based Trajectory Analysis

Michael G. Fehlings, MD, PhD ; Jetan Badhiwala; Jefferson R. Wilson, MD, PhD; James S. Harrop, MD; Bizhan Aarabi, MD ; Robert G. Grossman, MD

Introduction
The outcomes of incomplete cervical spinal cord injury (SCI) are heterogeneous and difficult to predict using conventional approaches.
Objectives
To accurately predict the outcome of cervical incomplete SCI patients based on longitudinal temporal profiles of recovery in upper limb motor function.
Methods
Patients with cervical incomplete SCI (AIS B-D; C1-C8) were identified from a prospective, multi-center dataset. A group-based trajectory model was fit to upper extremity motor scores assessed out to 1 year. Multivariable multinomial logistic regression was performed to characterize recovery trajectories. A prediction model using baseline clinical data was developed by recursive partitioning.
Results
801 patients were eligible. Four distinct trajectory groups were identified (Fig 1,2):

"Poor outcome": Severe injury, very minimal recovery
"Moderate recovery": Moderate-to-severe injury, moderate recovery
"Good recovery": Moderate injury, good recovery
"Excellent outcome": Mild injury, recovery to normal/near-normal

On adjusted analyses (Table 1), older age was associated with lower likelihood of "excellent outcome" (P=0.020). AIS C and D SCI were associated with "moderate recovery", "good recovery", and "excellent outcome" (P<0.001). Mid-cervical injuries occurred more frequently in "moderate recovery", "good recovery", and "excellent outcome" (P<0.001) groups. Early surgical decompression (<24 hrs) was associated with increased propensity for "good recovery" (P=0.039) and "excellent outcome" (P=0.048). A classification model

based on recursive partitioning could predict trajectory group using age, AIS grade, and neurological level with an AUC of 0.81.

Conclusion

Patients with cervical incomplete SCI demonstrate distinct temporal profiles of recovery in upper limb motor function that can be accurately predicted based on age, AIS grade and neurological level.

10:10 – 10:20 A Critical Reappraisal of Corticospinal Tract Somatotopy and its Role in Traumatic Cervical Spinal Cord Syndromes

Allan D. Levi, MD, PhD; Aditya Vedantam, MD

Introduction

Early studies hypothesized somatotopic (laminar) organization of the cervical CST (Fig 1) with selective injury of the medial positioned hand and arm CST fibers to explain the pathophysiology of CCS.

Objectives

To provide current ex vivo data and new in vivo data with imaging and fiber tract analysis to evaluate selective involvement of the medial CST in central cord syndrome.

Methods

Subjects with traumatic central cord syndrome with T2* or GRE cervical spinal cord MRI were selected for this study. Axial images of the cervical spinal cord were registered to the PAM 50 anatomical spinal cord template using the Spinal Cord Toolbox. White matter/gray matter (WMGM) ratios, a measure of white matter injury, were calculated for the medial and lateral half of the CST in the C3-C6 segments. These ratios were compared to each other for the right and left CST using standard t test.

Results

Data from 1. Non-human primate central nervous system (CNS) tract tracing studies 2. selective ablative studies of the CST in primates 3. evolutionary assessment of the CST in mammals and 4. neuropathological examination of CCS do not support somatotopic localization of the CST. Ten subjects (9 males, mean age 55.6±10.9 years) with central cord syndrome (AIS C=1, AIS D = 9) were included in this study. The neurological level of injury was C4 (n=5), C5 (n=4) and C3 (n=1). Mechanism of injury was motor vehicle crash (n=5), fall from standing (n=3) and fall from height (n=2). The mean admission upper extremity motor score was 27.4±11.8 and the mean lower extremity motor score was 43.4±10.3. Mean WMGM ratio was not statistically different between the medial half and lateral half of the right CST (1.5±0.19 vs 1.51±0.33, p=0.96) or the left CST (medial 1.59±0.25 vs lateral 1.56±0.34, p=0.82) (Fig 2).

Conclusion

In contrast to historical concepts, the current evidence suggests that there is no somatotopic organization of the corticospinal tract within the spinal cord in humans and as well as the critical importance of the CST for hand function (Fig 3). These results further emphasize the need to reappraise prior theories on the selective involvement of medial CSTs in central cord syndrome.

10:20 – 10:30 Identifying Patient Specific Variables Influencing Decisions to Perform Fusion in Grade I Spondylolisthesis

Daniel K. Resnick, MD; Bradley Schmidt; Vikas Kumar Parmar, MD; Zoher Ghogawala, MD, FAANS

Introduction

The role of fusion as an adjunct to decompression in patients with stenosis associated with spondylolisthesis remains an area of controversy. We recently reported significant variation in treatment recommendations regarding fusion from an expert panel using data from the SLIP II study. We found that this variability was due to both surgeon specific and patient specific variables.

Objectives
We sought to explore patient specific variables driving the recommendation for fusion by the expert panel.
Methods
A pilot study using de-identified data from the SLIP II study was performed. Expert panel recommendations were examined and patients whose recommendations reflected a consensus for decompression alone or decompression and fusion were identified. A consensus was defined as >80% agreement among panelists. Five patients were identified where there was >80% consensus for decompression alone and ten patients were identified where there was >80% consensus for decompression and fusion. Each of these 15 cases was then carefully examined to determine which characteristics (from the clinical vignette, imaging, and other patient collected data) most consistently differed between the two cohorts.
Results
With regard to demographic factors, the fusion cohort was younger (60 years vs 67.2 years, $p = 0.077$), and had a lower health state based on EQ-5D scores (0.50 vs 0.68, $p = 0.04$). There was no difference in the ODI between the cohorts (37.2 vs 37, $p=0.49$).
With regard to imaging characteristics, we found that greater degree of spondylolisthesis (24% vs 12%, $p < 0.001$), greater facet angle from horizontal (54 vs 48 degrees $p=0.12$), and movement on dynamic images all predicted a recommendation for fusion.
Conclusion
In examining the subset of patients with consensus from the expert panel regarding the utility of fusion, it is apparent that younger age, lower EQ-5D, sagittally-oriented facets, mobile spondylolisthesis, and degree of anterolisthesis were all associated recommendation for fusion. This information provides significant evidence that particular patient characteristics drive senior surgeon decision making regarding the utility of fusion as an adjunct to decompression in this patient population. This has important policy implications and may help to explain disparate results from contemporary clinical trials.

10:30 – 10:40 Cochrane Analysis: Definitive Statistics or Biased Opinion

Richard G. Fessler, MD, PhD

Introduction
Lumbar spinal stenosis (LSS) is a common degenerative condition among the elderly population and a leading cause of morbidity in this age group. A recent Cochrane analysis reviewed the evidence for surgical vs non-surgical treatment from five prospective, randomized, controlled studies (RCT) and concluded that, "No clear benefits were observed with surgery versus non-surgical treatment". This is despite the fact that all five of the reports analyzed concluded that surgery provided superior outcome compared to non-surgical therapy.
Objectives
This report analyzes, in detail, the Cochrane analysis of Zaina, each of the five studies included in the Zaina analysis, and the Cochrane methodology itself. Unlike the ultimate in objectivity sought after by the creators of the Cochrane tool, what is revealed is a remarkably subjective methodology fraught with the potential for bias.
Methods
Five prospective, randomized, controlled trials analyzed in the report of Zaina1 were reviewed and analyzed using the criteria described in that report, with the modifications suggested by Furlan2 and Higgins3.
Results
Unlike what is widely considered the most objective evaluation of clinical reports, what is revealed is a remarkably subjective methodology, strongly subject to the authors biases. This evaluation strongly suggests that the conclusions of the original published manuscripts cited in the report of Zaina are much more reliable than the conclusions stated in the Zaina Cochrane analysis itself.
Conclusion

Like all published manuscripts, Cochrane analyses must be reviewed with appropriate skepticism. Readers must closely analyze and consider the quality of the analyzed data, the statistics utilized, the review and the Cochrane methodology itself before accepting the validity of the conclusions.

10:40 – 10:50 Minimally Invasive TLIF Results in Less Adjacent Segment Disease and All-Cause Reoperation than Open TLIF Embolization: Factors Associated with Resolution

Barry Cheaney II, BS; Joseph Girard Nugent, MHS, CRT-I; Brittany Stedelin; Diana Ko; Timothy Y. Wang, MD; James T Obayashi, BS; Ahmed M.T. Raslan, MBBS MCh; **Khoi Duc Than, MD**

Introduction

Transforaminal lumbar interbody fusion (TLIF) is well established and traditionally performed as an open (O-TLIF) procedure. To minimize tissue trauma associated with the O-TLIF approach, the minimally invasive (MIS-TLIF) approach was developed.

Objectives

There is a paucity of information regarding reoperations due to adjacent segment disease (ASD) following either TLIF approach. We report on these occurrences in patients who underwent single-level O-TLIF versus MIS-TLIF.

Methods

A propensity score (PS) model was generated to account for the likelihood of receiving an open vs. MIS approach based on surgical indications and comorbidities. A 1-to-1 optimal match was performed using the PS estimated by logistic regression to generate 66 pairs. The outcomes were compared using univariate and multivariate Cox proportional hazards models to generate an adjusted Hazard Ratio (aHR).

Results

A total of 132 PS matched patients (66 pairs) were included in the final analysis. There were no statistically significant differences in patient demographics between the two groups. In the O-TLIF group, there were a total of 29 (43.9%) reoperations, 20 (30.3%) due to ASD. In the MIS-TLIF group, there were a total of 10 (15.2%) reoperations, 2 (3.0%) due to ASD. Kaplan-Meier time-to-event analysis revealed the risk for reoperation due to ASD ($p < 0.0001$) and all-cause reoperation ($p = 0.0008$) were significantly higher in the O-TLIF group among matched pairs. Multivariate Cox proportional hazards analysis revealed that O-TLIF significantly increased the risk of all-cause reoperation (HR 3.20, 95% CI 1.55-6.58, $p = 0.002$) and increased the risk of reoperation due to ASD (HR 11.63, 95% CI 2.71-49.95, $p = 0.001$) after adjustment for confounding factors. There was no statistically significant difference in follow-up time (months) between O-TLIF (33.46 ± 35.17) and MIS-TLIF (38.72 ± 35.45) ($p = 0.393$).

Conclusion

We demonstrate that MIS-TLIF results in less all-cause reoperations, specifically due to ASD, when compared to O-TLIF.

10:50 – 10:55 Wrap-up/ Transition

10:55 – 11:50 Peer Reviewed Abstract Session IV: Tumor Biology & Treatment
Moderators: Zadeh & Markert

10:55 – 11:05 A Call to Improve SRS Dose Guidelines for Patients with Brain Metastases

L. Dade Lunsford, MD; Zhishuo Wei; Hideyuki Kano, MD PhD; Ajay Niranjan, MD MBA

Introduction
Stereotactic Radiosurgery (SRS) is the most widely used strategy for treatment of brain metastases. While many centers still rely on 30-year-old RTOG guidelines, current automated dose planning techniques create "conformal" plans using higher marginal isodoses and low maximal doses.
Objectives
We retrospectively reviewed outcomes in non-small cell lung cancer (NSCLC) patients in order to develop an alternative SRS dose strategy.
Methods
Between 2014 and 2020 330 NSCLC patients (median age= 65 years) with 2908 brain metastases underwent SRS. The total tumor volume ranged from .05 to 33.09 cc (median= 3.17). The median tumor margin dose was 18 Gy.
Results
Median overall survival after SRS was 15.5 months. Sixty-six tumors (2.27%) in 50 patients (14.75%) progressed. For tumors ≤0.25cc local tumor control was 98 % compared to 95% for tumors >0.25 cc (p=0.001). If >65% of >0.25 cc tumor received at least 24 Gy, tumor control increased to 99% (p= 0.009). The risk of adverse radiation effects for tumors that received at least 24 gy to >65% of the tumor was 3.8%, statistically unchanged from the 4.8% rate for tumors treated so that <65% received >24 Gy (p=0.13).
Conclusion
In this study the best local control rates were obtained when >65% of the tumor volume received at least 24 Gy. This planning methodology reduces reliance on strict conformality and prescribed margin doses. In order to ensure that each treated metastasis responds, lower prescription isodoses are necessary to reach the threshold goal of >65% of the volume receiving at least 24 Gy.

11:05 – 11:15 Surgical Management of Incidentally Discovered Low Grade Gliomas

Mitchel S. Berger, MD

Introduction
Although most patients with low grade glioma (LGG) present after a seizure, a small proportion of patients have an imaging diagnosis or a sign or symptom not related to the tumor.
Objectives
To determine the optimal management plan for adult patients with incidentally discovered LGGs.
Methods
Patients were identified from a prospective registry of patients undergoing glioma resection who were considered incidental. Tumor volumes, growth rate and extent of resection were calculated from pre- and post-operative volumetric FLAIR sequences.
Results
113 of 657 (17.2%) first-time resections for low grade glioma were for incidental lesions. Headaches (without mass effect) (34.5%) or trauma (16.8%). Incidental tumors (iLGG) were significantly smaller. The median observation time for iLGG was 3.1 months (range: 1 month - 12 years), and there was a median growth rate was 3.9 cm3/year. Complete resection of the FLAIR abnormality was achieved in 57% of patients with incidental lesions but only 23.8% of symptomatic (sLGG) lesions ($p < 0.001$), and the residual volumes were smaller for iLGGs (2.9 cm3 vs 13.5 cm3, $p < 0.0001$). Overall survival was significantly longer for patients with incidental tumors (median survival not reached for iLGG versus 14.6 years for sLGG, $p < 0.0001$).
Conclusion
Patient age, tumor location, and molecular genetics were not different between iLGG and sLGG. Incidental tumors are smaller than symptomatic tumors, a greater extent of resection can be achieved for iLGG, and overall survival is improved when compared to sLGG.

11:15 – 11:25 Bavituximab Treatment of Newly Diagnosed Glioblastoma Impacts Myeloid Targets

William Curry MD; Leland Richardson; K. Ina Ly; Bryan D. Choi, MD; Elizabeth Gerstner

Introduction

Glioblastoma and tumor endothelial cells express phosphatidylserine, an immunosuppressive membrane phospholipid. Bavituximab – a chimeric monoclonal antibody – competitively binds to the β2-glycoprotein 1-phosphatidylserine complex, resulting in anti-tumor immune activation and anti-angiogenesis. Bavituximab may act in the tumor immune microenvironment (TIME) through myeloid cells.

Objectives

The objective of this clinical study was to determine the survival benefit and the activity of adding Bavituximab to the Stupp protocol in patients with newly diagnosed glioblastoma. This report focuses on interactions between bavituximab and the cellular elements of host immunity in glioblastoma patients.

Methods

33 adults with newly diagnosed glioblastoma were enrolled in this phase II trial (NCT03139916). Bavituximab was given weekly, starting week 1 of Stupp. The primary objective was % overall survival at 12 months. We collected peripheral blood mononuclear cells (PBMCs) at enrollment and at regular intervals thereafter. Tumor tissue was banked and analyzed retrospectively. The nCounter Myeloid Innate Immunity Panel, which includes 770 myeloid-associated genes, was used to profile the myeloid transcriptome in both PBMCs and tumor tissue. Multispectral immunofluorescence was used to characterize the intratumoral immune environment.

Results

In the TIME, bavituximab-treated patients saw a reduction in the number of MDSCs. While the pretreatment RNA profile in PBMCs was not associated with outcome, above-median progression-free and overall survival were associated with significantly enriched expression of myeloid-associated genes in pretreatment tumor tissue.

Conclusion

Bavituximab reduces MDSCs in the glioblastoma microenvironment. Elevated tumor expression of myeloid-associated genes may be a predictive biomarker for response to bavituximab.

11:25 – 11:35 Hijacking Sexual Privilege in Glioblastoma

Martyn Sharpe; Amanda Jenson, MD; Alexandra Baskin; **David S. Baskin, MD**

Introduction

Regulatory T-cells (Tregs) are an immunosuppressive class of T-cells that normally arrest a pro-inflammatory immune response to "Self" tissues. A Treg subclass is maintained in men and women, orchestrating anti-inflammatory responses toward reproductive tissues. These Tregs recognize seminal epitopes, suppressing antibody responses. We postulated that GBMs express sperm-specific and pregnancy-specific proteins to hijack reproductive-associated Treg immune-privilege.

Objectives

-To characterize the immunological niche of GBMs with respect to patient outcome.

-To examine novel pharmaceutical approaches to aid a pro-inflammatory response in the tumor milieu.

Methods

We analyzed four transcriptome GBM databases for hypoxia-responsive, RORC-Treg, steroidogenic pathway, and sperm/placenta-specific genes. GBM patient sera bound to cynomolgus monkey testicle, indicating the presence of circulating anti-sperm/anti-testicular antibodies.

Results

In silico analysis revealed reproductive-associated RORC-Tregs in tumors of GBM patients with poorer outcomes. These tumors have a steroidogenic signature with the synthesis of androgen and male-specific

antigens providing a niche for these immunosuppressive cells. A second steroidogenic signature mimics placental attributes. Estrogenic tumors are associated with infiltrating myeloid-derived suppressor cells (MDSCs) and tumor-associated macrophages (TAMs).
Serum of GBM patients and age-matched controls were interrogated for the presence of anti-sperm/testicular antibodies, found at greater than six-fold levels in GBM patients compared to controls.
Conclusion
We demonstrate that RORC-Tregs drive poor patient outcome. We show tumor Treg levels correlate highly with androgen levels. These Tregs appear to be derived from a population normally present in the patient's reproductive system. Secondarily, GBMs emulate placenta to hijack sexual privilege, attracting MDSCs/TAMs. These findings unlock a whole new pharmacopoeia of FDA-approved drugs for use in GBM patients.

11:35 – 11:45 Identification of Cancer-Associated Fibroblasts in Glioblastoma and Defining Their Pro-tumoral Effects

Saket Jain; Jonathan Rick; Rushikesh Sanjeev Joshi, BS; Angad Beniwal; Jordan Spatz, PhD; Alexander Chih-Chieh Chang; Alan T. Nguyen; Sweta Sudhir; Ankush Chandra, MD, MS; Alexander F Haddad, BS; Harsh Wadhwa; Sumedh S. Shah, MD; Lin Wang; Garima Yagnik; Joseph Costello; Aaron Diaz; **Manish K. Aghi, MD PhD**

Introduction
While cancer-associated fibroblasts (CAFs) with pro-tumoral effects have been demonstrated in systemic cancers, CAFs have been presumed absent in glioblastoma given the lack of fibroblasts in healthy brain.
Objectives
We sought to identify CAFs in glioblastoma.
Methods
We used serial trypsinization, a technique described for CAF isolation in other cancers; machine-learning morphology analysis; single-cell and bulk RNA sequencing with slingshot lineage trajectory analysis; and neurosphere implantation to identify glioblastoma CAFs and define their effects.
Results
Serial trypsinization of primary glioblastoma cultures yielded cells that morphologically resemble fibroblasts and transcriptomically resemble CAFs from other cancers, with trajectory analysis revealing mesenchymal lineage of these cells. Glioblastoma CAFs were chemotactically attracted to glioblastoma stem cells (GSCs) and CAFs enriched GSCs. Glioblastoma CAFs were enriched in the subventricular zone which houses neural stem cells that produce GSCs. To investigate CAF/GSC interaction mediators, we created a resource of inferred crosstalk by mapping expression of receptors to their cognate ligands, identifying PDGF-beta and TGF-beta as mediators of GSC's chemotactic and proliferative effects on CAFs, and osteopontin and hepatocyte growth factor as mediators of CAF-induced GSC enrichment. Glioblastoma CAFs also induced pro-tumoral M2 macrophage polarization by producing the EDA fibronectin variant which binds macrophage toll-like receptor 4 in a targetable manner. Including CAFs in GSC-derived xenografts induced in vivo growth.
Conclusion
These findings identify GBM CAFs and reveal their involvement with GBM stem cells, making them an intriguing target.

11:45 – 11:50 Wrap-up/ Transition

11:50 – 12:05 Break

12:05 – 12:45	**Presidential Address**
12:05 – 12:15	Introduction of the Academy President: Howard Riina
12:15 – 12:45	Presidential Address: Douglas Kondziolka

1:30 – 4:30	**Academy Spine Emerging Investigators' Program**
	Program Directors: Christopher Shaffrey, Russell Lonser, Gregory Zipfel

7:30 – 7:35 WELCOME & REMARKS

Sepideh Amin-Hanjani, MD

7:35 – 8:30 Peer Reviewed Abstract Session V: Neurosurgical Trials: From Concept to Completion
Moderators: Shenandoah Robinson & Bob Carter

7:35 – 7:45 Middle Meningeal Artery Embolization for Chronic Subdural Hemorrhage: Rationale and Clinical Trial Design

Adam S. Arthur, MD, MPH; David Fiorella, MD PhD

Introduction
Chronic subdural hemorrhage (CSDH) is one of the most common neurosurgical diagnoses and the number of affected patients is growing. This is likely related to both the aging of the population and increasing usage of antithrombotic and antiplatelet medications. Embolization of the subdural pseudomembranes via the middle meningeal artery is a relatively new and untested treatment. We set out to design and undertake a large scale, international, multicenter, randomized clinical trial to test this therapy.
Objectives
To describe the challenges, choices, design and implementation of the Squid Trial for the Embolization of the Middle Meningeal Artery for the Treatment of Chronic Subdural Hematoma (STEM).
Methods
STEM is an international multicenter randomized controlled FDA investigational device exemption (IDE) trial. The trial will examine the effectiveness and safety of embolization of the middle meningeal artery with Squid, a liquid embolic. The primary outcome is treatment failure defined by residual or reaccumulation of the subdural fluid at 180 days or major disabling stroke, myocardial infarction or death from any neurologic cause. Secondary endpoints include patient-reported quality of life measures, neuropsychological testing and hospital and ICU length of stay.
Results
The STEM trial will enroll up to 300 patients at 25 U.S. and 10 international sites and examine the potential of this treatment both as a stand-alone therapy and as an adjunct to surgical drainage.
Conclusion
The STEM trial is actively enrolling and will study the treatment of one of the most common and devastating diseases in neurosurgery.

7:45 – 7:55 HSV G207 Immunovirotherapy with or without Radiation for Recurrent High-grade Brain Tumors in Children

James M. Johnston, MD; Gregory K. Friedman, MD; Asim Bag; Joshua Bernstock, MSc MPH; John Fiveash, MD; Rong Li; Kara Kachurak; James M. Markert, MD; G. Yancey Gillespie

Introduction
Outcomes for recurrent pediatric high-grade glioma (HGG) are poor, with a well-established 5.6-month historical median survival. Oncolytic HSV G207 was safe in adult trials, and preclinical studies utilizing patient-derived HGG xenografts suggested that pediatric HGG are more sensitive to G207 than adult HGG.

Objectives
We report results of a first-in-children Phase I trial of intratumoral HSV G207 immunovirotherapy in recurrent supratentorial pediatric HGG (NCT02457845).
Methods
We used a 3+3 design with four treatment groups: 107 or 108 plaque-forming units (pfu) of G207 alone and then with a 5 Gy radiation dose to the gross tumor volume. Up to four catheters were inserted intratumorally before 2.4ml of G207 was infused over six hours. For groups 3 and 4, radiation was given within 24 hours of G207. Patients were followed for virus shedding, seroconversion, radiographic response, neuropathologic response and overall survival.
Results
We treated twelve children (age range 7-18 years) with recurrent IDH-1 wild type, supratentorial HGG. Catheter placement and virus infusion were well-tolerated with no dose limiting toxicities or serious adverse events. Median overall survival was 12.2 months (95% CI, 5.2-18.6), with 4/11 patients (36%) alive 18 months post-treatment.
Conclusion
Intratumoral treatment of recurrent pediatric HGG with G207 was safe at a maximum planned dose of 108 pfu + 5 Gy. Responses were frequent and G207 converted immunologically "cold" tumors to "hot" in subjects with rebiopsy after treatment. A Phase I trial of intratumoral G207 for treatment of recurrent cerebellar tumors is ongoing with two patients treated safely to date (NCT03911388).

7:55 – 8:05 Reproducibility of Clinical trials using CMV-targeted Dendritic Cell vaccines in Patients with Glioblastoma

John H. Sampson, MD PhD MHSc MBA; Kristen Batich; Patrick Healy; Michael Gunn; Min-Nung Huang; Duane Mitchell; James Herndon, PhD; Gloria Broadwater; Kelly Hotchkiss; Luis Sanchez-Perez, PhD; Smita Nair; Kendra Congdon; Pamela Norberg; Kent Weinhold; Gerald Archer; Elizabeth Reap; Weihua Xie; E Albracht; Katherine B Peters, MD PhD; Dina Randazzo; Margaret Johnson; Annick Desjardins, MD FRCPC; Henry Friedman, MD; Gordana Vlahovic, MD MHS; David A. Reardon, MD; James J Vredenburgh, MD; Darrell Bigner; Mustafa Khasraw; Roger Mclendon; Eric M. Thompson, MD; Steven Cook; Peter Edward Fecci, MD, PhD; Patrick James Codd, MD; Scott Floyd; Zachary J Reitman, BS; John Kirkpatrick; Allan H. Friedman, MD, FAANS, FACS; David Ashley; Daniel Landi

Introduction
Vaccination with dendritic cells (DCs) fares poorly in primary and recurrent glioblastoma (GBM). Moreover, GBM vaccine trials are often underpowered due to limited sample size.
Objective
-To report the follow-up data of three serially conducted dendritic cell vaccine trials targeting cytomegalovirus in glioblastoma
- To report in a larger confirmatory trial the repeated enhanced dendritic cell migration using Td vaccine site preconditioning
-To illustrate the reproducibility of long-term survival outcomes in trials employing cytomegalovirus dendritic cell vaccine trials for glioblastoma
Methods
To address these limitations, we conducted three sequential clinical trials utilizing Cytomegalovirus (CMV)-specific DC vaccines in patients with primary GBM. Autologous DCs were generated and electroporated with mRNA encoding for the CMV protein pp65. Serial vaccination was given throughout adjuvant temozolomide cycles, and 111Indium radiolabeling was implemented to assess migration efficiency of DC vaccines. Patients were followed for median overall survival (mOS) and OS.
Results

Our initial study was the phase II ATTAC study (NCT00639639; total n=12) with 6 patients randomized to vaccine site preconditioning with tetanus-diphtheria (Td) toxoid. This led to an expanded cohort trial (ATTAC-GM; NCT00639639) of 11 patients receiving CMV DC vaccines containing granulocyte-macrophage colony-stimulating factor (GM-CSF). Follow-up data from ATTAC and ATTAC-GM revealed 5-year OS rates of 33.3% (mOS 38.3 months; CI95 17.5-undefined) and 36.4% (mOS 37.7 months; CI95 18.2-109.1), respectively. ATTAC additionally revealed a significant increase in DC migration to draining lymph nodes following Td preconditioning (P=0.049). Increased DC migration was associated with OS (Cox proportional hazards model, HR=0.820, P=0.023). Td-mediated increased migration has been recapitulated in our larger confirmatory trial ELEVATE (NCT02366728) of 43 patients randomized to preconditioning (Wilcoxon rank sum, Td n=24, unpulsed DC n=19; 24h, P=0.031 and 48h, P=0.0195). In ELEVATE, median follow-up of 42.2 months revealed significantly longer OS in patients randomized to Td (P=0.026). The 3-year OS for Td-treated patients in ELEVATE was 34% (CI95 19-63%) compared to 6% given unpulsed DCs (CI95 1-42%).

Conclusion

We report reproducibility of our findings across three sequential clinical trials using CMV pp65 DCs. Despite their small numbers, these successive trials demonstrate consistent survival outcomes, thus supporting the efficacy of CMV DC vaccine therapy in GBM.

8:05 – 8:15 Deep Brain Stimulation of the Nucleus Accumbens for Opioid Use Disorder: Initial Results of an On-going Clinical Trial

Ali R. Rezai, MD; Manish Ranjan, MCh; Pierre-François D'Haese; Marc Haut; Wanhong Zheng; Laura Lander; Nicholas Brandmeir; Victor Finomore; Sally Hodder; James Berry; James Mahoney

Introduction

Novel treatments for refractory opioid use disorder (OUD) are needed given high treatment failure rates and associated mortality.

Objectives

We initiated an FDA/IRB-approved clinical trial sponsored by the National Institute on Drug Abuse (NIDA) to assess the safety, feasibility and efficacy of Nucleus Accumbens (NAc) and ventral internal capsule DBS in OUD.

Methods

Eligible participants with a 5-year history of treatment-refractory OUD and multiple overdoses underwent bilateral NAc DBS implantation. Safety and effects on substance abstinence, craving, mood, and executive functions were assessed. MRI tractography, 18fluoro-Deoxy-Glucose (FDG) PET scans, and electrophysiological local field potential recordings from the NAc DBS were performed to assess DBS effects.

Results

Two participants underwent NAc DBS implantation with no complications. The first participant achieved over 600 days of continuous abstinence to date (average relapse time prior to DBS was 1-2 weeks). Post-DBS improvements were noted in craving, depression, anxiety, executive functions, behavioral self-regulation, and functional status evidenced by his return to full-time employment. PET demonstrated increases in glucose metabolism in the dorsolateral and medial prefrontal cortex. Tractography suggested optimal therapeutic response linked to the mesial frontal cortex. The second participant was non-compliant with study requirements; DBS was explanted 15 weeks post-implantation. Additional subjects are being enrolled, the latest clinical, imaging and physiological outcomes will be presented.

Conclusion

NAc DBS is safe and can reduce substance use, craving and improve behavior and executive functions in refractory OUD. DBS for OUD is promising but challenging given the nature and severity of the disease and further investigation is warranted.

8:15 – 8:25	**Comparative Effectiveness of Surgical Approaches for Cervical Myelopathy: Results from the CSM-S Trial**

Zoher Ghogawala, MD; Melissa Dunbar; Janis Breeze; Adam S. Kanter, MD ; Praveen V. Mummaneni, MD; Erica Fay Bisson, MD; James S. Harrop, MD; Subu N. Magge, MD; Robert F. Heary, MD; Michael P. Steinmetz, MD; Michael G. Fehlings, MD, PhD; Todd Albert, MD; Paul M. Arnold, MD; K. Daniel Riew, MD; Marjorie C. Wang, MD MPH; Robert G. Whitmore, MD; John Heller; Frederick G. Barker, MD; Edward C. Benzel, MD

Introduction

The CSM-S study is a randomized prospective study conducted to compare the effectiveness of ventral versus dorsal (fusion or laminoplasty) surgery for patients with multi-level CSM. We have previously reported 2 year results that identified laminoplasty as having superior outcomes.

Objectives

To compare health-related QOL outcomes from surgery for CSM at 3 and 4 years after surgery.

Methods

A multi-center prospective, randomized clinical trial was conducted on patients aged 45-80 years with multi-level CSM. Patients were screened and enrolled over a 4 year period (2014-2018) from 15 sites. Patients were randomized to ventral or dorsal surgery (2:3 randomization). Dorsal surgical approach (dorsal fusion or laminoplasty) was at the discretion of surgeon and patient. Outcome assessments (SF-36 and EQ-5D) were obtained pre-operatively, 3 months, 6 months, and at 1, 2, 3 and 4 years post-operatively. Complications were assessed by an independent study coordinator at 1 month and 1 year post-operatively. We conducted a pre-specified analysis of patients as treated.

Results

A total of 15 sites randomized 163 patients. 63 (38.7%) were randomized to ventral surgery and 100 (61.3%) to dorsal. Average age was 62.2 years and 49% were male. Baseline characteristics were comparable between ventral fusion, dorsal fusion, and laminoplasty groups. 66 patients ultimately underwent ventral fusion (VF) and 97 (69 dorsal fusion (DF) and 28 dorsal laminoplasty (DL)) underwent dorsal surgery. Patients, regardless of strategy, demonstrated significant improvements in HR-QOL over a four year period post-operatively. DL was associated with the lowest complication rate 10.7% vs. 29.0% (DF) vs. 47.0% (VF) (P=0.002). As randomized, there were no significant differences in SF36-PCS at 2 years between ventral and dorsal surgery. At three years (similar to what was reported previously from 2 year data), DL has superior outcomes in primary outcome SF-36 PCS (9.1) when compared with VF (3.9; P<0.001) and DF (5.0; P=0.004). Moreover at 4 years, DL had superior outcomes in primary outcome SF-36 PCS (10.8) when compared with VF (3.3; P=0.001) and DF (5.4; P=0.001). At both 3 and 4 years, DL also had superior EQ-5D scores [0.21 (3 years), 0.22 (4 years)] when compared with VF [0.10; P=0.001 (3 years), 0.10; P=0.01 (4 years)] and DL had superior EQ-5D scores compared with DF [0.12; P=0.001 (3 years), 0.12; P=0.001 (4 years)].

Conclusion

Patients undergoing surgery for CSM demonstrate improved overall quality of life. In this trial where equipoise was verified, the superior improvement observed at 1 and 2 years in health-related quality of life following dorsal laminoplasty (as selected by surgeon) for CSM was maintained over 4 years.

8:25 – 8:30	**Wrap-up/ Transition**

8:30 – 9:25	**Peer Reviewed Abstract Session VI: Vascular Science** Moderators: Brian Hoh & Fady Charbel

8:30 – 8:40 Selective Endothelial Hyperactivation of Oncogenic KRAS Induces Brain Arteriovenous Malformations in Mice

Peng Roc Chen, MD; Eunsu Park; Eunhee Kim

Introduction

Brain arteriovenous malformations (bAVMs) are a leading cause of hemorrhagic stroke and neurological deficits in children and young adults, however, no pharmacological intervention is available to treat these patients. Although more than 95% of bAVMs are sporadic without family history, the pathogenesis of sporadic bAVMs is largely unknown, which may account for the lack of therapeutic options. KRAS mutations are frequently observed in cancer, and a recent unprecedented finding of these mutations in human sporadic bAVMs offers a new direction in the bAVM research.

Objectives

Using a novel adeno-associated virus targeting brain endothelium (AAV-BR1), the current study tested if endothelial KRAS G12V mutation induces sporadic bAVMs in mice.

Methods

Five-week-old mice were systemically injected with either AAV-BR1-GFP or -KRASG12V. At 8 weeks after the AAV injection, bAVM formation and characteristics were addressed by histological and molecular analyses. The effect of MEK/ERK inhibition on KRASG12V -induced bAVMs was determined by treatment of trametinib, a US Food and Drug Administration (FDA)-approved MEK/ERK inhibitor.

Results

The viral-mediated KRAS G12V overexpression induced bAVMs, which were composed of a tangled nidus mirroring the distinctive morphology of human bAVMs. The bAVMs were accompanied by focal angiogenesis, intracerebral hemorrhages, altered vascular constituents, neuroinflammation, and impaired sensory/cognitive/motor functions. Finally, we confirmed that bAVM growth was inhibited by trametinib treatment.

Conclusion

Our innovative approach using AAV-BR1 confirms that KRAS mutations promote bAVM development via the MEK/ERK pathway, and provides a novel preclinical mouse model of bAVMs which will be useful to develop a therapeutic strategy for patients with bAVM.

8:40 – 8:50 PPIL4, a Novel Wnt Signaling Molecule, is Mutated in Intracranial Aneurysm Patients

Murat Gunel, MD; Tanyeri Barak, MD; Emma Ristori; Adife Gulhan Ercan Sencicek, PhD; Danielle F Miyagishima, BA; Andrew Prendergast; Ketu Mishra Gorur, PhD; Stefania Nicoli

Introduction

Intracranial aneurysm (IA) rupture leads to catastrophic subarachnoid hemorrhage (SAH). Despite several studies performed conclusive identification of specific genes or molecular pathways involved in brain aneurysm formation and rupture has yet to be elucidated.

Objectives

IA rupture generally occurs without any warning signs, underscoring the importance of identifying individuals at risk. Genetic risk factors play an important role in disease pathogenesis and at-risk patients might be identified before catastrophic IA rupture.

Methods

Using WES, we sequenced 491 sporadic IA patients and identified that PPIL4 was significantly enriched in European IA cohort. We generated a Crispr-Cas9 induced mutant ppil4 zebrafish line. Using transgenic lines in zebrafish, we further assessed cellular signaling mechanisms affected by ppil4 abrogation.

Results
Using exome sequencing, we have identified rare, deleterious mutations in PPIL4 in both familial and singleton IA cases. Ppil4 depletion causes defects in cerebrovascular morphology, cerebral hemorrhage and reduction in Wnt signaling activity both in zebrafish brain parenchyma and cerebrovascular endothelial cells in vivo. Wild type but not IA-mutant PPIL4 potentiates WNT signaling via binding JMJD6, a known angiogenesis regulator and Wnt activator.
Conclusion
Wnt signaling is indispensable for CNS specific angiogenesis and involved in pathogenesis of several cerebrovascular disorders. Our findings identify a novel PPIL4-dependent Wnt signaling mechanism critical for cerebrovascular wall integrity and shed novel insight into the pathogenesis of human IA, with diagnostic and clinical implications.

8:50 – 9:00 Air Pollution Exposure and Chronic Cerebral Hypoperfusion Exhibit Synergistic Effects on White Matter Injury

William Mack MD; Qinghai Liu, MD; Krista Lamorie-Foote, BA; Kristina Shkirkova; Todd Morgan; Constantinos Sioutas; Berislav Zlokovic; Caleb Finch; Mikko Huuskonen

Introduction
Exposure to air pollution particulate matter (PM) is associated with increased risk of dementia and accelerated cognitive loss. Vascular contributions to cognitive impairment are well recognized. Chronic cerebral hypoperfusion (CCH) promotes neuroinflammation and blood-brain barrier weakening, which may augment neurotoxic PM effects.
Objectives
This study examined interactions of nanoscale particulate matter (nPM, aerodynamic diameter ≤200 nm) and CCH secondary to bilateral carotid artery stenosis (BCAS) in a murine model to produce white matter injury. We predicted synergies of nPM with BCAS.
Methods
nPM was collected near an LA freeway. Mice (C57BL/6J males) were randomized to four exposure paradigms: 1) filtered air, 2) nPM, 3) filter+BCAS, 4) nPM+BCAS. Histochemical/ western blot analyses, transcriptome analysis, MRI, and behavioral assessments were performed.
Results
The joint nPM+BCAS group exhibited synergistic white matter injury with greater loss of corpus callosum volume on T2 MRI ($p<0.05$) and blood brain barrier breakdown on perfusion/ permeability sequences ($p<0.05$). Histochemistry verified microglial-specific inflammatory responses with synergistic effects on C5 immunofluorescence and nitrate concentrations ($p<0.05$). Transcriptomic responses (RNAseq) showed greater impact of nPM+BCAS than individual additive effects, consistent with pro-inflammatory pathway changes. While nPM exposure alone did not alter working memory, the nPM+BCAS cohort demonstrated impaired working memory compared to the filter+BCAS group ($p<0.05$).
Conclusion
Our data indicate that nPM and CCH contribute to white matter injury in a synergistic manner, suggesting adverse neurological effects aggravated in a susceptible clinical population exposed to air pollution. These findings could have implications for individuals with cerebral hypoperfusion from carotid stenosis, intracranial atherosclerosis, or cerebral small vessel disease.

9:00 – 9:10 VWF Inhibitor Recanalizes Middle Cerebral Artery after 6 Hours of Large Vessel Occlusion Stroke

Shahid Mehdi Nimjee, MD PhD; Amanda Sahar Zakeri; Debra Wheeler; Aarushi Kini; Arianna Carfora; Taggart Stork; Matthew Joseph, BS; Surya Gnyawali; Cole Anderson; Mohammad Shujaat

Introduction
Acute ischemic stroke (AIS) is the leading cause of combined morbidity and mortality worldwide. Recombinant tissue plasminogen activator (rtPA) is the only approved pharmacological treatment for AIS but is limited to treating patients within 4.5 hours of stroke onset because of the risk of intracranial hemorrhage. Moreover, it is ineffective in treating large vessel occlusion (LVO) stroke. Endovascular mechanical thrombectomy (MT) effectively recanalizes LVO stroke but it limited to highly-specialized hospitals, leaving the vast majority without timely acute treatment.
Objectives
We hypothesize that targeted von Willebrand Factor (VWF) inhibition by DTRI-031 will recanalize arterial thrombosis in a canine model of LVO stroke.
Methods
Utilizing a canine embolic middle cerebral artery occlusion (eMCAO) model of LVO stroke, we assessed DTRI-031 administration at 0.5mg/kg 6 hours after stroke induction on platelet activity by PFA-100, vessel recanalization by digital subtraction angiography, infarct volume, and intracranial hemorrhage by MRI.
Results
DTRI-031 administration after 6 hours of LVO stroke resulted in complete inhibition of platelet activity. Moreover, it recanalized MCAO to >TICI 2A in 62.5% and >TICI 2B in 50% of canines (n=8). Negative control group demonstrated no revascularization (n=7). Recanalization resulted in reduced infarct volume compared to negative control ($p<0.05$). DTRI-031 administration induced no intracranial hemorrhage.
Conclusion
VWF inhibition by DTRI-031 completely inhibited platelet activity, and effectively recanalizes LVO when administered 6 hours after stroke onset. Recanalization resulted in reduced infarct volume, without any incidence of intracranial hemorrhage. Targeted therapy against VWF represents a robust yet safe approach to treat AIS.

9:10 – 9:20 Cognitive Neurological Sequela Following Aneurysmal SAH

Gavin W. Britz, MD, Regnier-Golanov AS, Joseph Meno, Golanov EV

Introduction
Aneurysmal SAH is a devastating disease with as many as 95% of patients experiencing permanent disabilities which includes impaired memory and cognitive disturbances (1). It has been reported by our laboratory that even long term mortality is increased in those patients with ruptured and unruptured aneurysms (2). Previous research into the pathophysiology of SAH consequences have focused mainly on hypoperfusion due to delayed cerebral ischemia developing within few days after the ictus, which however occurs only in about 20% of SAH survivors while 95% suffer from long-term brain and cognitive disturbances, including dementia, cerebral amyloid angiopathy (3). Our laboratory initially focused on the pial and penetrating arterioles which were found to be abnormal following an SAH but this did not completely explain the sequela (4, 5). The long-term cognitive deficits following SAH result from morphological brain damage comparable to those observed in Alzheimer's disease, including atrophy of the temporomesial/hippocampal area (6), which correlates with decreased neurocognitive scores (7). Therefore the focus has now changed and we hypothesize that long-term neurocognitive abnormalities following SAH are triggered by damage of major hippocampal afferent pathways followed by complement activation and neuroinflammatory response which gets worse with age and aggravated by abnormal cerebrospinal flow affecting the glymphatic flow of the brain.
Objectives
Evaluate cognitive neurological sequela following aneurysmal SAH.

Methods

Subarachnoid hemorrhage (SAH) and cerebrospinal fluid (CSF) flow. Using microsphere movements and contrast-assisted MRI to monitor CSF flow in mice, we evaluated CSF flow and the role that glia limitans and tissue factor play. In addition, generation and deposition of endogenous fibrin was evaluated.

SAH and Hippocampus (Hpc). The hippocampus was evaluated for myelination, atrophy, spatial learning, memory retention and cognitive decline, number of dendritic spines, in vivo long-term potentiation, and levels of complement components (innate immune response). In addition, Analysis of the whole Hpc transcriptome was completed.

Results

Subarachnoid hemorrhage (SAH) and cerebrospinal fluid (CSF) flow. We established that CSF flow is stalled for up to 30 days following the SAH and that glia limitans and tissue factor play a significant role in limiting spread of blood in subarachnoid space. Based on the arrest of CSF along the paravascular space of the circle of Willis vessels. Further analysis revealed for the first time that normal human astrocytes stimulated with proinflammatory stimuli increase expression of fibrinogen chains confirmed by Western blot and RT-qPCR. Four days following SAH, fibrinogen chains Aα, Bβ, and γ associated with glia limitans and superficial brain layers increased. Comparable results were obtained with normal human neurons. These data suggest that fibrin associated with amyloid plaques may be of endogenous origin (8). In addition, this may result in abnormal glymphatic flow and prevent clearance waste products from the brain.

SAH and Hippocampus (Hpc). We observed significant decrease of myelination in Hpc and its atrophy 30 days after the SAH. Thirty days after SAH, affected animals demonstrated decrease in spatial learning, memory retention and cognitive decline, which worsened in aged animals. No signs of blood and overt cell loss was observed in Hpc. However, number of dendritic spines in Hpc significantly decreased and correlated with suppression of in vivo long-term potentiation. LTP suppression was reversed by complement C3 antibodies. Following SAH, we observed neuroinflammation in the Hpc: levels of complement components (innate immune response) increased; microscopic (conventional, confocal, super-resolution) analysis revealed cell and areas specific distribution of complement in the Hpc; Astro- and microglia, showed inflammatory phenotype and phagocytosis of pre-and postsynaptic elements. In accord with the clinical observations that SAH has the highest risk factor of dementia development compared to other strokes, our SAH data showed increased levels of Abeta and decreased ApoE expression, suggesting that our SAH model might be a model of sporadic Alzheimer's disease. Pro-inflammatory HMGB1protein and annexin increased and glycosylation of Hpc parenchyma changed. Cultured human astrocytes demonstrated increased production of the complement in response to proinflammatory signals. Analysis of the whole Hpc transcriptome revealed significant up regulation of 642 genes and down regulation of 398 genes in SAH vs. Control group. Overexpressed were the genes associated with the immune and antigen processing and presentation, extracellular matrix, and activation of complement pathway. Down regulated were genes related to demyelination and oligodendrocytes. TNF-α and IL1-β were identified as upstream regulators of the Hpc inflammation, and type I and II interferons were identified as super-regulators. DNA motifs common for numerous up regulated genes were Krüppel-Like factors and Interferon-regulatory binding motifs (9).

Conclusion

Abnormal CSF flow interrupting the glymphatic flow and complement induced dendritic pruning along with demyelination are important factors in the development of cognitive neurological sequela following aneurysmal SAH.

9:20 – 9:25 Wrap-up/ Transition

9:25 – 9:35 Break

9:35 – 10:00 **Past Presidential Address**

9:35 – 9:40 Introduction of the Academy Past President: Matthew Howard

9:40 – 10:00 Past Presidential Address: M. Sean Grady

10:00 – 11:05 **Peer Reviewed Abstract Session VII: Novel Technologies & Approaches**

Moderators: Karin Muraszko & Howard Riina

10:00 – 10:10 **Brain Surface Cooling in Human Neurosurgical Patients: Safety and Efficacy**

Jeremy D.W. Greenlee, MD; Kenji Ibayashi; Hiroyuki Oya, MD, PhD; Hiroto Kawasaki, MD; Christopher Kovach; Michael Long

Introduction

Functional brain mapping is a commonly used adjunct to facilitate safe and efficacious cranial surgery. A variety of modalities have been developed for this purpose as we strive to treat our patients harboring intraaxial neoplasm, vascular lesion, or medically refractory epilepsy. Brain surface cooling is one functional mapping technique.

Objectives

Investigate the utility of brain surface cooling during craniotomy and use this method to probe neural mechanisms of human language function.

Methods

Two types of cooling probes have been used during awake or asleep craniotomy. Initially, a stainless-steel chamber was passively cooled via chilled hypertonic saline infusion. A second smaller version was a titanium chamber with an attached peltier element for active cooling. In all cases brain surface temperature was continuously monitored via an embedded surface thermocouple to ensure temperatures did not go below 0 C.

Results

Since 2007, 40 patients have undergone cooling. Prior to 2007, 18 additional patients underwent cooling as part of pilot protocols. Our data showed no intraoperative seizures due to cooling, and no difference in incidence of post-operative seizure compared to patients treated with traditional intraoperative electrical stimulation functional mapping. Our findings also identified cortical cooling to be an effective method of reversible functional disruption with alterations in speech behavior.

Conclusion

Our work demonstrates the safety and effectiveness of brain surface cooling during cranial surgery. Like any surgical technique, this method has strengths and weaknesses that require further study and refinement to best compare to other methods and improve patient outcomes.

10:10 – 10:20 **Ultrahigh-Resolution MRI: Assessment of a Novel Endosphenoidal Minicoil Designed for Pituitary Gland Imaging**

Marvin Bergsneider, MD; Jiahao Lin; Siyuan Liu; Giyarpuram Prashant, MD; Sophie Peeters, MD; Rob Candler; Kyung Sung

Introduction

The identification of a microadenoma in Cushing's disease remains elusive in up to 40% of patients. Low signal-to-noise ratio (SNR) is the main limitation of MRI resolution. SNR is related to magnet strength and

the distance from source to receiver coil. We constructed a prototype 2-cm diameter coil (Fig. 2) designed to be positioned immediately adjacent to the sella turcica (Fig. 1) for intra-operative imaging.

Objectives

To measure SNR achieved by the protocol coil relative to clinical head coils.

Methods

An agar-based phantom (Fig. 3) with Gd-solution filled columns of diameter ranging from 1 - 2.8 mm was utilized. HiRes 2D PD-TSE sequences (Siemens Prisma 3T; Resolution 0.2x0.2x0.7 mm) were obtained. SNR was measured at various coil angles (relative to the static b0 field) and distances.

Results

The minicoil SNR values exceeded those of the clinical head coil at all angles and distances measured (Fig. 4). At an anticipated minicoil distance of 10 mm and 12 degrees angle (Fig. 1), SNR was 700% greater than that measured using the head coil. The HiRes PD-TSE images (Fig. 5) demonstrate the marked improvement in image resolution with the ability to discern at 1 mm (even at 1.6 cm distance depth).

Conclusion

The minicoil achieved a 7x-fold or greater increase in SNR relative to the clinical multiarray surface coils when utilized in a standard clinical 3T MRI. For reference, utilizing a 7T MRI (without the minicoil) would result in only a 2.3x-fold increase in SNR. A disadvantage to this approach is the need for an intra-operative MRI study.

10:20 – 10:30 Use of a Handheld Raman Spectroscopy Device to Characterize the Tumor Bulk, Margin, and Surrounding Brain during Surgery

Constantinos G. Hadjipanayis, MD PhD

Introduction

Intraoperative technologies are currently being developed to better detect and delineate brain tumors to maximize extent of resection. Raman spectroscopy is a sensitive, label-free, modality that gives spectral tissue characteristics based on molecular signatures resulting from inelastic scattering of incident light.

Objectives

A new clinical trial was initiated in the US for the first time that utilized a handheld Raman spectroscopy device for real-time differentiation of brain tumor tissue from the surrounding brain tissue during surgery.

Methods

Twelve patients undergoing a craniotomy for brain tumor resection were enrolled into an IRB-approved prospective study. A handheld Spectroscopy laser probe was used to perform Raman spectrum measurements from the tumor bulk, margin, and the surrounding brain during tumor resection. Corresponding specimens of tissue biopsies were sent for histopathology examination to determine the density of tumoral cells.

Results

Raman signatures were obtained from 2 glioblastomas, 7 brain metastases, 2 meningiomas, and 1 cavernoma. 126 Raman measurements were made for all tumors including the tumor bulk, margin, and the surrounding brain. Histopathologic assessment of each sample was used as the ground truth for labeling the corresponding Raman signature. Selected Raman peaks show clear differentiation of normal from tumor tissue. A Raman peak at 510 cm-1 for normal and infiltrating tumor in glioblastomas revealed a median intensity ratio of 2.5.

Conclusion

Use of the handheld Raman spectroscopy device can sensitively detect different portions of a tumor during surgery. Spatially precise, real-time in vivo detection of the presence of tumor cells may guide the neurosurgeon to perform safe maximal resection of brain tumors.

10:30 – 10:40 Molecular Nanoprobes for Rapid and Specific Diagnosis of Malignant Brain Tumors

Peter Nakaji, MD; Joseph F. Georges, DO PhD; Xiaowei Liu; Xiaodong Qi; Zein Al-Atrache, DO; Trent Anderson; Hao Yan, PhD

Introduction
Neurosurgeons often rely on frozen sections to assist differentiating operative lesions from nonoperative lesions intraoperatively. However, this technique can fail to distinguish non-operative versus operative lesions such as lymphoma (PCNSL) versus glioblastoma (GBM). More specific immunohistochemistry (IHC) is not available in an intraoperative time frame.
Objectives
Because diagnostic uncertainty may decrease quality of care, improved intraoperative brain tumor diagnostics are needed. We explore a strategy using aptamer-based molecular nanoprobes to make rapid and specific intraoperative diagnoses.
Methods
GBM and PCNSL-specific fluorescent aptamers were engineered, targeting GFAP and a CD20 immunoglobulin, TD05, respectively. Aptamer affinity to positive and negative controls was evaluated by flow cytometry and live-cell imaging. PCNSL and GBM rodent xenograft biopsies were utilized to optimize a rapid and specific staining protocol. Image data were statistically analyzed and then evaluated by clinical pathologists.
Results
GFAP aptamers generated a 3.4-fold fluorescence increase compared to negative controls by flow cytometry ($p<0.01$) and 2.3-fold in cell culture ($p< 0.01$). PCNSL-specific aptamers showed high affinity across experiments, labeling 80.75 ± 2.52% lymphoma cells vs. 8.25 ± 1.51% GBM cells from biopsies ($p< 0.001$). PCNSL-specific aptamers diagnosed xenograft biopsies within 11 minutes (Figure 1). A randomized image set of aptamer-labeled biopsies was interpreted with 100% accuracy by two clinical pathologists.
Conclusion
Aptamers are molecular nanoprobes that can bind targets with near-IHC affinity. These molecules may show utility as rapid intraoperative diagnostic agents for GBM and PCNSL. Their clinical application may improve both speed and accuracy of brain tumor diagnoses, allowing for intraoperative changes in surgical strategy.

10:40 – 10:50 Spinal Column Shortening for Tethered Cord Syndrome: Short-Term Outcomes

Andrew H. Jea, MD

Introduction
Tethered cord syndrome (TCS) is a clinical and radiographic diagnosis from pathologic stretch of the spinal cord leading to progressive loss of neurological function. The gold standard treatment for TCS is a tethered cord release (TCR). However, detethering involves significant risks of spinal cord injury and high rates of retethering. To mitigate these risks, the concept of spinal column shortening (SCS) to decrease spinal cord tension has become an alternative to detethering.
Objectives
In this study, we applied SCS to pediatric and transitioning adults affected by secondary TCS, and report radiographic, clinical, patient-Reported, and urodynamic short-term outcomes.
Methods
A retrospective review of a prospective database at our tertiary pediatric institution was performed. We used the Pediatric Quality of Life Inventory (PedsQL) patient- and parent-reported outcomes (PROs) and urodynamics to evaluate the outcomes of TCS treated with SCS.
Results
41 patients with secondary TCS were treated with SCS. The average age at the time of surgery was 15.9 years (range, 5-55 years). Preoperative symptoms evaluated included pain (33 patients), weakness (30 patients), and

bladder/bowel dysfunction (39 patients). The most common level of spinal column osteotomy was at T12, with spinal fusion between T10-L2. Follow-up time was 22.6 months on average (range, 8-45 months). For patients with at least 12 months of follow-up, subjective clinical improvements were reported in 91.3% of patients with preoperative pain (n=23, p<0.01), 66.7% of patients with weakness (n=24, p<0.01), and 51.7% of patients with bladder/bowel dysfunction (n=29, p<0.01). The median difference in initial and most recent PedsQL was +5 for patient-reported scores (n=19, p=0.04) and +5 for parent-reported scores (n=19, p=0.08). Formal urodynamics performed at a median 3.5 months after surgery documented stable to improved bladder function in 16 of 17 patients, with a median improvement in one classification category (n=17, p=0.01).

Conclusion

SCS continues to represent a safe and efficacious alternative to traditional spinal cord untethering for TCS in children and transitional adults as documented by objective formal urodynamics and PROs.

10:50 – 11:00 Increasing Local Blood Flow to the Spinal Cord with Focused Low-Intensity

Nicholas Theodore, MD; Yohannes Tsehay; Enoch Zeng; Carly Weber-Levine; Tolulope Awasika; Ann Liu MD; Jeffrey Ehresman BS; Eli Curry; Fariba Aghabaglou; Amir Manbachi Ph.D

Introduction

Spinal cord injury (SCI) is a devastating condition that affects about 17,000 individuals every year in the United States, with approximately a quarter million people living with the ramifications of the initial trauma. After the primary, a secondary phase occurs when the spinal cord continues to sustain injury due to ischemia which stems from a loss of autoregulation. Even with our current medical and surgical interventions, patients continue to experience poor outcomes. Animal experiments have shown that ultrasound stimulation of brain tissue leads to an increase in blood flow to the stimulated area.

Objectives

Determine if ultrasound stimulation of the spinal cord leads to an increase in blood flow in a healthy rodent spinal cord.

Methods

Three male adult Sprague-Dawley rats were used in this study. Laminectomies were performed at T11, and laser speckle contrast imaging (LSCI) was used to measure changes in blood flow to the spinal cord. Ultrasound stimulation was performed with a transducer sonicating at 500 kHz frequency at 50% duty cycle. LSCI was performed for three minutes total: the first minute to establish a baseline, the second to assess changes during stimulation, and the last minute to assess post-stimulation changes. Three rounds of experiments were performed for each rat. The stimulation and post-stimulation data were normalized using baseline recordings, and average percent changes in blood flow were computed along with 95% confidence intervals.

Results

Results: 8 of the 9 experiments were completed successfully. An increase in blood flow with ultrasound stimulation was observed in all of the experiments. On average, blood flow increased by 9.5 +/- 2.7%. An average increase of 2.5 +/- 3.7% was observed in the post-stimulation period.

Conclusion

An increase in blood flow was observed during ultrasound stimulation of the spinal cord, and flow can remain elevated even after the sonication period. The usage of ultrasound stimulation following spinal cord injury may lead to improved perfusion and could ultimately help ameliorate the effects seen with secondary injury.

11:00 – 11:05 Wrap-up/ Transition

11:05 – 11:20 Break

11:20 – 12:15 Peer Reviewed Abstract Session VIII: Epilepsy & Functional
Moderators: Daniel Yoshor & Alexandra Golby

11:20 – 11:30 Electrophysiological Fingerprints of Mesial Temporal Lobe Seizures Across Limbic Thalamic Nuclei

Kristen O. Riley, MD; Adeel Ilyas, MD

Introduction
Preclinical and functional imaging studies have confirmed that the anterior (ANT), centromedian (CeM) and mediodorsal (MD) thalamic nuclei play a diverse role in the ictogenesis of limbic seizures. However, published data on changes within the nuclei at ictal onset is lacking.
Objectives
To analyze electrographic recordings from each nucleus to establish spectral signatures within the ANT, CeM and that demarcate ictal onset.
Methods
Following IRB approval, adults (N=22) with suspected mesial temporal lobe epilepsy undergoing stereo-EEG were recurited prospectively for electrode implantation into one of the thalamic nuclei (ANT, CeM or MD). Ictal recruitment of the thalamus was confirmed visually and with validated quantitative metrics. Spectral analysis was performed via a time-frequency decomposition of thalamic ictal EEG.
Results
A total of 129 amygdala-hippocampal onset seizures were analyzed. Thalamic recruitment was confirmed in 111 (86%) of these seizures. The spectral signatures within each of the ANT, CeM and MD were distinct. Recruitment in the ANT and CeM were characterized by an early increase in spectral activity within the first 5 seconds of seizure onset, whereas recruitment of MD was characterized by delayed changes in spectral activity. Electrographic seizure onset pattern correlated with the recruitment latencies in the thalamic subnuclei.
Conclusion
The spectral signatures and recruitment latencies across thalamic subnuclei were distinct and were correlated with hippocampal electrographic seizure onset pattern. The ANT and CeM had the shortest latencies. These results hold promises for future closed-loop deep brain stimulation for epilepsy.

11:30 – 11:40 Anterior TransMaxillary Temporal Lobectomy (ATM-TL) for Hyper-selective Amygdalohippocampectomy: A Feasibility Study

Paul A. Gardner, MD; Jorge Alvaro Gonzalez-Martinez, MD, PhD; Michael Maurice McDowell, MD; Omuvwie Igberhi Orhorhoro, MBBS; Georgios Andrea Zenonos, MD; Carl H. Snyderman, MD

Introduction
Mesial temporal lobe epilepsy surgery may result in cognitive decline due to unnecessary violation of functional cortical and subcortical areas. Hyper-selective approaches using anterior trans-facial corridors could provide optimal resection while preserving non-involved brain tissue.
Objectives
An anatomical and clinical feasibility study of a novel endoscopic transmaxillary approach for medically refractory epilepsy was developed and applied.
Methods

24 cadaveric brain hemispheres were studied for anterior temporal surface anatomy; two for white matter dissection; and eight for evaluating various endoscopic corridors to the anterior and mesial temporal lobe structures. Transorbital, endonasal and transmaxillary endoscopic approaches were analyzed with 0o endoscope and neuronavigation. Accessibility, visualization and completeness of mesial and temporal pole resections were analyzed. Development of the technique in ex-vivo anatomical studies was then followed by its application in a patient with medically refractory mesial temporal lobe epilepsy.

Results

The ATM-TL was optimal for direct visualization of the temporal pole and natural alignment with the mesial temporal lobe structures. The ATM-TL allowed direct access lateral and inferior to the maxillary and mandibular nerves with a caudal-rostral trajectory allowing for a selective amygdalohippocampectomy with preservation of the trigeminal branches and the lateral temporal neocortex. These results were replicated clinically in a patient with left sided temporal lobe epilepsy and large meningo-encephalocele extending into the left sphenoid sinus lateral recess.

Conclusion

The ATM-TL approach is a new, hyper-selective alternative temporal lobectomy approach. It requires an experienced, multi-disciplinary team tand further studies are necessary to validate its safety, efficacy and potential cognitive benefit.

11:40 – 11:50 Epilepsy Surgery in Infants up to Three months of Age: a Multicenter, Multinational Study

Howard L. Weiner, MD; Jonathan Roth; Shlomi Constantini, MD; Margaret Ekstein, MD; Shimrit Uliel Sibony

Introduction

Drug resistant epilepsy (DRE) during the first few months of life is challenging, and necessitates aggressive treatment, including possible curative surgery. As the most common causes of DRE in infancy are related to extensive developmental anomalies, surgery often entails extensive tissue resections or disconnection. The literature on "ultra-early" epilepsy surgery is sparse, with limited data concerning efficacy controlling the seizures, and safety.

Objectives

The current study's goal is to review the safety and efficacy of ultra-early epilepsy surgery performed before the age of 3 months.

Methods

To achieve a large sample size and external validity, a multinational, multicenter retrospectively study was performed, focusing on epilepsy surgery for infants under 3 months of age. Collected data included epilepsy characteristics, surgical details, epilepsy outcome, and complications.

Results

Sixty-four patients underwent 69 surgeries before the age of 3 months. The most common pathologies were cortical dysplasia (28), hemimegalencephaly (17), and tubers (5). The most common procedures were hemispheric surgeries (48 procedures). Two cases were intentionally staged, and none were unexpectedly aborted. Nearly all patients received blood products. There were no peri-operative deaths and no major unexpected permanent morbidities. 25% of patients undergoing hemispheric surgeries developed hydrocephalus. Excellent epilepsy outcome (ILAE grade I) was achieved in 66% of cases over a median follow up of 41 months (19-104 IQR). The number of antiseizure medications was significantly reduced (median 2 drugs, 1-3 IQR, $p<0.0001$). Outcome was not significantly associated with the type of surgery (hemispheric or more limited resections).

Conclusion

Epilepsy surgery during the first few months of life is associated with excellent seizure control, and is not associated with more morbidity than surgery in older infants. Thus, surgical treatment should not be postponed to treat DRE in very young infants based on their age. Surgery should be performed by highly experienced teams.

11:50 – 12:00 Multi Cyclic Square Wave Voltammetry for Neurochemical Closed-Loop DBS

Aaron E. Rusheen, BS; Hojin Shin; Jason Yuen; Abhinav Goyal; Juan Rojas Cabrera; Kevin Bennet, MBA; Chris Kimble; Dong Pyo Jang, PhD; Charles Blaha; Yoonbae Oh; **Kendall H. Lee, MD PhD**

Introduction

We have developed multiple cyclic square wave voltammetry (M-CSWV) for real-time basal extracellular dopamine and serotonin recordings in vivo. We use M-CSWV to probe how DBS improves the pathologic neurotransmitter dynamics in Tourette syndrome (TS) and in addiction.

Objectives

To measure basal neurotransmitter levels in vivo for the development of electrochemical closed-loop DBS system.

Methods

Basal (M-CSWV) and phasic release (FSCV) recordings were conducted in the striatum, nucleus accumbens, and substantia nigra pars reticulata during DBS in a rat models of TS, addiction, and normal rat. Tics were monitored with electromyography. Pharmacologic modulations with sulpiride (D2 receptor antagonist), SCH 23390 (D1 receptor antagonist), escitalopram, and cocaine were performed for mechanistic understanding.

Results

In TS rats, CM/Pf DBS elevated basal striatal dopamine levels by 10.6 6.8 nM and elicited phasic release of 75 24.9 nM. Furthermore, DBS reduced tic frequency by 28% 6% ($p < 0.001$). In addiction model rats, iv cocaine resulted in elevation of basal dopamine concentration from 134 32 nM to 281 60 nM in the nucleus accumbens. In normal rats, escitalopram increased average basal serotonin levels (52 5.8 nM) in the substantia nigra pars reticulata by 1.5-fold higher.

Conclusion

Our results demonstrate that M-CSWV can accurately measure basal neurotransmitter levels in normal and pathologic disease states. Excitingly, these results demonstrate the feasibility of electrochemical closed-loop DBS system. Indeed, we have integrated this technology into Mayo developed WINCS MAVEN, a novel device that will allow for electrochemical closed loop DBS.

12:00 – 12:10 DBS Can Slow Parkinson's Disease Progression - Class II Evidence

Peter Konrad, MD PhD; Mallory Hacker; David Charles, MD

Introduction

Deep brain stimulation (DBS) is an accepted symptomatic treatment for mid- to late-stage Parkinson's disease (PD). However, 14 years ago the authors and others introduced the idea that DBS could also slow PD. Investigating this idea, the authors led a randomized, pilot study in 30 patients on safety and tolerability of DBS in early PD.

Objectives

This report provides 5-year follow up of this unique cohort and continued disease-modifying effect of DBS.

Methods

STN DBS in early PD pilot study was a prospective, randomized, controlled, single-blind clinical trial (FDA-IDEG050016; VU-IRB#040797). Thirty participants with early diagnosis of PD were randomized between optimal drug therapy (ODT) vs ODT+DBS. Twenty-eight subjects were followed for 5 years. In addition to

safety outcomes, parameters reflecting symptom progression were followed at each annual visit, including UPDRS scores, quality of life, and medication use.

Results

Worsening of overall motor symptoms were 2.3 times more likely in ODT vs ODT-DBS (p=0.08). Notably, rest tremor was 4.8 times less likely in ODT-DBS subjects (p<0.001). L-DOPA requirements were 3.8 times higher in the ODT group than ODT-DBS group (p=0.02) ; and polypharmacy use was 16.7 times lower in the ODT-DBS group (p=0.01) . The odds of dyskinesia in ODT-DBS were 0.35 times lower than in ODT group (p=0.06).

Conclusion

This is the first and only clinical trial of STN DBS in early-stage PD reporting long term effects on significant disease parameters. The results suggest that early STN DBS+ODT is safe in PD and provides pilot evidence for disease modification effects of DBS applied to the STN.

12:10 – 12:15 Wrap-up/ Transition

12:15 – 12:50 Peer Reviewed Abstract Session IX: Vascular Practice
Moderators: Jacques Morcos & Cargill Alleyne

12:15 – 12:25 Variation in Carotid Artery Stenosis Measurements Among Facilities Seeking IAC Carotid Stenting Facility Accreditation

Mary Beth Farrell; Erik B. Lehman, MSc; David Sacks; Cathy Sila, MD; John Terry; **Kevin M Cockroft MD**

Introduction

Degree of stenosis is an important factor in treatment decision making for patients with cervical carotid stenosis. This is especially true for asymptomatic patients being considered for carotid artery stenting. While participants in clinical trials are typically trained in specific measurement systems, real world assessors may be less rigorous.

Objectives

The aim of this study was to compare the percent stenosis as measured by the physician operator with that measured by a panel of independent expert reviewers.

Methods

Representative images were selected from random cases submitted to the Intersocietal Accreditation Commission (IAC) as part of Carotid Stenting Facility accreditation. Operator-reported stenosis (ORS) as documented in the patient's operative report was compared to reviewer-measured stenosis (RMS) determined using NASCET criteria by five clinicians experienced in treating carotid artery disease. Median percent stenosis was compared using Wilcoxon Signed Rank test as measurements were not normally distributed.

Results

68 unique angiograms were reviewed. Median patient age was 70.0 (IQR 66.0, 79.5) and 25 (37%) were female. The median ORS was 90.0% (80.0%, 90.0%) as compared to median RMS of 61.1% (49.8%, 73.6%), yielding a median difference of 21.8% (13.7%, 34.4%), p<0.001. The median difference in ORS and RMS for asymptomatic versus symptomatic patients was not statistically different (24.6% versus 19.6%, respectively, p=0.406). However, when analyzed according to initial accreditation decision, there was a significant difference between those facilities that were granted initial accreditation and those whose accreditation was delayed (17.8% versus 25.5%, respectively, p=0.035).

Conclusion

Real world operators tend to overestimate the degree of cervical carotid artery stenosis. Stenosis measurements from facilities that were granted initial IAC accreditation were closer to expert measurements than those facilities whose accreditation was delayed. Since decisions regarding carotid revascularization are often based at least partially on percent stenosis, such measuring discrepancies may lead to procedural over utilization.

12:25 – 12:35 Direct Bypass Surgery for Moyamoya/Ischemia: Patency, Flow Measurements and Outcomes in 162 cases

Jacques J. Morcos, MD; Nickalus Khan, MD; Aria Jamshidi; Victor M Lu, MD; Michael A Silva, MD; Angela M. Richardson, MD, PhD

Introduction
EC-IC direct bypass surgery is commonly used for ischemic vasculopathy but large long-term studies with detailed analysis are lacking.
Objectives
We report clinical outcomes, intraoperative blood flow analysis, long term follow up and patency rates from a single surgeon's series of Moyamoya disease, Moyamoya syndrome, and steno-occlusive disease at a single institution over a 21-year period.
Methods
With IRB approval, we reviewed medical and imaging records for all patients who underwent cerebral revascularization by the senior author between August 1999 and November 2020.
Results
A total of 162 procedures/124 patients were identified. Mean clinical follow up time was 2 years 11 months. Mean imaging follow up time was 1 year 9 months. The combined immediate and long term postoperative stroke and/or intracerebral hemorrhage rate was 6.2%. Seventeen bypasses (10%) occluded at long term. There was significant difference ($p<0.0001$, Fisher's Exact Test) in the long term patency rate based on the presence/absence of complete collateralization on preoperative angiography. Cut flow index (CFI) did not predict long term patency. Overall, patients had a significant clinical improvement with mean mRS 1.8 preoperatively and 1.2 postoperatively. We compare and contrast our findings with other large series.
Conclusion
Direct bypass surgery in our series resulted in improved functional outcome, with a long term patency rate of 90%. Complete preoperative collateralization on preop angiography predicted tendency of bypass to occlude at long term. There was no correlation between bypass type, clinical syndrome, or CFI and long term occlusions. The role of bypass surgery and the need for surgical expertise remain strong in the treatment of Moyamoya variants and a select group of atherosclerotic steno-occlusive patients.

12:35 – 12:45 Short and Long Term Outcomes of Moyamoya Patients Post Revascularization

Gary K. Steinberg, MD, PhD; Mario Teo; Kumar Abhinav; Teresa Bell-Stephens; Venkatesh S Madhugiri, MBBS MCh; Eric S Sussman, MD; Rohaid Ali; Rogelio Esparza; Michael Zhang, MD; Tej Azad

Introduction
The post-bypass stroke risk factors and long-term outcomes of moyamoya patients are not well documented.
Objectives
We studied 30-day stroke risk and investigated patients' long-term physical, functional, and social well-being.
Methods

This was a single institution, combined MMD database interrogation and questionnaire study. From 1991-2014, 1250 revascularization procedures (1118 direct, 132 indirect) were performed in 769 patients. F/U was obtained on 96.3% of patients and 391 patients completed questionnaires.

Results

Among 548 F/221 M, mean age 32 yo (range 1-69 yo), 358 bypasses were performed in 205 pediatric patients (73% direct), and 892 in 564 adults (96% direct). 52 patients (6.8%) developed major strokes with worsening mRS within 30-days postop (5.3% and 2.6% after the first and second bypasses respectively). Logistic regression analysis showed older age, modified MRI (mMRI) score, and Hemodynamic Reserve (HDR) score were significantly associated with postoperative stroke. With mean follow up of 7.3 years (0.1-26 yrs), long term stroke risk was 0.6%/pt/yr. 75% of patients had excellent outcomes (mRS 0-1). 84% of patients reported resolution or improvement in their preoperative headache; 83% remained in employment or education; 87% were self-caring.

Conclusion

In this large, single center surgical series, the majority of the adult and pediatric cohorts had direct revascularization, with a 6.8%/pt 30-days major stroke risk, and 0.6%/pt/year long-term stroke risk. We also identified various risk factors that were highly correlated with postoperative morbidity (age, mMRI score, HDR score), with ongoing work to develop the predictive modelling for future patient selection and treatment.

12:45 – 12:50	**Wrap-up/ Transition**

1:30 – 2:00	**Joint Academy Emerging Investigator's Program**
	Program Directors:
	Christopher Shaffrey, Russell Lonser, Gregory Zipfel, Jeffrey Ojemann, Emad Eskandar
1:30 – 2:00	Introduction
2:00 – 4:30	Meetings with Established Investigator Faculty

7:30 – 8:20 Special Abstract Session: The Oldfield Session
Moderators: Nino Chiocca & Mark Johnson

7:30 – 7:35 Session Introduction

Russell R. Lonser, MD

7:35 – 7:45 Phase II trial of BRAF/MEK Inhibition in Newly Diagnosed Papillary Craniopharyngiomas (PCP): Alliance A071601

Frederick G. Barker, MD; Priscilla Brastianos, MD; Erin Twohy; Susan Geyer; Elizabeth Gerstner; Timothy Kaufmann, MD; Daniel P. Cahill, MD; Sandro Santagata, MD PhD; Helen Shih; Paul D. Brown, MD; Evanthia Galanis

Introduction
95% of papillary craniopharyngiomas (PCP) harbor BRAF-V600E mutations. We evaluated BRAF/MEK inhibition efficacy in patients (pts) with previously-untreated PCP.
Objectives
This single arm, Simon two-stage phase 2 trial had 89% power to detect a true RR of at least 30% (vs. null RR 5%; alpha=0.04). In this design, >2 confirmed responses in 16 evaluable pts would be considered promising activity.
Methods
Eligible pts without prior radiation whose PCP harbored BRAF mutations received oral vemurafenib/cobimetinib in 28-day-cycles. Centrally-reviewed volumetric response rate (RR) was the primary endpoint, with partial response defined as >20% volumetric decrease.
Results
Of 16 pts evaluated, 56% were female; median age was 49.5 years. Median follow-up was 22mo (95%CI:16-26.5) and median treatment cycles was 8. Three patients progressed after therapy was discontinued; none have died. 14/15 pts with centrally-reviewed volumetric data had responses (93%; 95%CI: 68%-99.8%). Of 16 patients evaluable based on local review, 15 had responses (93.75%; 95%CI: 70%-99.8%). Median tumor volume reduction was -83% (range:-52% to -99%). The one nonresponder stopped treatment after 2 days for toxicity. Median progression-free survival was not reached. Grade 3 toxicities at least possibly related to treatment occurred in 12 pts (rash in 6). Two grade 4 toxicities occurred: hyperglycemia (n=1) and increased CPK (n=1). Three pts discontinued treatment for adverse events.
Conclusion
Vemurafenib/cobimetinib provided volumetric response in all pts who received 1 or more therapy cycles. BRAF/MEK inhibitors are an active treatment for previously untreated PCP.

7:45 – 7:55 The Discrepancy Between Histologic and Molecular Grading of Meningioma: a Single Institution Series

Ian F. Dunn, MD; Amanda Roehrkasse; Jo Elle Peterson; Kar-Ming Fung; Panayiotis Emmanuel Pelargos, MD

Introduction

Advances in mutational and copy number profiling of meningiomas have highlighted the power of molecular signatures in predicting tumor behavior. Because these data are not routinely used, reconciling this predictive information with current WHO guidelines has added complexity to their interpretation and deployment.

Objectives

To compare the molecular profile with histologic grade in a prospective series of meningiomas to assess the degree of concordance with the WHO classification, the currently accepted method of predicting meningioma behavior.

Methods

We report a three-year single institutional experience comparing WHO histopathologic classification of meningiomas with the corresponding "molecular grade" F as suggested by advanced profiling. Genetic alterations with prognostic implications and copy number profiles were compared with the histopathological grade.

Results

151 total meningioma cases were included for analysis (85% WHO grade I, 15% WHO grade II/III). Of 129 samples with copy number data, 27% featured discordant copy number profiles from their histologic grade (Fig. 1, 2). 29% of WHO grade I tumors featured copy number profiles consistent with atypical or anaplastic meningioma, and 19% of WHO grade II meningiomas had copy number profiles consistent with grade I tumors. 6% of grade I meningiomas harbored alterations in negative prognostic markers TERT, CDKN2A/B, or BAP1.

Conclusion

We identified significant discrepancies when comparing molecular signatures of meningiomas to the standard WHO histopathologic grade. Further work will clarify how best to incorporate advanced profiling to aid in predicting tumor behavior and in clinical decision making.

7:55 – 8:05 Evidence of Familial Clustering of Sporadic Unilateral Vestibular Schwannoma from Multigenerational Genetic Databases

Richard Gurgel; **William T. Couldwell, MD, PhD**; Lisa Cannon-Albright

Introduction

Unlike the autosomal dominant inheritance of neurofibromatosis 2, there are no known familial risk factors for sporadic unilateral vestibular schwannomas (VS).

Objectives

To analyze familial clustering to test the hypothesis of a genetic contribution to predisposition to sporadic unilateral VS.

Methods

Familial clustering of individuals with unilateral VS was analyzed in two genealogical resources with linked diagnosis data, the Veteran's Health Administration (VHA) genealogy database and the Utah Population Database (UPDB). We tested for excess relatedness, relative risks (RR) in close and distant relatives, and pedigrees with a significant excess of unilateral VS among descendants.

Results

The average pairwise relatedness of the VHA VS cases significantly exceeded the expected relatedness ($p=0.016$), even when ignoring relationships closer than third degree ($p=0.002$). In the VHA resource, RR for third- to fifth-degree relatives developing VS were 60.83 ($p=0.0005$, 95% CI 7.37-219.73) and 11.88 ($p=0.013$, 95% CI 1.44-42.90), and no VS-affected first-, second-, or fourth-degree relatives were observed. In the UPDB population, no first- or second-degree relatives with VS were observed. RR for fifth-degree relatives developing a VS was 2.23 ($p=0.009$, 95% CI 1.15-3.90), and no VS-affected first- through fourth-degree relatives of VS cases were observed in the UPDB resource.

Conclusion

These results provide strong evidence for an inherited predisposition to sporadic, unilateral VS, although the affect is seen in distant family members, primarily third- and fifth-degree relatives. The high-risk unilateral VS pedigrees identified in two independent resources provide a powerful resource that can be pursued for predisposition gene identification.

8:05 – 8:15	**Magnetic Resonance Imaging-guided Gene Therapy for Aromatic L-amino Acid Decarboxylase Deficiency**

Russell R. Lonser, MD; Nalin Gupta, MD PhD; Toni Pearson; Jill Heathcock; Paul S. Larson, MD; James Bradley Elder, MD; Jeffrey R. Leonard, MD; Krystof Bankiewicz

Introduction

Aromatic L-amino acid decarboxylase (AADC) deficiency is a rare genetic disorder that results in deficient dopamine and serotonin synthesis. It results in profound neurologic disability, including severe developmental disability, global hypotonia, autonomic disturbances and oculogyric crises (OGCs).

Objectives

To define the safety and efficacy of gene therapy for treatment of AADC deficiency, we infused a viral vector (adeno-associated virus, serotype-2) expressing AADC (AAV2-hAADC) into the midbrain of children with AADC deficiency.

Methods

Children with AADC deficiency underwent real-time magnetic resonance (MR)-imaging guided convection-enhanced delivery (CED) of AAV2-hAADC into the bilateral substantia nigra (SN) and ventral tegmental area (VTA) (ClinicalTrials.gov Identifier NCT02852213). Clinical, imaging, laboratory and operative findings were analyzed.

Results

Eight children (range, 4 to 9 years) underwent CED of AAV2-hAADC to the bilateral (SN) and (VTA) (total infusion volume, 80 microliters per side) (dose cohorts, 1.3 x 1011 and 4.2 x 1011 vg). Real-time MR-imaging clearly defined infusion progression and total volume (end of infusion) of the regions perfused with AAV2-hAADC. Seven patients (88%) had complete resolution of OGCs by 3 months after infusion. Of the 5 patients that have long-term follow-up (18 months or more after infusion), 4 (80%) obtained the ability to sit independently and 2 (40%) can ambulate with minimal support. MR-imaging of delivery revealed 98% and 70% of the SN and VTA were perfused. Dopamine metabolism was increased in all 8 patients (100%) and 18F-DOPA positron emission tomography scanning revealed increased uptake within the midbrain and the striatum. Bilateral SN and VTA convective perfusion of AAV2-hAADC was well-tolerated and safe in all patients.

Conclusion

Image-guided convective perfusion of bilateral midbrain targets (SN and VTA) with AAV2-hAADC in children with AADC deficiency is feasible and safe. It results in the restoration of dopamine and serotonin production with corresponding clinical improvements.

8:15 – 8:20	**Wrap-up/ Transition**

8:20 – 9:10	**Academy Award Presentation and Lecture**
8:20 – 8:25	Introduction of Academy Award Winner By: Geoff Manley
8:25 – 8:35	Academy Award Winner Lecture
8:35 – 8:40	Introduction of NREF Academy Winners (3) By: Russell Lonser

8:40 – 8:55	American Academy Young Clinician Investigator & Research Fellowship Grant Recipients
8:55 – 9:05	Emerging Investigator Program By: Gregory Zipfel
9:05 – 9:10	Wrap-up/ Transition

9:10 – 9:55	**Peer Reviewed Abstract Session X: First in Human** Moderators: Griffith R. Harsh & Linda Liau

9:10 – 9:20	**A First-in-Human Phase 0/1 Clinical Trial of 5-Aminolevulinic Acid Sonodynamic Therapy in Recurrent Glioblastoma**

Nader Sanai, MD; An-Chi Tien; Artak Tovmasyan; Yu-Wei Chang; Tigran Margaryan; Kristin Hendrickson; Jennifer Eschbacher; Wonsuk Yoo; Jocelyn Harmon; Christopher Quarles; Lea Alhilali; Igorq Barani; Shwetal Mehta; Zaman Mirzadeh

Introduction
5-aminoleveulinic acid sonodynamic therapy (5-ALA SDT) is a drug-device strategy that exploits the metabolic liabilities of cancer. Following administration of 5-ALA, aberrant tumor cell metabolism accumulates protoporphyrin-IX (PpIX). Activation of PpIX by non-invasive, non-ablative magnetic resonance-guided focused ultrasound (MRgFUS) induces reactive oxygen species and tumor cell death.
Objective
This first-in-human Phase 0/1 study investigates the feasibility, safety, and biology of 5-ALA SDT in recurrent glioblastoma (GBM).
Methods
Six hours before SDT, adult patients with recurrent GBM receive Sonala-001 (10mg/kg), an IV formulation of 5-ALA. In a Dose-Escalation Arm, 9-18 patients are assigned to one of three ascending acoustic energy doses of MRgFUS (200J/400J/800J), followed by a four-day interval to tumor resection. In each patient, half the tumor volume is targeted with MRgFUS and the other half serves as an internal control. Using tumor pharmacodynamic endpoints, the Minimum Biological Dose (MBD) associated with 5-ALA SDT response is identified. In a subsequent Time-Escalation Arm, 12 patients are treated at the MBD with varying time-intervals between SDT and resection.
Results
Accrual to the 200J dose level (n=3) is complete. The median Cmax for 5-ALA and PpIX were 307 μM and 319 nM, respectively. The oxidative stress biomarkers 4-hydroxynonenal, glutathione, cysteine, and thiol were significantly elevated in treated tumor vs. control. Similarly, the apoptosis biomarker cleaved-caspase-3 was increased in treated tumor vs. control (median, 48.6% vs. 29.6%, p=0.05).
Conclusion
We report a new therapeutic modality for recurrent glioblastoma patients. 5-ALA SDT is safe at 200J and leads to targeted oxidative stress and tumor cell death in human glioblastoma.

9:20 – 9:30	**First in human CAN-3110 (ICP-34.5 Expressing HSV-1 Oncolytic Virus) in Patients with Recurrent High-grade Glioma**

E. Antonio Chiocca, MD PhD

Introduction

Recurrent glioma patients have few therapeutic options and an expected survival of only 7 to 10 months. New treatments to improve the prognosis of this patient population are a dire medical need. Oncolytic viruses (OVs) are emerging as important new agents for cancer treatment. The first FDA approved OV was talimogene laherparepvec (Imlygic, T-Vec) for treatment of melanoma. T-Vec, as other clinical HSV-1 based OVs, is deleted in the ICP34.5 gene, responsible for HSV-1 neurovirulence. However, deletion of ICP34.5 also impedes efficient viral replication. CAN-3110 (rQNestin34.5v2) maintains a copy of the HSV1 ICP34.5 gene under transcriptional control of the tumor-specific promoter for nestin to drive robust tumor-selective replication. CAN-3110 replicates in malignant glioma cells far above levels seen with ICP34.5 deleted viruses. This potency also created the hypothetical risk for increased neurovirulence, thus the regulatory advice to conduct a cautious nine-dose-level Phase-1 dose escalation study in patients with recurrent high-grade glioma (HGG).

Objectives

To determine the safety and obtain preliminary efficacy as well as biologic data for this fist in human new oncolytic HSV1.

Methods

From September 2017 to February 2020, thirty patients with biopsy-confirmed recurrent high-grade glioma were treated in an open label clinical trial. Patients with multifocal, multicentric, tumors larger than 5 cm, and tumors that had recurred multiple times were eligible. All patients received best standard of care treatments as indicated by their physician. CAN-3110 was injected intratumorally starting at 1x106 plaque forming units (pfu) and dose-escalating (3+3 design) by half log increments up to 1x1010 pfu. Tissue (when possible) and blood samples were obtained before and during treatment for experimental medicine analysis.

Results

CAN-3110 was well tolerated with no dose limiting toxicity observed. The initial tissue diagnosis of the recurrent tumor for the 30 subjects was 26 glioblastoma, 3 anaplastic oligodendroglioma, and 1 anaplastic astrocytoma. The median overall survival (mOS) of the entire group is 11.7 months. Post-treatment tissue is available for 18/30 subjects and revealed persistence of HSV antigen and CD8+ T cell infiltrates. Additional immunologic (including T cell receptor repertoire), transcriptomic and single cell RNA sequencing analyses are ongoing.

Conclusion

Administration of CAN-3110 into recurrent glioma was well tolerated without evidence of ICP34.5-induced encephalitis/meningitis. Histological and molecular analyses showed evidence that CAN-3110 injection was associated with immune activation and viral antigen persistence. Although clinical efficacy cannot be determined in this small phase 1 study, OS of CAN-3110 treated subjects compares favorably to historical reports and warrants further clinical studies.

9:30 – 9:40 GD2-CAR T-cell Therapy for H3K27M-mutated Diffuse Midline Gliomas

Gerald A. Grant, MD

Introduction

Diffuse intrinsic pontine glioma (DIPG) and other H3K27M-mutated diffuse midline gliomas (DMGs) are universally fatal central nervous system (CNS) tumors that occur most commonly in children and young adults. The average life expectancy is ten months from diagnosis and 5-year survival is less than 1%.

Objectives

Disialoganglioside GD2 is highly and uniformly expressed on H3K27M+ DMG cells. Furthermore, intravenously administered GD2.4-1BB.z chimeric antigen receptor (CAR) T-cells eradicated established DIPGs in patient-derived orthotopic murine models. These data provided the rationale for a first-in-human/first-in-child Phase 1 clinical trial (NCT04196413).

Methods

Patients were eligible for enrollment if they had a pathologically confirmed diagnosis of H3K27M-mutated DIPG or spinal cord DMG, had completed standard radiotherapy, and were not receiving corticosteroid therapy. All patients had an Ommaya reservoir placed prior to infusion for management of elevated intracranial pressure. We present the clinical experience from the first four patients with H3K27M+ DMG (pontine or spinal cord) treated with GD2-CAR T-cells administered intravenously.

Results

Three of four patients exhibited marked clinical and/or radiographic improvement, underscoring the promise of this approach for H3K27M+ DMG therapy in this disease. The toxicity predicted in preclinical models was reversible with intensive supportive care. Patients who exhibited clinical benefit were eligible for a second administration of GD2 CAR T-cells. Three patients received a second dose delivered intracerebroventricularly (ICV) through an Ommaya catheter. Serum levels of lactate dehydrogenase (LDH) increased in all patients after GD2-CAR T-cell treatment, tracking with evidence of inflammation, and with clinical and radiographic improvement in those patients who exhibited benefit. Cell-free tumor DNA (cfDNA) was detected in CSF using digital droplet PCR analysis of the tumor-specific H3K27M mutation.

Conclusion

The promising early experience with GD2-CAR T-cells for DIPG and spinal cord DMG described here sets the stage for further optimization of this approach for this historically lethal CNS cancer.

9:40 – 9:50 Therapeutic Delivery in Neurosurgical Oncology: Leveraging the OR to Overcome Barriers to Success

Michael A. Vogelbaum, MD PhD

Introduction

The successes associated with use of targeted small molecule drugs and immunotherapies in many forms of cancer have failed to translate into survival benefits for patients with gliomas.

Objectives

Most clinical trials in NeuroOncology lack disease-site relevant pharmacokinetic (PK) and pharmacodynamic (PD) analyses, which are routinely conducted during clinical development of systemically administered therapeutics.

Methods

An expert panel reviewed the use of Window of Opportunity (WoO) clinical trial designs in in NeuroOncology (Vogelbaum et al., NeuroOncology, 2020). Independently, a prospective first-in-human clinical trial of a novel therapeutic antibody (OS2966) for recurrent GBM was designed with both WoO and direct therapeutic delivery features. This is a 2-part study with an initial infusion of OS2966 into enhancing tumor tissue after pretreatment biopsy, followed by resection of the mass 1-10 days later and infusion of OS2966 into the tumor infiltrated brain.

Results

Over the past 3+ decades, only 22 trials of systemic agents for GBM have included assessment of PK and/or PD endpoints. Of those, only 50% included the use of a therapeutic dose of the investigational agent, and only 68% included assessments of tumor tissue effects. Only 25% included assessments of drug levels in non-enhancing, tumor infiltrated brain. OS2966 has been delivered to 2 patients to date. Details regarding the extent of target tissue coverage and analysis of pre- and post-treatment blood and tumor samples will be presented.

Conclusion

Neurosurgeons have unique access that permits assessment of the biological promise of therapeutics administered both systemically and directly to the site of disease.

9:50 – 9:55 Wrap-up/ Transition

9:55 – 10:10 Break

10:10 – 11:15 Peer Reviewed Abstract Session XI: Skull Base and Vascular
Moderators: Nelson Oyesiku & Michael McDermott

10:10 – 10:20 Defining Clinically Significant Tumor Growth in Vestibular Schwannoma: Moving Beyond Minimum Detectable Growth (≥2mm)

Michael J. Link, MD; Matthew L Carlson; Robert Macielak; Christine Lohse; Jamie Joseph Van Gompel, MD; Brian Neff; Colin L.W. Driscoll, MD

Introduction
Detection of vestibular schwannoma (VS) growth during observation leads to definitive treatment at most centers globally. Although ≥2mm represents an established benchmark of tumor growth on serial MRI studies, 2mm of linear tumor growth is unlikely to significantly alter microsurgical outcomes.
Objectives
The objective of the current work was to characterize a clinical threshold in tumor size at which outcomes appreciably worsen.
Methods
Single-institutional retrospective review of a consecutive series of patients with sporadic VS who underwent microsurgical resection between January 2000 and May 2020 was performed. Preoperative tumor size cutpoints were defined in 1mm increments and used to identify optimal size thresholds for three primary outcomes: (1) the ability to achieve gross total resection (GTR); (2) maintenance of normal House-Brackmann (HB) grade I facial nerve function; and (3) preservation of serviceable hearing AAO-HNS class A/B. Optimal size thresholds were obtained by maximizing c-indexes from logistic regression models.
Results
Of 603 patients meeting inclusion criteria, 502 (83%) had tumors with cerebellopontine angle (CPA) extension. CPA tumor size was significantly associated with achieving GTR, postoperative HB grade I facial nerve function, and maintenance of serviceable hearing (all $p<0.001$). The optimal tumor size threshold to distinguish between GTR and less than GTR was 17mm of CPA extension (c-index 0.73). In the immediate postoperative period, the size threshold between HB grade I and HB grade >I was 17mm of CPA extension (c-index 0.65). At most recent evaluation, the size threshold between HB grade I and HB grade >I was 23mm (c-index 0.68) and between class A/B and C/D hearing was 18mm (c-index 0.68). Tumors within 3mm of the 17mm CPA threshold displayed similarly strong c-indices. IAC tumor size was not found to portend worse outcomes for all measures; thus, no thresholds were determined.
Conclusion
The probability of incurring less optimal microsurgical outcomes begins to significantly increase at 14-20mm of CPA extension. While many factors ultimately influence decision making, defining clinically significant growth based on tumor size approaching this threshold range represents a pragmatic, evidence-based approach that moves beyond reflexively recommending treatment for all tumors after detecting ≥2mm of tumor growth on serial MRI studies. These data are particularly relevant in light of evidence demonstrating that an episode of documented tumor growth does not necessarily foreshadow future growth.

10:20 – 10:30 68Ga-DOTATATE PET for Postoperative Gamma Knife Radiosurgery Planning in Patients with Meningioma

Michael Schulder, MD; David J. Park; Daniel Ma; Anuj Goenka

Introduction
Patients with meningiomas are typically treated with maximal safe surgical resection. After subtotal resection or at the time of tumor recurrence, stereotactic radiosurgery (SRS) is often used as the treatment of choice. While contrast-enhanced magnetic resonance imaging (MRI) is typically used for SRS target delineation, differentiating tumor growth from postoperative change can be challenging. 68Ga-DOTATATE, a positron emission tomography (PET) radiotracer targeting the somatostatin receptor type 2 (SSTR2), has been shown to be a reliable biomarker of meningiomas.
Objectives
The aim of this study was to evaluate the impact of 68Ga-DOTATATE on treatment planning for SRS in patients with meningiomas.
Methods
We present a consecutive case series of 13 patients with pathology-proven meningioma who received a 68Ga-DOTATATE PET prior to SRS between April 2019 and April 2021. Treatment planning was done at first using MRI only. The DOTATATE-PET images were then used to assess the accurate identification of tumor.
Results
Ten of the patients had WHO grade 2 meningioma and 3 patients had WHO grade 1 tumor. Nine patients had recurrent meningiomas and 4 patients had newly diagnosed (ND) disease. Overall, the 68Ga-DOTATATE PET scan revealed additional tumor beyond what was seen on MRI in 5/13 patients. In one patient, after 68Ga-DOTATATE PET identified previously unrecognized recurrent meningioma in the resection cavity, SRS was performed (Image 1).The SRS plan was changed to intensity-modulated radiation therapy (IMRT) for 3 patients (2 with recurrent and 1 with ND tumor; Images 2 and 3) and to octreotide injection in 1 patient (with widespread recurrent grade 2 meningioma). In three others, questionable tumor recurrence was seen on MRI, and then confirmed on 68Ga-DOTATATE-PET, but patient observation only was continued due to the small lesion size. In the remaining 5 patients SRS was administered as originally planned.
Conclusion
Incorporation of 68Ga-DOTATATE PET data changed SRS treatment in 5/13 our patients with meningioma after prior surgery. We have made this a routine part of our treatment planning and we recommend this method to optimize the use of SRS or RT in these patients. Further data collection is ongoing.

10:30 – 10:40 Risks and Outcomes Following Repeat Operation for Recurrent Craniopharyngioma

Philip V. Theodosopoulos, MD; Michael William McDermott, MD; Ethan A. Winkler, MD PhD; Jacob Young, MD; Alexander Arash Aabedi, BS; Ryan R. L. Phelps, BA

Introduction
The management of recurrent craniopharyngioma is complex with limited data to guide decision-making. Some reports suggest reoperation should be avoided due to an increased complication profile, while others have demonstrated that safe reoperation can be performed. For other types of skull base lesions, maximal safe resection followed by adjuvant therapy has replaced radical gross total resection due to the favorable morbidity profiles.
Objectives
In this manuscript, we describe our experience with adult patients with craniopharyngioma and investigate outcomes following repeat operation.
Methods

Seventy-one patients underwent resection over a nine-year period for craniopharyngioma and were retrospectively reviewed. Patients were separated into primary resection and reoperation cohorts and stratified by surgical approach (endonasal versus transcranial) and survival analyses were performed based on cohort and surgical approach. Multivariate logistic regression was performed to identify factors associated with tumor recurrence.

Results

Fifty patients underwent primary resection while 21 underwent reoperation for recurrence. Besides lower tumor volumes and prior radiotherapy in the reoperation cohort, there were no significant differences in baseline characteristics. 50 endonasal transsphenoidal surgeries and 21 craniotomies were performed. Surgical approaches were similarly distributed across cohorts. Patients undergoing craniotomy were more likely to have larger tumor volumes and extrasellar lesions. Subtotal resection was achieved in 83% of all cases. There were no differences in extent of resection, visual outcomes, subsequent neuroendocrine function, and complications across cohorts and surgical approaches. Craniotomy was associated with longer lengths of stays in the hospital. The median time to recurrence was 87 months overall and there were no differences by cohort and approach. The five-year survival rate was 81.1% after reoperation versus 93.2% after primary resection. There was a tendency towards lower recurrence among patients with smaller tumors, higher extents of resection, and adjuvant radiotherapy, though this did not reach statistical significance.

Conclusion

Compared to primary resection, reoperation for craniopharyngioma recurrence is associated with similar functional and survival outcomes in light of individualized surgical approaches. Maximal safe resection followed by adjuvant radiotherapy for residual tumor likely preserves vision and endocrine function without sacrificing overall patient survival.

10:40 – 10:50 A Comparison of Treating Surgeon Vs Independent Core Lab Assessment of Post-Aneurysm Treatment Imaging Outcome

Bernard R. Bendok, MD MSCI; Rudy J. Rahme, MD

Introduction

Aneurysm occlusion scales are used to evaluate the outcome of aneurysm treatment and monitor recurrence. However, these scales require a subjective interpretation of objective imaging data with possible discrepancies between reviewers.

Objective

We propose the null hypothesis that the interpretation of aneurysm occlusion on post-treatment imaging is congruent amongst reviewers. Therefore, we analyzed data from the Hydrogel Endovascular Aneurysm Treatment (HEAT) trial to assess the interrater reliability of aneurysm occlusion scales between treating physicians and a third-party blinded core lab.

Methods

The HEAT trial included 600 aneurysms treated with coiling across 46 sites. The treating site and the core lab independently reviewed the immediate post-operative and follow-up imaging using multiple aneurysm occlusion scales. The primary endpoint was the inter-rater reliability of the Raymond-Roy scale (RROC) in the immediate post-operative setting. Secondary endpoints included interrater reliability with the Meyer scale, and with a binary recanalization scale (Yes/no). Factors affecting inter-rater reliability were also assessed.

Results

There was minimal interrater reliability with the RROC at initial post treatment assessment (Weighted Kappa 0.24 [0.22-0.28]). The reliability improved too weak at the 1st and 2nd follow-ups (Weighted Kappa 0.45 [0.43-0.45] and 0.42 [0.34-0.48] respectively). The interrater reliability with the Meyer scale was also minimal (weighted kappa 0.22 [0.13-0.37]). Similarly, the binary recanalization scale had weak interrater reliability at 1st follow-up (0.52[0.42-0.61]) which decreased to minimal at 2nd follow-up (0.34[0.23-0.45]). There was a

trend towards better interrater agreement with lower number of items on a scale. There was significantly higher interrater agreement with smaller aneurysm (±7mm) (0.41 [0.396-0.437] vs 0.37 [0.338-0.391] for aneurysms >7mm) and with posterior circulation aneurysms (0.41[0.37-0.43] vs anterior circulation aneurysms (0.31[0.29-0.32]) with the RROC. Interpretation of diagnostic angiogram yielded higher interrater reliability than magnetic resonance angiography on the binary recanalization scale (0.56 [0.45-0.66] vs 0.34[0.24-0.44].

Conclusion

Analysis of prospectively collected data from a randomized controlled clinical trial supports rejecting the null hypothesis that interpretation of aneurysm occlusion on post-treatment imaging is similar between the treating surgeon and an independent core lab. These results challenge the data and conclusions acquired from studies and trials conducted without an independent core lab.

10:50 – 11:00 The Effects of Prophylactic Lumbar Drainage for the Retrosigmoid Approach on Post-operative Infarct

Nicholas C. Bambakidis, MD; Marte van Keulen; David Penn; Alejandro Rivas; Sarah E Mowry; Maroun Semaan; Cliff Megerian, MD

Introduction

Prophylactic lumbar drainage (LD) with the retrosigmoid approach can improve the safety of vestibular schwannoma resection by increasing brain relaxation and expanding the operative corridor. Removing CSF can decrease damage to cerebellar and brainstem tissue by reducing needed retraction for adequate visualization and tumor removal. We hypothesized that use of LD decreases the incidence of post-operative DWI signal.

Objectives

To determine whether routine use of lumbar drainage aids in the prevention of retraction-associated cerebellar injury during vestibular schwannoma resection.

Methods

A retrospective review of vestibular schwannomas resected via a retrosigmoid approach between 2010 and 2021 was conducted. Post-operative MRI was reviewed for presence of DWI signal in the cerebellum, middle cerebral peduncle (MCP), and brainstem.

Results

In our cohort of 225 patients, we performed 89 retrosigmoid approaches. Eight patients were excluded for lack of imaging and for CSF drainage via lumbar puncture (n=81). Of the remaining cases, 82.7% (n=67) of patients underwent placement of pre-operative LD and 17.3% (n=14) did not. In patients that received LD, 55.2% had some posterior fossa DWI signal present compared to 64.3% of non-LD patients (P=0.570). Of the patients that had LD placement, 48% had cerebellar DWI signal, 34% had MCP DWI signal, and 12% had brainstem DWI signal compared to 57% (P=1.00), 36% (P=1.00), and 21% (P=0.393), respectively in the non-LD cohort.

Conclusion

While these data only indicate a trend towards decreased retraction injury with LD placement, the risks and benefits of increased operative time and complications from LD placement should be weighed against other methods of brain relaxation. Larger randomized trials or meta-analyses may reveal significant results better guiding clinical decision making.

11:00 – 11:10 A Novel Taxonomy for Brainstem Cavernous Malformations

Michael T. Lawton, MD; Joshua Catapano, MD; Kavelin NA Rumalla, BA; Visish M. Srinivasan, MD

Introduction
Pathological taxonomy is a practical tool that has successfully guided clinical decision-making and improved outcomes for patients with brain arteriovenous malformations. Brainstem cavernous malformations (BSCMs) are similarly complex lesions with variability in size, shape, and position. A novel taxonomy for BSCMs is proposed to correlate pathoanatomy, clinical presentation, and surgical approach selection.
Objectives
Pathological taxonomy is a practical tool that has successfully guided clinical decision-making and improved outcomes for patients with brain arteriovenous malformations. Brainstem cavernous malformations (BSCMs) are similarly complex lesions with variability in size, shape, and position. A novel taxonomy for BSCMs is proposed to correlate pathoanatomy, clinical presentation, and surgical approach selection.
Methods
Taxonomy for BSCM is based upon type or location (midbrain, pons, or medulla) and subtype or surface presentation (anterior, posterior, lateral, anterolateral, posterolateral, etc.). The taxonomy was applied to a two-surgeon experience over a 30-year period (1990-2019) with 601 patients who underwent microsurgical resection of BSCMs, of whom 551 had complete data.
Results
BSCM types included midbrain (151, 27%), pontine (323, 59%), and medullary (77, 14%). Five distinct subtypes were defined for midbrain BSCMs: interpeduncular (7, 4.6%), peduncular (37, 24.5%), tegmental (73, 48.3%), quadrigeminal (27, 17.9%), and periaqueductal (7, 4.6%). Six distinct subtypes were defined for pontine BSCMs: basilar (6, 1.9%), peritrigeminal (53, 16.4%), middle peduncular (100, 31.0%), inferior peduncular (47, 14.6%), rhomboid (80, 24.8%), and supraolivary (37, 11.5%). Five distinct subtypes were defined for the medullary BSCMs: pyramidal (3, 3.9%), olivary (35, 45.5%), cuneate (24, 31.2%), gracile (5, 6.5%), and trigonal (10, 12.9%). Each subtype was associated with a recognizable constellation of neurological symptoms/signs. A single surgical approach was preferred for each BSCM subtype in >90% of cases. For example, approaches to midbrain BSCMs included: interpeduncular (transsylvian-interpeduncular), peduncular (transsylvian-transpeduncular), tegmental (lateral supracerebellar-infratentorial [SCIT-Lat]), quadrigeminal (midline SCIT), and periaqueductal (transcallosal-transchoroidal fissure). Favorable outcomes were observed in 81% of patients with follow-up and no significant difference in outcomes were observed between subtypes (P=0.92).
Conclusion
The study confirms our hypothesis that a taxonomy for BSCMs meaningfully guides microsurgical resection strategy. A standardized taxonomy may increase diagnostic acumen at bedside, identify optimal surgical approaches, improve patient outcomes, and clarify clinical communications and publications.

11:10 – 11:15 Wrap-up/ Transition

11:15 – 12:30 Peer Reviewed Abstract Session XII: Trauma and Various Topics
Moderators: Shelly Timmons & Gerry Grant

11:15 – 11:25 Longitudinal Comparison of Learning Objectives and Intent-to-change Statements by Neurosurgical CME Participants

Randy L. Jensen, MD

Introduction
Continuing medical education (CME) activities are required for physician board certification, licensure, and hospital privileges. CME activities are designed to specifically address professional knowledge or practice gaps.

Many CME organizers use statements taken from participants of their "intent-to-change" as data to determine whether the CME activity content achieved a stated learning objective.

Objectives

We examined the longitudinal relationship of learning objectives and intent-to-change data with the hypothesis that it might lead to an understanding the efficacy of CME for closing identified knowledge gaps and for determining unmet needs for future CME planning.

Methods

We performed a retrospective mixed-method thematic content analysis of written and electronic records from specific CME activities. Specifically, the data were first analyzed using a quantitative, deductive content analysis approach to examine whether meeting objectives result in specific intent-to-change statements in learners' evaluation of the CME activity on a direct basis for one year as well as longitudinally over 6 consecutive years. Intent-to-change data that did not align with meeting objectives were further analyzed inductively using a qualitative content analysis approach to explore potential unintended learning themes.

Results

We examined a total of 85 CME activities, averaging 12 – 16 meetings per year over 6 years. This yielded a total of 424 meeting objectives averaging 58 – 83 meeting objectives each year. The objectives were compared with a total of 1950 intent-to-change statements (146 – 588 intent-to-change statements in a given year). Intent-to-change statements were not related to any meeting objective an average of 37.3% of the time. Approximately a quarter of these unmatched statements led to subsequent CME activity new learning objectives. However, the majority of intent-to-change statements were repeated over a number of years without an obvious change in subsequent meeting learning objectives. An examination of CME learning objectives found that 15% of objectives had no intent-to-change statements associated with those objectives. When these learning objectives were analyzed for common themes, we observed that objectives focused on specific (procedural, clinical and medical practice) topics failed to correspond with intent-to-change statements for just one year, while broader (declarative knowledge, academic, scholarly) learning objectives were more likely to lack a corresponding intent-to-change statement for multiple years. On the other hand, CME learning objectives on general topics were more commonly found to be unmatched to intent-to-change statement for multiple years. A number of CME learning objectives are repeated for the same meeting for a number of subsequent years without change. We did not find that repeating a given objective related to unmatched status to intent-to-change statements.

Conclusion

An examination of CME learning objectives and participant intent-to-change statements provides a rich source of information for examination of both meeting planner and learner attitudes and motivation for progression of medical knowledge.

11:25 – 11:35 QuickBrain MRI as a Replacement for CT in First-line Imaging for Select Pediatric Head Trauma

Nathan R. Selden, MD, PhD

Introduction

The current standard of care for initial neuroimaging in injured pediatric patients suspected of having traumatic brain injury (TBI) is computed tomography (CT) which carries risks associated with radiation exposure.

Objectives

The primary objective of this trial was to evaluate the ability of a rapid brain MRI (qbMRI) protocol to detect clinically important traumatic brain injuries (ciTBI) in the emergency department. The secondary objective of this trial was to compare qbMRI to CT in identifying radiographic TBI.

Methods

This was a prospective study of trauma patients less than 15 years of age with suspected TBI at a level 1 pediatric trauma center in Portland, Oregon between August 2017 to March 2019. All patients in whom a head CT was deemed clinically necessary were approached for enrollment to also obtain a qbMRI in the acute setting. Clinically important traumatic brain injury was defined as need for neurological surgery procedure, intubation or pediatric intensive care unit stay greater than 24 hours, total hospital length of stay greater than 48 hours or death.

Results

A total of 73 patients underwent both CT and qbMRI. The median age was 4 years (IQR 1-10 years). Twenty-two patients (30%) of patients had a clinically important traumatic brain injury, and of those, there were 2 deaths (9.1%). QbMRI acquisition time had a median of 4 minutes and 52 seconds (IQR: 3 minutes 49 seconds-5 minutes 47 seconds). QbMRI had sensitivity for detecting clinically important TBI (ciTBI) of 95% (95% CI: 77%-99%). For any radiographic injury, qbMRI had a sensitivity of 89% (95% CI: 78%-94%).

Conclusion

Our results suggest that qbMRI has good sensitivity to detect ciTBI. Further multi-institutional, prospective trials are warranted to either support or refute these findings.

11:35 – 11:45 Effect of Mannitol and Hypertonic Saline on an In Vitro Blood-brain Barrier Model with Human Brain Endothelial Cells

Ajith J. Thomas, MD; Chida Kohei; Franciele Kipper; Khalid Hanafy; Justin M Moore, B.Med.Sci (hon) MD PhD; Christopher S. Ogilvy, MD

Introduction

Mannitol and hypertonic saline (HTS) disrupts the Blood Brain Barrier (BBB).

Objectives

In an in vitro model of BBB, we compared the effect of mannitol and HTS on permeability, signaling pathways, and metabolites.

Methods

Human brain microvascular endothelial cells were grown as monolayers and treated with Mannitol and HTS. Transendothelial electric resistance changes were ascertained with ECIS platform real time. Metabolites were studied using Liquid chromatography and mass spectrometry. Changes in proteins participating in barrier function were assessed by immunohistochemistry. Signaling pathways were assessed with Western blot.

Results

HTS seemed to have a similar effect on permeability as Mannitol. Endothelial nitric oxide synthesis (eNOS) was inactivated via treatment with either mannitol or HTS. Downstream, AMP kinase and p38MAP kinase was activated. Histology demonstrated disruption of adherens junctions and paracellular gap formation by 5 minutes returning to baseline 30 minutes after treatment.

Conclusion

HTS is as effective as Mannitol on permeabilization of the endothelial monolayer.

11:45 – 11:55 Physiological Significance of Intracranial B waves

David W. Newell, MD; Maiken Nedergaard, MD PhD; Rune Aaslid

Introduction

Slow spontaneous cerebral blood flow(CBF) and cerebrospinal fluid oscillations(CSF) driving glymphatic flow in the brain, occur at a similar frequency as intracranial B- waves.

Objective

Our objective was to re-analyze our previously published recordings of B waves, and compare the results to published MRI frequency measurements of CBF and CSF slow wave oscillations.

Methods

B- waves in 20 patients with severe head injury (previously reported), and 6 additional head injury patients, were analyzed, including middle cerebral artery(MCA) velocity using transcranial Doppler(TCD), and ICP. The frequency was compared to published spontaneous fluctuations of CBF measured using functional MRI(f-MRI) BOLD sequence, EEG, and CSF movement using MRI, in humans.

Results

Frequency analysis revealed MCA velocity and ICP fluctuations during B waves showed cross-correlation of the -d/dt FV vs. the -d/dt ICP signals show a similar correlation and time relationship as the published -d/dt MRI BOLD descriptions. In 26 patients demonstrating clear B-wave activity, the FV signal had maximum activity at 0.025-0.03Hz, and time derivative maximum at 0.035 Hz. The frequency range was between 0.3-4 cycles per minute or 0.024-0.067 Hz.

Conclusion

Re-analysis of our B wave measurements, compared to spontaneous f-MRI BOLD, EEG, and CSF oscillations in the brain, indicate that both methods (TCD and MRI) are measuring a similar physiologic process. The slow oscillations causing intracranial B waves can provide a driving force for CSF movement and glymphatic flow of fluid in the brain, even when the ICP is not significantly affected, and have important clinical implications.

11:55 – 12:05 Human Brain Growth: Avoiding the Mismeasure of Man

Steven J. Schiff, MD, PhD

Introduction

The study of brain size and growth has a long and contentious history, yet normal brain volume development has yet to be fully described. In particular, the normal brain growth and cerebrospinal fluid (CSF) accumulation relationship is critical to characterize because it is impacted in numerous conditions of early childhood where brain growth and fluid accumulation are affected such as infection, hemorrhage, hydrocephalus, and a broad range of congenital disorders.

Objectives

This study aims to describe normal brain volume growth, with respect to age, sex, and cerebrospinal accumulation.

Methods

We analyzed 1067 magnetic resonance imaging (MRI) scans from 505 healthy pediatric subjects from birth to age 18 to quantify component and regional brain volumes. The volume trajectories were compared between the sexes and hemispheres using Smoothing Spline ANOVA. Population growth curves were developed using Generalized Additive Models for Location, Scale, and Shape.

Results

Brain volume peaked at 10-12 years of age. Males exhibited larger age-adjusted total brain volumes than females, and body size normalization procedures did not eliminate this difference. The ratio of brain to CSF volume, however, we discovered a novel universal age-dependent relationship independent of sex or body size.

Conclusion

These findings enable the application of normative growth curves in managing a broad range of childhood disease where cognitive development, brain growth, and fluid accumulation are interrelated. With the advent of new inexpensive commercial low-field MRI, the prospect of incorporating normative brain growth into the management of many neurosurgical conditions of childhood is now widely feasible.

12:05 – 12:15 Lumbar fusion without General Anesthesia, Lessons Learned from the First 300 cases

Michael Y. Wang, MD, FAANS

Introduction

The growth in lumbar fusion surgery with the aging population has resulted in increased costs, morbidity due to complications, and burdens on the health care system. This has in turn driven the need for more cost-effective ways to successfully treat degenerative spine pathologies. One methodology for confronting these challenges is by performing MIS (minimally invasive surgery) as a response to these challenges.

Objectives

This retrospective study was undertaken to evaluate if using an ultra-MIS approach to treat common spinal pathologies could result in an effective and cost efficient way of treating large volumes of patients.

Methods

Clinical and radiographic outcomes with a consecutive series of 300 patients was retrospectively reviewed. All patients underwent endoscopic lumbar fusion without the use of general anesthesia. The results measured included clinical outcome metrics using PROM's, radiographic measures of successful fusion, and acute care data, including intraoperative outcomes, complication rates, cost of surgery, and rates of surgical revision.

Results

1-, 2-, and 3-level lumbar fusion surgeries were identified and interventions were associated with clinical improvement exceeding the MCID (minimal clinically important difference) with a reduction on average of 23 points on the ODI (Oswestry disability index). Operative times averaged 79 minutes with 80cc of blood loss. Length of stay averaged 1.4 + 1.1 days. 5 patients had to be converted to general anesthesia, and none were converted to open surgery. There was one case of a delayed nonunion requiring surgical revision. When compared with more traditional MIS fusion, an average of 15.2% cost savings.

Conclusion

Utilizing MIS endoscopic awake lumbar fusion offers the promise of a more efficient, cost-effective surgery for treating select spinal pathologies. However, the generalizability of these results will require multi-institutional studies.

12:15 – 12:25 Clinical and Genomic Predictors of Seizures in Meningiomas

Jennifer A. Moliterno Gunel, MD; Trisha Gupte; Lan Jin; Mark W Youngblood, MD PhD; Robert Fulbright; Zeynep Erson Omay, PhD

Introduction

The association of seizures with meningiomas is poorly understood.

Objectives

We sought to investigate the relationship between seizures, clinical variables and the underlying meningioma genomic subgroup.

Methods

Clinical and genomic sequencing data on 394 patients treated for meningioma were reviewed and analyzed using logistic regression models and mediation analyses.

Results

Seventeen percent of the cohort presented with preoperative seizures. In univariate analysis, patients with preoperative seizures were more likely to have tumors with a somatic NF2 mutation ($p = 0.020$), WHO II or III grade ($p = 0.029$), atypical histology ($p = 0.004$), edema ($p < 0.001$), brain invasion ($p = 0.009$), and worse progression free survival (HR 2.68, 95% CI 1.30-5.50). In multivariate analysis, edema (OR 3.11, $p=0.003$) and atypical histology (OR 2.00, $p=0.041$) were positive predictors of preoperative seizures, while genomic subgroup was not, such that the effect of an NF2 mutation was indirectly mediated through atypical histology and edema ($p=0.012$). Seizure freedom was achieved in 83.3% of patients. Preoperative seizures (OR 3.54,

p=0.009), recurrent tumors (OR 2.89, p=0.035), and tumors requiring postoperative radiation (OR 2.82, p=0.033) were significant predictors of postoperative seizures in multivariate analysis.

Conclusion

NF2 mutations in meningioma are significantly associated with preoperative seizures, with its effect mediated through edema and atypical histology. Patients who undergo radiation and/or have recurrence are at risk for postoperative seizures, regardless of the extent of resection. Preoperative seizures may portend a more aggressive molecular entity and challenging clinical course with a higher risk of recurrence.

12:25 – 12:30 Wrap-up/ Transition

12:30 Closing Remarks & Meeting Adjourn

MEMBERS

	ELECTED	STATUS
HIROSHI ABE (Yoko) University of Hokkaido hiro-abe@dream.ocn.ne.jp	1999	SENIOR CORRESPONDING | RETIRED
AVIVA ABOSCH (Joseph Dowd) University of Nebraska aviva.abosch@unmc.edu	2015	ACTIVE
P. DAVID ADELSON (Barbara) Phoenix Children's Hospital dadelson@phoenixchildrens.com	2011	ACTIVE
MANISH AGHI University of California, San Francisco manish.aghi@ucsf.edu	2016	ACTIVE
FELIPE C. ALBUQUERQUE (Ruth Bristol) Barrow Neurological Institute felipe.albuquerque@barrowbrainandspine.com	2013	ACTIVE
EBEN ALEXANDER, III (Karen Newell) ebenalex3@gmail.com		SENIOR | RETIRED
CARGILL H. ALLEYNE, Jr. (Audrey) Medical College of Georgia at Augusta University ca@augusta.edu	2017	ACTIVE

SEPIDEH AMIN-HANJANI University of Illinois at Chicago hanjani@uic.edu	2017	ACTIVE
MICHAEL L. J. APUZZO University of Southern California apuzzo@usc.edu	1988	SENIOR | RETIRED
HAJIME ARAI (Jun) Juntendo University mogawa@juntendo.ac.jp	2012	CORRESPONDING
MIGUEL ARRAEZ-SANCHEZ (Cinta Manrique) Carlos Haya University Hospital marraezs@commalaga.com	2010	CORRESPONDING
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TAE SUNG PARK (Mee Aeng) Washington Univ., St. Louis Children's Hospital park@nsurg.wustl.edu	1996	SENIOR
RUSSEL H. PATTERSON, Jr. (Julie) Cornell University Medical College patt10019@verizon.net	1971	SENIOR | RETIRED
SYDNEY J. PEERLESS (Ann) speerless@earthlink.net	1977	SENIOR | RETIRED
JOHN D. PICKARD (Mary) University Cambridge jdpsecretary@medschl.cam.ac.uk	2001	SENIOR CORRESPONDING
DAVID G. PIEPGRAS (Jane) Mayo Clinic piepgras.david@mayo.edu	1987	SENIOR | RETIRED

LAWRENCE H. PITTS (Mary) University of California, San Francisco lhpitts@yahoo.com	1997	SENIOR |RETIRED
IAN F. POLLACK (Connie) Children's Hospital of Pittsburgh ian.pollack@chp.edu	2012	ACTIVE
BRUCE E. POLLOCK (Kristen) Mayo Clinic pollock.bruce@mayo.edu	2004	ACTIVE
WAI SANG POON (Gillian Kew) Chinese University of Hong Kong wpoon@surgery.cuhk.edu.hk	2008	CORRESPONDING
A. JOHN POPP (Margaret Vosburgh) Stanford University SOM ajpmd123@gmail.com	2001	SENIOR | RETIRED
ROBERT W. PORTER (Dean) University of California, Irvine rporter785@aol.com	1962	SENIOR | RETIRED
KALMON D. POST (Linda Farber-Post) Mount Sinai Medical Center kalmon.post@mountsinai.org	1995	SENIOR
CHARLES J. PRESTIGIACOMO (Cynthia) University of Cincinnati cjp9@me.com	2010	ACTIVE
DONALD O. QUEST New York Neurological Institute doq1@columbia.edu	1986	SENIOR | RETIRED

ANDREAS RAABE Inselspital andreas.raabe@insel.ch	2019	CORRESPONDING
COREY RAFFEL (Kathy) University of California, San Francisco raffelc@neurosurg.ucsf.edu	1998	SENIOR | RETIRED
GANESH RAO M.D. Anderson Cancer Center grao@mdanderson.org	2016	ACTIVE
ROBERT A. RATCHESON (Peggy) Case Western Reserve University rar@case.edu	1986	SENIOR | RETIRED
JEAN M. REGIS Hospital d'adulte de la Timone jean.regis@ap-hm.fr	2019	CORRESPONDING
DANIEL K. RESNICK (Rachel Groman) University of Wisconsin-Madison resnick@neurosurgery.wisc.edu	2011	ACTIVE
HANS-JUERGEN REULEN (Ute) LMU Munich hjreulen@gmx.de	1998	SENIOR CORRESPONDING
ALI R. REZAI University of West Virginia ali.rezai@hsc.wvu.edu	2014	ACTIVE
J. CHARLES RICH jcrich1709@gmail.com	1987	SENIOR | RETIRED

HOWARD A. RIINA (Anne) NYU Langone Medical Center howard.riina@nyumc.org	2008	ACTIVE
DAVID W. ROBERTS (Kathryn) Dartmouth-Hitchcock Medical Center david.w.roberts@dartmouth.edu	1996	SENIOR | RETIRED
JON H. ROBERTSON (Carol Anne) Semmes-Murphey Clinic jrobertson@semmes-murphey.com	1992	SENIOR | RETIRED
SHENANDOAH ROBINSON (Alan R. Cohen) Johns Hopkins University srobin81@jhmi.edu	2010	ACTIVE
GERALD "RUSTY" RODTS, Jr. (Kelly) Emory University School of Medicine grodts@emory.edu	2003	ACTIVE
ROBERT H. ROSENWASSER (Deborah August) Thomas Jefferson University Hospital robert.rosenwasser@jefferson.edu	1996	SENIOR
JAMES T. RUTKA (Mari) Hospital for Sick Children, University of Toronto james.rutka@sickkids.ca	1996	SENIOR
MADJID SAMII International Neuroscience Institute samii@inihannover.de	1996	SENIOR CORRESPONDING
JOHN H. SAMPSON (Mary) Duke University Medical Center john.sampson@duke.edu	2013	ACTIVE

DUKE S. SAMSON (Patricia Bergen) University of Texas, Southwestern Medical School duke.samson@utsouthwestern.edu	1994	SENIOR | RETIRED
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STEVEN J. SCHIFF (Eleanor) Pennsylvania State University steve.j.schiff@gmail.com	2014	ACTIVE
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YONG-KWANG TU (Charlotte) National Taiwan University Hospital yktu@ntu.edu.tw	2007	SENIOR CORRESPONDING

UGUR TURE Yeditepe University School of Medicine drture@yahoo.com	2016	CORRESPONDING
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MICHAEL A. VOGELBAUM (Judith Rosman) Cleveland Clinic Foundation Michael.Vogelbaum@moffitt.org	2012	ACTIVE
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BRYCE K. A. WEIR (Mary Lou) University of Alberta & Chicago brycekeithweir@gmail.com	1984	SENIOR | RETIRED
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JEFFREY H. WISOFF (Deborah) NYU Langone Medical Center jhw1@nyulangone.org	2012	SENIOR
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DANIEL YOSHOR (Shira) University of Pennsylvania Daniel.yoshor@pennmedicine.upenn.edu	2016	ACTIVE

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ERIC L. ZAGER (Marirosa Colon) University of Pennsylvania Hospital Eric.Zager@pennmedicine.upenn.edu	2006	SENIOR
NICHOLAS T. ZERVAS (Thalia) Massachusetts General Hospital nzervas@partners.org	1972	SENIOR | RETIRED
GREGORY J. ZIPFEL (Mary Jo) Washington University School of Medicine zipfelg@wustl.edu	2013	ACTIVE

IN MEMORIUM
DECEASED MEMBERS

	ELECTED	DECEASED
EBEN ALEXANDER, JR.	1950	2004
JOAO (JOHN) ANTUNES	2001	2016
JAMES R. ATKINSON	1970	1978
PERCIVAL BAILEY (Honorary)	1960	1973
GEORGE BAKER	1940	1993
H. THOMAS BALLANTINE, JR	1951	1996
DONALD P. BECKER	1990	2020
WILLIAM F. BESWICK	1959	1971
EDWIN B. BOLDREY	1941	1988
E. HARRY BOTTERELL	1938	1997
ROBERT BOURKE	1983	1996
SPENCER BRADEN, Founder	1938	1969
F. KEITH BRADFORD	1938	1971
JEAN BRIHAYE	1975	1999
JACOB S. BROADKY	1977	2019
HOWARD BROWN	1939	1990
KARL-AUGUST BUSHE	1972	1999
FERNANDO CABIESES	1966	2009
JUAN CARDENAS	1966	1996

HARVEY CHENAULT	1949	2006
SHELLEY CHOU	1974	2001
JUAN CARLOS CHRISTENSEN	1970	2003
GALE CLARK	1970	1996
W. KEMP CLARK	1970	2007
DONALD COBURN	1938	1988
WILLIAM FRANCIS COLLINS JR.	1963	2009
EDWARD CONNOLLY	1972	2015
JAMES W. CORRELL	1966	2004
WINCHELL McK. CRAIG (Honorary)	1942	1960
EDWARD DAVIS	1949	1988
COURTLAND HARWELL DAVIS, JR.	1967	2018
JACQUES C. DE VILLIERS	1986	2015
RICHARD DESAUSSURE, JR	1962	2008
HERMANN DIETZ	1980	2016
PEARDON DONAGHY	1970	1991
CHARLES DRAKE	1958	1998
FRANCIS ECHLIN	1944	1988
DEAN ECHOLS, Founder	1938	1991
GEORGE EHNI	1964	1986
ARTHUR ELVIDGE	1939	1985
THEODORE ERICKSON	1940	1986
JOSEPH EVANS, Founder	1938	1985
WILLIAM H. FEINDEL	1959	2014
ROBERT FISHER	1955	2003
ELDON L FOLTZ	1960	2013
RICHARD A. R. FRASER	1976	2017
JOHN FRENCH	1951	1989
LYLE FRENCH	1954	2004
JAMES GALBRAITH	1947	1997
HENRY GARRETSON	1973	2007

F. JOHN GILLINGHAM	1962	2020
SIDNEY GOLDRING	1964	2004
PHILIP GORDY	1968	2017
EVERETT GRANTHAM	1942	1997
JOHN GREEN	1953	1990
JAMES GREENWOOD, JR.	1952	1992
WESLEY GUSTAFSON	1942	1975
WALLACE HAMBY	1941	1999
HANNIBAL HAMLIN	1949	1982
JOHN HANBERY	1959	1996
JOHN HANKINSON	1973	2007
GRIFFITH R. HARSH, III	1980	2019
MAJOR GEN. GEORGE HAYES	1962	2002
MARK PETER HEILBRUN	1984	2010
E. BRUCE HENDRICK	1968	2001
JESS HERRMANN	1938	1944
HENRY HEYL	1951	1975
JULIAN HOFF	1975	2007
HAROLD HOFFMAN	1982	2004
EDGAR HOUSEPIAN	1976	2014
WILLIAM HUNT	1970	1999
OLAN HYNDMAN	1942	1966
FABIAN ISMAT	1989	2019
SHOZO ISHII	1975	2012
KENNETH JAMIESON	1970	1976
JOHN JANE, SR.	2011	2015
PETER JANNETTA	1994	2016
SIR GEOFFREY JEFFERSON (Honorary)	1951	1961
HANS-PETER JENSEN	1980	2000
RICHARD JOHNSON	1974	1997
WILLIAM KEITH, Founder	1938	1987

ROBERT KING	1958	2008
KATSUTOSHI KITAMURA	1970	2005
ROBERT KNIGHTON	1966	2004
RICHARD KRAMER	1978	2001
HUGO KRAYENBUHL (Honorary)	1974	1985
KRISTIAN KRISTIANSEN	1967	1993
THEODORE KURZE	1967	2002
LAURI LAITINEN	1972	2007
THOMAS LANGFITT	1971	2005
SANFORD LARSON	1989	2012
GUY LAZORTHES (Honorary)	1973	2018
WALPOLE LEWIN	1973	1980
RAEBURN LLEWELLYN	1963	2009
VALENTINE LOGUE (Honorary)	1974	2000
H.C. RUEDIGER LORENZ	1998	2008
HERBERT LOURIE	1965	1987
ALFRED LUESSENHOP	1977	2009
WILLEM LUYENDIJK	1973	1995
ROBERT MACIUNAS	1999	2011
ERNEST MACK	1956	2000
M. STEPHEN MAHALEY	1972	1992
LEONARD MALIS	1973	2005
GEORGE MALTBY	1942	1988
FRANK MARGUTH	1978	1991
DONALD MATSON	1950	1969
FRANK MAYFIELD, Founder	1938	1991
AUGUSTUS McCRAVEY	1944	1990
KENNETH McKENZIE (Honorary)	1960	1964
ROBERT L. McLAURIN	1955	2015
J. MICHAEL MCWHORTER	1989	2004
WILLIAM MEACHAM	1952	1999

JAMES MEREDITH	1946	1962
J. DOUGLAS MILLER	1988	1995
W. JASON MIXTER (Honorary)	1951	1968
EDMUND MORRISSEY	1941	1986
JOHN F. (SEAN) MULLAN	1963	2015
FRANCIS MURPHEY, Founder	1938	1994
BLAINE NASHOLD, JR.	1967	2014
GOSTA NORLEN (Honorary)	1973	1985
FRANK NULSEN	1956	1994
SIXTO OBRADOR (Honorary)	1973	1978
GUY ODOM	1946	2001
ROBERT OJEMANN	1968	2010
EDWARD OLDFIELD	1975	2017
PIETRO PAOLETTI	1989	1991
ANDREW T. PARSA	2012	2015
WILDER PENFIELD (Honorary)	1960	1979
HELMUT PENZHOLZ	1978	1985
PHANOR PEROT, JR.	1970	2011
BERNARD PERTUISET (Honorary)	1986	2000
BYRON CONE PEVEHOUSE	1964	2010
HANS-WERNER PIA	1978	1986
J. LAWRENCE POOL	1940	2004
ROBERT PUDENZ	1943	1998
JOHN E. RAAF, Founder	1938	2000
B. RAMAMURTHI	1973	2003
AIDAN RANEY	1946	2002
RUPERT B. RANEY	1939	1959
JOSEPH RANSOHOFF	1965	2001
THEODORE RASMUSSEN	1947	2002
BRONSON RAY (Honorary)	1992	1993
DAVID REEVES	1939	1970

DAVID REYNOLDS	1964	1978
ALBERT RHOTON, JR.	1984	2016
HUGO RIZZOLI	1973	2014
THEODORE ROBERTS	1976	2007
JAMES T. ROBERTSON	1971	2019
R. C. L. ROBERTSON	1946	1985
STEWART ROWE	1938	1984
KEIJI SANO (Honorary)	1975	2011
RICHARD SCHNEIDER	1970	1986
KURT-FRIEDRICH SCHURMANN	1978	2005
HENRY SCHWARTZ	1942	1988
WILLIAM SCOVILLE	1944	1984
R. EUSTACE SEMMES (Honorary)	1955	1982
C. HUNTER SHELDEN	1941	2003
JAMES C. SIMMONS	1975	2019
ROBERT SMITH	1989	2003
SAMUEL SNODGRASS	1939	1975
GLEN SPURLING (Honorary)	1942	1968
C. WILLIAM STEWART	1948	1948
KENICHIRO SUGITA	1988	1994
THORALF SUNDT, JR.	1971	1992
ANTHONY SUSEN	1965	2008
HENDRIK SVIEN	1957	1972
HOMER SWANSON	1949	1987
WILLIAM SWEET	1950	2001
LINDSAY SYMON	1982	2019
SUZIE CUNNINGHAM TINDALL	1990	2016
JOHN S. TYTUS	1967	2011
ALFRED UIHLEIN	1950	1990
KJELD VAERNET	1970	2006
JOHN VAN GILDER	1980	2007

A. EARL WALKER	1938	1995
EXUM WALKER	1938	2001
ARTHUR WARD, JR.	1953	1997
E. SYDNEY WATKINS	1975	2012
THOMAS WEAVER, JR.	1943	1985
W. KEASLEY WELCH	1957	1996
BENJAMIN WHITCOMB	1947	1998
LOWELL E. WHITE, JR.	1971	2018
ROBERT WILKINS	1973	2017
CHARLES B. WILSON	1966	2018
BARNES WOODHALL	1941	1985
FRANK WRENN	1973	1990
DAVID YASHON	1972	2016