



THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

84TH ANNUAL MEETING

SEPTEMBER 28 – OCTOBER 1, 2022



American
Association of
Neurological
Surgeons

Jointly Provided by the AANS

FUTURE MEETINGS

October 4-7, 2023

The Cloister at Sea Island
Sea Island, Georgia

October 16-19, 2024

The Ritz-Carlton
Half Moon Bay, CA

Mark your calendars now!

GENERAL INFORMATION

HOTEL INFORMATION

THE BROADMOOR

1 Lake Avenue, Colorado Springs, Co 80906

844-602-3343



REGISTRATION LOCATION:

WWW.AMERICANACADEMYNS.ORG

REGISTRATION:

On-site Registration is currently open.

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

A Special Thanks to the following exhibitors supporting the

**THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
84TH ANNUAL SCIENTIFIC MEETING**

Please take time to visit with them during the Break

- BrainLab
- Elekta
- Integra LifeSciences
- Leica Microsystems
- Stryker
- Zap Surgical
- Carl Zeiss Meditec, US





THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY
84TH ANNUAL SCIENTIFIC MEETING

PROGRAM SUMMARY

WEDNESDAY, SEPTEMBER 28

1:00 – 6:30 pm	Registration	Pre–Function Space of Broadmoor Hall B
3:30 – 5:00 pm	Executive Committee Meeting	El Pomar Room
6:30 – 8:30 pm	Opening Reception	West Tower Lawn

THURSDAY, SEPTEMBER 29

6:00 am – 4:00 pm	Registration	Pre–Function Space of Broadmoor Hall B
6:30 – 7:30 am	Members Breakfast & Business Meeting (Voting Membership Only)	Penrose Room
7:00 – 10:00 am	Guest & Spouse/Partner Breakfast	Donald Ross
7:30 – 7:35 am	Welcoming Remarks	Broadmoor Hall B
7:35 – 7:45 am	Round Robin Roundup!	Broadmoor Hall B
7:45 – 9:00 am	Peer Reviewed Abstract Session I: Spine Clinical Science	Broadmoor Hall B
9:00 – 9:55 am	Peer Reviewed Abstract Session II: Cerebrovascular Cutting Edge	Broadmoor Hall B
9:55 – 10:10 am	Break	Broadmoor Hall E
10:10 – 11:05 am	Peer Reviewed Abstract Session III: Clinical Science of Brain Tumors	Broadmoor Hall B
11:05 – 11:40 am	Peer Reviewed Abstract Session IV: AI Tools and Applications in Neurosurgery	Broadmoor Hall B
11:40 – 11:55 am	Break	Broadmoor Hall E
11:55 am – 12:45 pm	Guest Keynote Speaker	Broadmoor Hall B
1:30 – 4:30 pm	Academy Spine Emerging Investigators’ Program	Broadmoor Hall F
6:30 – 9:30 pm	Dinner	Lakeside Terrace

FRIDAY, SEPTEMBER 30

6:00 am – 4:00 pm	Registration	Pre–Function Space of Broadmoor Hall B
6:30 – 7:30 am	Members Breakfast & Business Meeting (Voting Membership Only)	Penrose Room
7:00 – 10:00 am	Guest & Spouse/Partner Breakfast	Donald Ross
7:30 – 7:35 am	Welcoming Remarks	Broadmoor Hall B
7:35 – 8:40 am	Peer Reviewed Abstract Session V: Basic Science	Broadmoor Hall B
8:40 – 9:35 am	Peer Reviewed Abstract Session VI: Functional	Broadmoor Hall B
9:35 – 9:50 am	Break	Broadmoor Hall E
9:50 – 10:55 am	Peer Reviewed Abstract Session VII: Technology and Translation	Broadmoor Hall B
10:55 – 11:50 am	Peer Reviewed Abstract Session VIII: Other and Education	Broadmoor Hall B
11:50 am – 12:00 pm	Break	Broadmoor Hall E
12:00 – 12:45 pm	Presidential Address	Broadmoor Hall B
1:30 – 4:30 pm	Joint Academy Emerging Investigators’ Program	Broadmoor Hall F
6:15 pm	Shuttle to Cheyenne Lodge Starts Service	Broadmoor South
6:30 – 9:30 pm	Gala Dinner (Black Tie Optional)	Cheyenne Lodge

SATURDAY, OCTOBER 1

7:00 – 12 pm	Registration	Pre–Function Space of Broadmoor Hall B
7:00 – 9:30 am	Members & Guests Breakfast	Main Ballroom
7:30 – 8:20 am	Special Abstract Session: The Oldfield Session	Broadmoor Hall B
8:20 – 9:10 am	Academy Award Presentation and Lecture	Broadmoor Hall B
9:10 – 9:25 am	Break	Broadmoor Hall E
9:25 – 10:10 am	Peer Reviewed Abstract Session IX: Brain Tumor	Broadmoor Hall B
10:10 – 10:55 am	Peer Reviewed Abstract Session X: Pediatrics	Broadmoor Hall B
10:55 – 11:10 am	Break	Broadmoor Hall E
11:10 am – 12:15 pm	Peer Reviewed Abstract Session XI: Clinical Science	Broadmoor Hall B
12:15 pm	Closing Remarks & Meeting Adjourn	Broadmoor Hall B



THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

2021 – 2022 OFFICERS

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James M. Markert, MD

PRESIDENT – ELECT

Frederick G. Barker II, MD

VICE PRESIDENT

Daniel Yoshor, MD

SECRETARY

E. Sander Connolly Jr., MD

TREASURER

Shenandoah Robinson, MD

HISTORIAN

Michael Schulder, MD

PAST PRESIDENT

Douglas Kondziolka, MD

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James M. Markert, MD

Frederick G. Barker II, MD

Douglas Kondziolka, MD

Daniel Yoshor, MD

E. Sander Connolly Jr., MD

Shenandoah Robinson, MD

Michael Schulder, MD

Howard A. Riina, MD

2021 – 2022 COMMITTEES

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Kendall Lee, MD, PhD
Michael Vogelbaum, MD, PhD

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Gerald Grant, MD
Praveen Mummaneni, MD

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E. Antonio “Nino” Chiocca, MD
Linda M. Liao, MD
James M. Markert, MD

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Aviva Abosch, MD, PhD

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James M. Markert, MD
E. Sander Connolly Jr., MD
Shenandoah Robinson, MD
Mark Johnson, MD, PhD
Nicholas Theodore, MD

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Douglas Kondziolka, MD
Jacques Morcos, MD

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Douglas Kondziolka, MD – Chair
James M. Markert, MD
Frederick G. Barker II, MD

SCIENTIFIC PROGRAM COMMITTEE

Alexandra Golby, MD – Chair
Jacques Morcos, MD
Daniel Resnick, MD
Zohar Ghogawala, MD

COMMUNICATIONS & ROUND ROBIN COMMITTEE

QUARTERLY NEWSLETTER

Mark N. Hadley, MD

LOCAL ARRANGEMENTS

Randy Jensen, MD, PhD – Chair

AANS JOINT SPONSORSHIP EDUCATION REPRESENTATIVE

Zohar Ghogawala, MD – Chair

WFNS DELEGATES

Jacques Morcos, MD – Senior Delegate
Nelson Oyesiku, MD, PhD – Second Delegate

RESEARCH ADVISORY COMMITTEE

Gregory Zipfel, MD – Chair
John Sampson, MD, PhD
Robert Gross, MD, PhD
Amy Heimberger, MD
Howard A. Riina, MD

PAST-PRESIDENTS

Dean H. Echols	1938 - 39	Byron C. Pevehouse	1982
Spence Braden	1940	Sidney Goldring	1983
Joseph P. Evans	1941	Russel H. Patterson, Jr	1984
Francis Murphey	1942	Thomas Langfitt	1985
Frank H. Mayfield	1943	Phanor L. Perot, Jr	1986
A. Earl Walker	1944	Shelley N. Chou	1987
Barnes Woodhall	1946	James T. Robertson	1988
William S. Keith	1947	Thoralf M. Sundt, Jr.	1989
Howard A. Brown	1948	Robert Ojemann	1990
John Raaf	1949	Nicholas Zervas	1991
E. Harry Botterell	1950	Henry Garretson	1992
Wallace B. Hamby	1951	George Tindall	1993
Henry G. Schwartz	1952	William A. Buchheit	1994
J. Lawrence Pool	1953	David L. Kelly, Jr	1995
Rupert B. Raney	1954	John M. Tew, Jr	1996
David L. Reeves	1955	Julian T. Hoff	1997
Stuart N. Rowe	1956	Edward Connolly	1998
Arthur R. Elvidge	1957	J. Charles Rich	1999
Jess D. Herrmann	1958	George A. Ojemann	2000
Edwin B. Boldrey	1959	Roberto C. Heros	2001
George S. Baker	1960	Donald O. Quest	2002
C. Hunter Shelden	1961 - 62	David G. Piegras	2003
Samuel R. Snodgrass	1963	Volker K.H. Sonntag	2004
Theodore Rasmussen	1964	Martin B. Camins	2005
Edmund J. Morrissey	1965	L. Nelson Hopkins	2006
George Maltby	1966	Richard Morawetz	2007
Guy L. Odom	1967	Robert F. Spetzler	2008
James G. Galbraith	1968	Ralph G. Dacey, Jr.	2009
Robert H. Pudenz	1969 - 70	Steven Giannotta	2010
William B. Scoville	1971	Robert A. Solomon	2011
Robert L. McLaurin	1972	James T. Rutka	2012
Lyle A. French	1973	Griffith R. Harsh	2013
Benjamin B. Whitcomb	1974	Fredric B. Meyer	2014
John R. Green	1975	Mitchel S. Berger	2015
William H. Feindel	1976	Mark N. Hadley	2016
William H. Sweet	1977	William T. Couldwell	2017
Arthur A. Ward	1978	Daniel L. Barrow	2018
Robert B. King	1979	E. Antonio Chiocca	2019
Eben Alexander, Jr.	1980	M. Sean Grady	2020
Joseph Ransohoff II	1981	Douglas Kondziolka	2021

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
Robert Pudenz	1965	Robert F. Spetzler	2006
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	Michael W. McDermott	2021
John F. Mullan	1982		
Hugo V. Rizzoli	1983		
James W Correll	1984		
E. Bruce Hendrick	1985		

PAST SECRETARY-TREASURERS

Francis Murphey	1938 - 1940
A. Earl Walker	1941 - 1943
Theodore C. Erickson	1944 - 1947
Wallace B. Hamby	1948 - 1950
Theodore B. Rasmussen	1951 - 1953
Eben Alexander	1954 - 1957
Robert L. McLaurin	1958 - 1962
Edward W. Davis	1963 - 1965
Robert G. Fisher	1966 - 1968
Byron C. Pevehouse	1969 - 1972

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	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 - 2019

OLDFIELD LECTURE

Russell Lonser	2018
Amy Heimberger	2019
Frederick G. Barker	2021
Todd Hollon	2022

MEETINGS OF THE ACADEMY

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, New York	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, West Virginia	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, Georgia	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, Arizona	October 19 - 22, 2011
The Chatham Bars Inn, Chatham, Massachusetts	October 17 - 20, 2012
The Resort at Pelican Hill, Newport Coast, California	September 25 - 28, 2013
WaterColor Inn & Resort, Santa Rosa Beach, Florida	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, California	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
The Inn at Spanish Bay, Pebble Beach, California	September 22 - 25, 2021

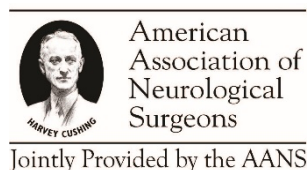


MISSION STATEMENT

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



LEARNING OBJECTIVES

- Describe the implications of artificial intelligence (AI) for brain imaging and exploring functional organization of the human brain
- Discuss new developments of surgical and other therapies for management of spinal pathology based on randomized trials
- Identify opportunities for enhancing diversity and scientific exploration in neurosurgical education
- Define the impact of novel neuroscience performed by neurosurgeons which leverages the unique access to the central nervous system

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 13.75 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 84th 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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The AANS and the American Academy of Neurological Surgery control the content and production of this CME activity and attempt to ensure the presentation of balanced, objective information. In accordance with the Standards for Commercial Support established by the ACCME, faculty, abstract reviewers, paper presenters/authors, co-authors, planning committee members, staff and any others involved in planning the educational content and the significant others of those mentioned must disclose any relationships they or their co-authors have with commercial interests which may be related to their content. The ACCME defines “relevant financial relationships” as financial relationships in any amount occurring within the past 12 months that create a conflict of interest.

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Faculty, planners of educational content and staff (and the significant others of those mentioned) who have disclosed a relationship with commercial interests whose products may have a relevance to their presentation are listed below.

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.

Name	Type of Disclosure	Entity/Company
P. David Adelson	Consulting Fee Speakers Bureau	Medtronic Integra, LivaNova
Cargill Alleyne	Receipt of IP/Patent	Co-ownership
Jeffrey Bruce	Future Stock Options	Theracle, Inc.
Terry Burns	Consulting Fee Contracted Research Future Stock Options	Alector, Predicine Abbvie, Aminex Therapeutics, Metvital Neurametrix
E. Antonio Chiocca	Consulting Fee Future Stock Options Own Stocks Receipt of IP/Patent Royalty Stock Options	Biogen, Candel, DNATRIX, Genenta, Insightec, Voyager Immunomic Seneca Mass General Brigham Brave Bio, Candel Bionaut, DNATRIX, Synthetic Biologics, Ternalys
Kevin Foley	Consulting Fee Own Stocks Receipt of IP/Patent Royalty	Medtronic Accelus, Companion Spine, Digital Surgery Systems, Discgenics, DuraStat, LaunchPad Medical, Medtronic, Neurogami, NuVasive, nView Medical, Practical Navigation/Fusion Robotics, RevBio, SpineWave, Tissue Differentiation Intelligence, Triad Life Sciences, True Digital Surgery, Vori Health Discgenics, Medtronic, NuVasive Medtronic

Name	Type of Disclosure	Entity/Company
Peter Gerszten	Fees for Non-CME Services	Zimmer Biomet
Murat Gunel	Own Stocks Stock Options	4Catalyzer 4Catalyzer, AI Therapeutics, Hyperfine
Benjamin Hendricks	Consulting Fee	Medtronic, Inc.
Todd Hollon	Future Stock Options Stock Options	Invenio Imaging Inc. Invenio Imaging Inc.
Bermans Iskandar	Employee/Executive Own Stocks	Madison Scientific Inc. Madison Scientific Inc.
Kendall Lee	Owner	NaviNetics
Eric Leuthardt	Consulting Fee Own Stocks Receipt of IP/Patent Royalty Stock Options	Acera, Alcyone, E15, Intellectual Ventures, Microbot, Monteris Medical, Neurolutions, Osteovantage, Pear Therapeutics Inc., Sante Ventures Caeli Vascular, Inner Cosmos, Neurolutions, Petal Surgical, Sora Neuroscience Caeli Vascular, Neurolutions, Osteovantage Ceroventions, Intellectual Ventures Acera, Caeli Vascular, Face to Face Biometrics, General Sensing, Immunovalent, Inner Cosmos, Kinetrix, NeuroDev, Neurolutions, Osteovantage, Pear Therapeutics, Sora Neuroscience
Elad Levy	Consulting Fee Own Stocks Receipt of IP/Patent	Clarion, GLG Consulting, Guidepoint Global, Imperative Care, Medtronic, StimMed, Misionix, Mosiac NeXtGen Biologics, RAPID Medical, Claret Medical, Cognition Medical, Imperative Care, Rebound Therapeutics, StimMed, Three Rivers Medical Bone Scalpel
Russell Lonser	Consulting Fee	Biogen, uniQure
Jennifer Moliterno	Consulting Fee	BK Medical
Praveen Mummaneni	Consulting Fee Own Stocks Royalty	DePuy Synthes, Globus Medical, Stryker Spinicity/ISD DePuy Synthes, Springer Publishers, Thieme Publishers

Name	Type of Disclosure	Entity/Company
Daniel Orringer	Consulting Fee Fees for Non-CME Services Future Stock Options Stock Options	DXCover, NX Development Corporation, Stryker Designs for Visions Invenio Imaging Inc. Invenio Imaging Inc.
Aditya Pandey	Own Stocks	FlexDex Surgical, NextGen Biologics
John Pollina	Consulting Fee Own Stocks Royalty Speakers Bureau	ATEC Spine REMI ATEC Spine Medtronic
Michael Schulder	Consulting Fee Own Stocks	Hyperfine Inc. Hyperfine Inc.
Daniel Sciubba	Consulting Fee Future Stock Options Own Stocks	Baxter, Depuy-Synthes, Medtronic, Stryker Augmedics BioPhy
Sameer Sheth	Consulting Fee	Abbott, Boston Scientific, Neuropace, Zimmer Biomet
Gary Steinberg	Consulting Fee Royalty	SanBio, Surgical Theater, Zeiss Peter Lasic US
Nicholas Theodore	Consulting Fee Own Stocks Royalty	Bioventus, Globus Medical Globus Medical Globus Medical
Juan Uribe	Consulting Fee	Mainstay, Misonix, NuVasive, SI Bone
Ben Waldau	Consulting Fee	Siemens, Stryker

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

Manish Aghi
Wajd Al-Holou
Wael Asaad
Garrett Banks
Nicholas Boulis
Kim Burchiel
Clark C. Chen
Robert Dempsey
Richard Ellenbogen
Andrew Hale
Casey Halpern
Daniel Hoh
Anna Huguenard
Mark Johnson
Kristopher Kahle
Douglas Kondziolka
Michael Lawton
Bradley Lega

Allan Levi
David Limbrick
Michael Link
Christopher Ogilvy
Jung Park
Aneek Patel
Pier Paolo Peruzzi
Elias Rizk
Aaron Rusheen
James Rutka
Nader Sanai
Steven Schiff
Raj Shrivastava
Xiaonan Richard Sun
Juan Uribe
Kareem Zaghoul
Jeffrey Zuccato

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Gary Steinberg, MD, PhD	Stanford University	Stanford, CA
Xiaonan Richard Sun, MD, PhD	Northwell Health	New Hyde Park, NY
Nicholas Theodore, MD	Johns Hopkins University	Baltimore, MD
Shelly Timmons, MD, PhD	Indiana University	Indianapolis, IN
Juan Uribe, MD	Barrow Neurological Institute	Phoenix, AZ
Ben Waldau, MD	University of California, Davis	Sacramento, CA
Daniel Yoshor, MD	University of Pennsylvania	Philadelphia, PA
Kareem Zaghloul, MD, PhD	National Institutes of Health	Bethesda, MD
Gregory Zipfel, MD	Washington University	St. Louis, MO
Jeffrey Zuccato, MD	University of Toronto	Toronto, ON Canada

GUESTS, LOCATIONS & HOSTS

Guest	City	Host
Wajd Al-Holou, MD	Ann Arbor, MI	Karin Muraszko
Zarina Ali, MD	Haddonfield, NJ	James M. Markert
John Andrews, MD	San Francisco, CA	Edward Chang
Wael Asaad, MD, PhD	Westwood, MA	Guest of the Academy
Nicholas Bambakidis, MD	Cleveland Heights, OH	Warren Selman
Garrett Banks, MD	Houston, TX	Sameer Sheth
Marvin Bergsneider, MD	Los Angeles, CA	Guest of the Academy
Nicholas Borg, MD	Omaha, NE	Aviva Abosch
Samuel Browd, MD	Seattle, WA	Richard Ellenbogen
Terry C. Burns, MD, PhD	Rochester, MN	Guest of the Academy
Andrew Chan, MD	New York, NY	Praveen Mummaneni
Dean Chou, MD	New York, NY	E. Sander Connolly
Melanie Hayden Gephardt, MD	Palo Alto, CA	Guest of the Academy
Andrew T. Hale, MD, PhD	Mountain Brook, AL	Guest of the Academy
Casey Halpern, MD	Philadelphia, PA	Daniel Yoshor
Benjamin Hendricks, MD	Phoenix, AZ	Michael Lawton
Daniel Hoh, MD	Gainesville, FL	Brian Hoh
Marshall Holland, MD	Birmingham, AL	Gregory Zipfel
Todd Hollon, MD	Ann Arbor, MI	B. Gregory Thompson
Anna Huguenard, MD	St. Louis, MO	Eric Leuthardt
Kristopher Kahle, MD	Boston, MA	Bob Carter
Shekar Kurpad, MD, PhD	Wauwatosa, WI	Guest of the Academy
Bradley Lega, MD	Dallas, TX	H. Hunt Batjer
Derek Li, MD	St. Louis, MO	Gregory Zipfel
William Mack, MD	Los Angeles, CA	Guest of the Academy
Kai Miller, MD	Rochester, MN	Fred Meyer
Akshitkumar Mistry, MD	Villa Hills, KY	Gregory Zipfel

Guest	City	Host
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Daniel Orringer, MD	New York, NY	Douglas Kondziolka
John O'Toole, MD	Chicago, IL	Richard Byrne
Aditya Pandey, MD	Ann Arbor, MI	Guest of the Academy
Jung Park, MD, PhD	Manhasset, NY	John Boockvar
Pier Paolo Peruzzi, MD, PhD	Boston, MA	E. Antonio Chiocca
John Pollina, MD	Buffalo, NY	Elad Levy
Wilson Z. Ray, MD	St. Louis, MO	Guest of the Academy
Elias Rizk, MD	Hershey, PA	Robert Harbaugh
Marie Roguski, MD	Boston, MA	Carl Heilman
Aaron Rusheen, PhD	Rochester, MN	Kendall Lee
Daniel Sciubba, MD	Great Neck, NY	Michael Schulder
Raj Shrivastava, MD	New York, NY	Joshua Bederson
Xiaonan Richard Sun, MD, PhD	New Hyde Park, NY	Michael Schulder
Juan Uribe, MD	Phoenix, AZ	Guest of the Academy
Aditya Vedantam, MD	Menomonee Falls, WI	Gregory Zipfel
Ananth Vellimana, MD	St. Louis, MO	Gregory Zipfel
Ben Waldau, MD	Sacramento, CA	Griffith Harsh
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Risheng Xu, MD, PhD	Baltimore, MD	Alan Cohen
Keny Kwok Hei Yu, PhD	New York, NY	Gregory Zipfel
Kareem Zaghloul, MD, PhD	Bethesda, MD	Russell Lonser
Jeffrey Zuccato, MD	Toronto, ON Canada	Gelareh Zadeh
Chad Jacobs	Austin, TX	BrainLab, Inc.
Sean Clark	Westchester, IL	BrainLab, Inc.
James Carter	Burlington, MA	Carl Zeiss Meditec, Inc.
Chris Danko	Dublin, CA	Carl Zeiss Meditec, Inc.
Misty Browd	Dallas, TX	Elekta

Guest	City	Host
Michael Diab	Chesterfield, MO	Elekta
Jacquelyn Tallarico	Atlanta, GA	Elekta
Sigmund Kulesa	Yardley, PA	Integra LifeSciences
Jason Marzuola	Princeton, NJ	Integra LifeSciences
Angela Davis	Deerfield, IL	Leica Microsystems
Rachel Flynn	Phoenix, AZ	Leica Microsystems
Matthew Welz	Buffalo Grove, IL	Leica Microsystems
Jim Marucci	Kalamazoo, MI	Stryker
Kylie Owens	Kalamazoo, MI	Stryker
Mark Raabe	Kalamazoo, MI	Stryker
John Adler, MD	San Carlos, CA	Zap Surgical
Chris Pegano	San Carlos, CA	Zap Surgical
Richard Rosene	Newport, RI	Zap Surgical



THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

84TH ANNUAL SCIENTIFIC MEETING SCIENTIFIC PROGRAM AT-A-GLANCE

WEDNESDAY, SEPTEMBER 28, 2022

REGISTRATION AND RECEPTION

THURSDAY, SEPTEMBER 29, 2022

7:30 – 7:35 WELCOMING REMARKS

Alexandra Golby, MD

7:35 – 7:45 Round Robin Roundup! The Academy Round Robin Letters, 1939-2022

Mark Hadley, MD

7:45 – 9:00 Peer Reviewed Abstract Session I: Spine Clinical Science

Moderators: Zoher Ghogawala and Daniel Resnick

7:45 – 7:55 Decompression With or Without Fusion for Grade 1 Degenerative Lumbar Spondylolisthesis: 60-Month Outcomes From the QOD

Andrew Kai-Hong Chan, MD; Erica Fay Randy Bisson, MD; Mohamad Bydon, MD; Steven D. Glassman, MD; Kevin T. Foley, MD; Christopher I. Shaffrey, MD; Eric A. Potts, MD; Mark Edwin Shaffrey, MD; Domagoj Coric, MD; John J. Knightly, MD; Paul Park, MD; Michael Y. Wang, MD; Kai-Ming G. Fu, MD, PhD; Jonathan Slotkin, MD; Anthony L. Asher, MD; Michael S. Virk, MD, PhD; Panagiotis Kerezoudis, MD; Jian Guan, MD; Vivian Le; Dean Chou, MD; Regis W. Haid, MD; **Praveen V. Mummaneni, MD**

Introduction

When comparing decompression with and without fusion, long-term outcomes are unclear following surgery for degenerative lumbar spondylolisthesis.

Objectives

We compare the 60-month outcomes for decompression alone and decompression with fusion for Meyerding grade 1 degenerative lumbar spondylolisthesis using the Quality Outcomes Database (QOD).

Methods

We conducted a retrospective analysis of prospectively-collected data from the QOD Spondylolisthesis module. Patients were enrolled who received single-segment surgery for Meyerding grade 1 degenerative lumbar spondylolisthesis. Sixty-month outcomes – Oswestry Disability Index (ODI), reaching ODI minimum

clinically important difference (MCID) (defined as an ODI improvement of 12.8), Numeric Rating Scale (NRS) Back Pain (NRS-BP), NRS Leg Pain (NRS-LP), EQ-5D, and NASS Satisfaction – were compared for patients receiving decompression alone versus decompression with fusion. Multivariable analyses were conducted, adjusting for variables reaching $p < 0.20$ on univariate comparisons.

Results

Overall, 608 patients were enrolled: 140 decompression alone (23.0%) and 468 (77.0%) decompression with fusion. The 60-month follow-up rate was 73.2%. In multivariable analyses, fusion was associated with a higher odds of reaching ODI MCID (OR=1.9, 95%CI[1.2-3.1], $p=0.01$), lower NRS-LP (B=-0.7, 95%CI[-1.3- -0.1], $p=0.01$), and higher NASS satisfaction (OR=1.9, 95%CI[1.2-3.0], $p=0.01$). Fusion was associated with similar NRS-BP (B=-0.3, 95%CI[-0.8-0.3], $p=0.36$), ODI (B=-2.5, 95%CI[-6.2-1.2], $p=0.18$), and EQ-5D (B=0.02, 95%CI[-0.02-0.06], $p=0.27$) compared to decompression alone.

Conclusion

In a long-term, 60-month comparison of outcomes, the addition of fusion to decompression was associated with superior outcomes for leg pain and satisfaction and nearly twice the odds of achieving an MCID in disability. Both procedures performed similarly for back pain and quality of life.

7:55 – 8:05	Coronal Malalignment Impact Best vs Worst Outcomes After Less Invasive Spine Surgery for Adult Spinal Deformity (ASD)
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Juan Uribe, MD; Jay D. Turner, MD, PhD; Paul Park, MD; Vivian Le; Richard G. Fessler, MD, PhD; Pierce D. Nunley, MD; Robert Eastlack, MD; David O. Okonkwo, MD, PhD; Khoi Duc Than, MD; Kai-Ming G. Fu, MD, PhD; Michael Y. Wang, MD; Adam S. Kanter, MD; Neel Anand, MD; Gregory M Mundis, MD; Shay Bess, MD; Dean Chou, MD; Praveen V. Mummaneni, MD, MBA; International Spine Study Group

Introduction

Minimally invasive approaches to ASD correction are growing in popularity. A better understanding of the factors that influence good versus poor outcomes with circumferential minimally invasive spine surgery (cMIS) deformity surgery is needed.

Objectives

Understand the factors that influence good versus poor clinical outcomes in a group of patients from a prospectively collected, multi-center database that had cMIS for ASD.

Methods

Data from a prospectively collected, multi-center database was retrospectively reviewed. Two cohorts of patients were generated based on ODI improvement at 2 yrs: top 20% of patients with greatest improvement, and bottom 20% of patients with least improvement/deterioration. Patient characteristics, radiographic parameters, treatment data, clinical outcomes and complications were compared. Univariate comparisons were performed using t-tests and nonparametric tests. Categorical variables were compared using Fisher exact test. Significance was set at $p\text{-value} \leq 0.05$.

Results

85 ASD patients treated with cMIS techniques were analyzed and 68 patients with 2-year ODI follow-up were identified. 14 patients were in the top 20% and 14 patients in the bottom 20% cohorts. There were no significant differences in baseline demographics between groups. Top 20% had higher baseline ODI compared to bottom 20% (56.7 and 47.6, respectively, $p=0.032$), but similar baseline SF36 PCS/MCS, NRS back/leg, EQ5D, and SRS22 ($p^3 0.08$). Patients in the bottom 20% had worse baseline coronal alignment (3.3 vs 1.2 cm, $p=0.03$) but similar max Cobb angles ($p=0.46$). Patients in the top 20% had significantly better improvement in all patient reported outcomes measures (ODI, SF36 PCS/MCS, NRS back/leg, EQ5D, SRS22) compared to the bottom 20% ($p < 0.05$). There were no significant differences in treatment data. Radiographic parameter outcomes were similar though coronal malalignment at 2 years approached significance ($p=0.061$). Radiographic complications were higher in the bottom 20% (7 vs 1, $p=0.02$).

Conclusion

In this prospective, multicenter study, coronal malalignment is associated with poor clinical outcomes after cMIS surgery for ASD. When the sagittal plane is appropriately treated, greater emphasis may need to be placed on the coronal plane to achieve good outcomes with cMIS techniques.

8:05 – 8:15	Enhanced recovery after surgery (ERAS) for posterior cervical spine surgery: a propensity-matched cohort study
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Daniel J. Hoh, MD; Ken Michael Porche, MD; Basma Mohamed

Introduction

Enhanced recovery after surgery (ERAS) is a multimodal strategy to optimize early postoperative outcomes. To date, ERAS for spine has been limited to lumbar surgery and ACDF. ERAS has not been studied for posterior cervical surgery, which may present greater opportunity for improvement than ACDF.

Objectives

A single institution, multi-surgeon study comparing posterior cervical surgery outcomes with ERAS vs. non-ERAS controls.

Methods

A retrospective consecutive cohort study was performed for posterior cervical surgery patients after ERAS implementation compared to propensity-matched historical controls (demographics, BMI, surgical levels, preoperative opioid MME, smoking). Included subjects underwent laminectomy with/without fusion or laminoplasty for cervical degenerative disease at the Univ. of Florida. Outcomes included: length of stay; day of 1st ambulation, bowel movement, void; pain score; opioid MME; discharge disposition; 30-day readmission rate.

Results

Cohorts were ERAS=127 vs. control=127. Patient characteristics, procedure and operative time were similar. The ERAS cohort had significantly improved length of stay (3.2 vs. 4.7 days, $p<.0001$), and home discharge rate (80% vs 50%, $p<.001$), without increase in readmission rate. The ERAS cohort had earlier day of 1st ambulation ($p=.003$), bowel movement ($p=.014$), and void ($p=.001$). ERAS demonstrated significantly lower composite complication rate (1.1 vs. 1.8, $p<.0001$). ERAS resulted in better maximum daily pain score ($p=.043$), and trended towards improved mean pain score ($p=.072$), although total opioid MME was similar.

Conclusion

Implementing a novel ERAS protocol significantly improved length of stay, return of physiological function, home discharge rate, complications, and maximum daily pain score after posterior cervical surgery.

8:15 – 8:25	Progenitor Cell Injection Produces Meaningful Improvements for Lumbar Disc Degeneration Patients For At Least 2 Years
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Kevin T. Foley, MD

Introduction

Allogeneic disc progenitor cells have demonstrated immunomodulatory and regenerative properties in animal studies. We report the results of an FDA-approved, prospective, randomized, double-blind clinical trial of these cells for treating symptomatic lumbar degenerative disc disease (DDD).

Objectives

The aim of this clinical study was to evaluate allogeneic disc progenitor cells injected into symptomatic human degenerated lumbar intervertebral discs for safety and preliminary efficacy as measured by reduction of pain and improvement in function and quality of life. Here we report the results of a 104-week FDA

Investigational New Drug (IND)-allowed, prospective, randomized, double-blind, multicenter clinical trial of these cells for treating symptomatic early to moderate lumbar DDD.

Methods

Subjects with symptomatic lumbar DDD were randomized to one of 4 treatments and received single intradiscal injections of low-dose cells (3,000,000 cells/mL; N=20), high-dose cells (9,000,000 cells/mL; N=20), vehicle (N=10) or placebo (N=10). Subjects were blinded to treatment and were assessed by blinded clinicians over 104 weeks for safety and efficacy parameters.

Results

60 subjects (median age 38, 60% male) were enrolled across 13 clinical sites. Low back pain VAS scores in the high-dose cell therapy group improved by more than 30% at weeks 12 (54.53%, $p=0.0056$), 26 (50.94%, $p=0.0140$), 52 (62.79%, $p=0.0005$), 78 (59.44%, $p=0.0034$), and 104 (60.3%, $p=0.002$). For the saline placebo group, VAS score improved after treatment but only demonstrated statistically significantly greater than 30% reduction at week 26. Further analysis showed that only the high-dose cell group showed reduction in back pain VAS that was statistically significantly greater than a minimum clinically important difference (MCID) of 20 mm at weeks 12 (-36.1, $p=0.009$), 26 (-35.2, $p=0.015$), 52 (-42.8, $p=0.001$), 78 (-40.2, $p=0.005$), and 104 (-39.4, $p=0.004$). Only the high-dose cell group exhibited an improvement from baseline in ODI that was statistically significantly greater than a MCID of 10 points at weeks 12 (-25.3, $p=0.001$), 26 (-25.9, $p=0.001$), 52 (-25.7, $p=0.004$), 78 (-30.8, $p=0.0001$), and 104 (-29.6, $p=0.0005$). Only the high-dose cell group showed improvement in EQ-5D that was statistically significantly greater than a MCID of 0.08 at weeks 12 (0.194, $p=0.0035$), 26 (0.202, $p=0.0005$), 52 (0.197, $p=0.003$), 78 (0.241, $p<0.0001$), and 104 (0.217, $p=0.002$). Statistically significant changes from baseline in disc volume were only observed in the high dose disc progenitor cell group and occurred at weeks 52 (+249.01 mm³, $p=0.0284$) and 104 (+402.1 mm³, $p=0.028$). No subjects in either of the two cell therapy treatment groups experienced serious treatment-emergent adverse events (TEAEs).

Conclusion

High-dose allogeneic disc progenitor cells produced clinically meaningful, statistically significant, and sustained improvements in back pain VAS, ODI, and EQ-5D in patients with symptomatic lumbar disc degeneration at 12 weeks post-injection. Clinical improvement was sustained at 26 weeks, 1 year, 78 weeks, and 2 years. Disc volume improved in the high-dose cell therapy group at 1 and 2 years.

8:25 – 8:35 Advances in Ultrasound for Spinal Cord Injury: From Imaging to Treatment
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Nicholas Theodore, MD; Andrew Hersh; Amir Manbachi, PhD; Carly Weber-Levine

Introduction

The primary phase of spinal cord injury (SCI) involves mechanical damage to the spinal cord and is followed by a secondary phase that includes ischemia, oxidative stress, loss of autoregulation, and inflammation. The microvasculature can be imaged after decompressive laminectomy to assess the extent of ischemia and regeneration. However, traditional methods using contrast-enhanced imaging are invasive and only last for short periods. Non-contrast advanced Doppler ultrasound techniques offers the potential to quantify spinal cord blood flow, informing prognosis and treatment paradigms.

Objectives

To develop and optimize ultrasound technologies for the real-time measurement of spinal cord blood flow.

Methods

Male adult Sprague-Dawley rats (n=10, 250-300 g) underwent a T10-12 laminectomy to expose the spinal cord. A stereotactic frame was used to position an ultra-high frequency ultrasound transducer (i22LH8, Canon) over the cord in the sagittal plane. Video clips of the microvasculature were recorded on a Canon Aplio i800 machine using an advanced modality known as Superb Microvascular Imaging (SMI, 12MHz). Ten SMI clips were recorded, corresponding to a total of 30-40 seconds of imaging. SCI was delivered using

a calibrated compression impactor, with 5 rats receiving a light injury (100 kDyn) and 5 receiving a severe injury (250 kDyn). Ultrasound imaging was performed again after injury. An in-house MATLAB algorithm was developed to generate velocity maps of the injury level from the SMI videos and quantify the velocity as a function of time. Velocities were normalized with respect to the cross-sectional area of the vessels. Paired t-tests were performed to determine statistically significant changes after SCI. As validation, similar experiments were performed in a porcine model of SCI (n=5) using a 20-gram weight drop from a height of 15 cm above the T5 vertebral level.

Results

Plots of the velocities over time illustrate cardiac cycles in vessels with sub-millimeter diameters. SMI is capable of detecting differences in velocity in the rodent microvasculature across the pre- and post-injury state for rats with the mild injury (0.18 ± 0.13 cm/s, $p=0.03$) and rats with the heavy injury (0.29 ± 0.12 cm/s, $p=0.04$). A statistically significant difference was also seen using SMI in the pig SCI model (pre- vs post-injury: 0.12 ± 0.04 cm/s). Moreover, individual vessels could be segmented and separated for analysis of velocity and flow.

Conclusion

Non-contrast Doppler ultrasound modalities can quantify the velocity of blood flow in the microvasculature of the spinal cord. Using rodent and porcine models, decreases in blood flow were detected in all animals after SCI, illustrating the ischemic effects of SCI. Ultrasound imaging may eventually be used to quantify spinal cord microvasculature in real-time, informing prognosis and recovery after injury and helping to determine individualized treatments for SCI, including therapeutic focused ultrasound.

8:35 – 8:45	Prolonged Opioid Use following Lumbar Fusion Surgery: A Meta-Analysis of Prevalence and Risk Factors
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Cathleen Kuo; Mohamed AR Soliman, MD, PhD; Joseph Iskander; Kyungduk Rho, MD; Asham Khan, MD; Patrick Jowdy, MD; **John Pollina, MD**; Jeffrey Paul Mullin, MD

Introduction

Persistent opioid utilization after spine surgery is a rising complication among both preoperatively opioid-naïve and opioid tolerance patients.

Objectives

To our knowledge, this is the first meta-analysis determine the prevalence and characterized the risk factors that predisposed patients to prolonged opioid use (≥ 3 months) following lumbar fusion surgery.

Methods

Studies were identified through a search in PubMed and EMBASE from inception to February 1, 2022. We included observational studies examining the rate of and risk factors of prolonged opioid use following lumbar fusion. Pooled odds ratios (ORs) or standardized mean differences (SMDs) with their corresponding 95% confidence intervals (CI) were estimated using the inverse-variance methods.

Results

In this meta-analysis of 12 studies encompassing 80,935 patients, 40.2% of patients continued to fill opioid prescriptions more than 3 months after lumbar fusion. Significant sociodemographic predictors included Medicare/Medicaid insurance plan (OR=1.60, 95%CI 1.36-1.88), African American (OR=1.29, 95%CI 1.18-1.41), patient from Southern United States (OR=1.18, 95%CI 1.11-1.25), and female sex (OR=1.10, 95%CI 1.01-1.20), while patient from the Midwest (OR=0.80, 95%CI 0.75-0.85) was a protective factor. Comorbidities associated with increased risk of prolonged opioid use were preoperative opioid use (OR=5.76, 95%CI 3.52-9.41), drug abuse (OR=3.11, 95%CI 2.37-4.08), alcohol abuse (OR=2.37, 95%CI 2.14-2.64), psychiatric disorders (OR=2.29, 95%CI 1.94-2.70), smoking history (OR=1.81, 95%CI 1.23-2.66), arthritis (OR=1.35, 95%CI 1.29-1.40), and higher American Society of Anesthesiologists score (SMD=0.72, 95%CI 0.61-0.82).

Conclusion

The high prevalence of prolonged opioid use following lumbar fusion underscored the importance to screen patients for comorbidities and implement targeted strategies to minimize opioid misuse.

8:45 – 8:55	Sacroiliac Joint Fusion Using Percutaneously Placed Titanium Screw Implants: A Prospective Outcomes Investigation
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Peter C. Gerszten, MD; Prateek Agarwal; Nima Alan, MD; Guy Beresteanu; Daryl Fields; Erin Paschel, PA

Introduction

Sacroiliac joint (SIJ) pain is a common cause of disabling back pain. It is frequently misdiagnosed as radicular pain originating from the lumbar spine. Recent evidence in the literature supports the clinical benefits of SIJ fusion compared to non-surgical management for SIJ mediated pain.

Objectives

This study was undertaken to prospectively evaluate a consecutive series of patients who underwent a percutaneous SIJ fusion procedure who had failed non-surgical management.

Methods

A prospective cohort investigation was performed on 211 consecutive patients who underwent SI joint fusion using the TriCor Sacroiliac Joint Fusion System (Zimmer Biomet) over a 5 year period. Twenty-five patients had a prior history of a lumbosacral fusion (12%). All patients failed SIJ injections as well as non-surgical treatments. Patients reported outcomes for leg and back pain (VAS), disability (ODI), quality of life (EQ-5D), and frequency of opioid medication use were collected preoperatively and at 1, 3, 6, 12 and 24-month time points postoperatively.

Results

The cohort included 139 women (2:1 ratio), mean age 55 years (range 20-87), BMI 31 kg/m² (range 19-41), and 24 smokers. Laterality was: 108 left, 87 right, and 26 bilateral (1 case at the same time). Improvement in VAS for back and leg pain was observed at 1 month as well as 24 months postoperatively with differences of 5.0 (p=0.001) and 4.2 (p=0.04, respectively). Sustained improvement in EQ-5D was observed from 0.44 to 0.71 at 24 months postoperatively (p=0.01). ODI scores decreased from 50 to 40 at 12 and 24 months (11.4 points, p=0.03). Ninety percent of patients reported satisfaction with having undergone the procedure ("very" and "somewhat") at 24 months. A single patient developed an S1 radiculopathy which required revision of the rostral-most screw and subsequent resolution of symptoms. Opioid dose decreased by 83% for patients with a history of prior opioid usage at 24 months compared to preoperative use.

Conclusion

Percutaneous SIJ fusion resulted in a significant improvement in EQ-5D, ODI, VAS, and opioid usage. SIJ fusion is a safe and effective procedure for patients with SIJ dysfunction. SIJ fusion surgery should be considered an essential component of the global care of patients with "lower back pain" and surgical spinal conditions.

8:55 – 9:00	Wrap-up/ Transition
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9:00 – 9:55	Peer Reviewed Abstract Session II: Cerebrovascular Cutting Edge Moderators: Jacques Morcos and Sepideh Amin-Hanjani
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9:00 – 9:10	Histotripsy based ICH liquefaction and evacuation in a swine ICH model
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Aditya S. Pandey, MD; Jonathan Sukovich; Tyler Gerhardson; Tim Hall; Zhen Xu

Introduction

ICH removal requires penetration of normal brain and manipulation of clot or introduction of tPA into the clot. There is an unmet need for the development of an incisionless tool which allows for immediate and targeted liquefaction and evacuation of ICH.

Objective

We aim to develop ultrasound-based histotripsy technique to allow for immediate and targeted liquefaction and evacuation of ICH thus bridging this technological-clinical gap.

Methods

Utilizing a swine ICH model (1.75 ml in frontal lobe), we utilized histotripsy to target, liquefy, and drain the ICH via needle aspiration and then survived animals for 7-8 days. Swine with ICH were divided into three groups: 6 with histotripsy treatment followed by evacuation, 6 with histotripsy treatment and no evacuation, and 6 with no histotripsy treatment and no evacuation of ICH. Swine were clinically evaluated for 7-8 days post histotripsy treatment and then sacrificed for MRI and histological analysis.

Results

Histotripsy treatment through an excised human skull led to liquefaction of 40 ml of Ex-vivo ICH within 30 minutes. In swine experiments, histotripsy was successful in liquefying the center of the clot (0.9 +/- 0.5 ml) while purposefully leaving the periphery of clot. The liquefied clot was easily drained and there was minimal cerebral edema surrounding the post evacuated ICH areas. There was no evidence of rehemorrhage during the survival time period. There were no changes to the clinical status of the swine post treatment with histotripsy.

Conclusion

Histotripsy can be utilized to successfully and safely target, liquefy, and drain ICH in a swine ICH model.

9:10 – 9:20	First-in-Human Phase 1/2a Study of Intracerebral Transplantation of Neural Stem Cells (NR1) for Chronic Ischemic Stroke
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Gary K. Steinberg, MD, PhD; Anthony Bet; Jennifer Williams; Kathy McDonald; Robert J. Diaz; Cindy H. Samos; Kirk Trisler; Judi Weissinger; Maria Coburn; Neil E Schwartz, MD, PhD

Introduction

Currently, no treatment exists to restore function in chronic stroke patients. Several prior intracerebral stem cell trials showed safety, but are not being further developed.

Objectives

NR1 is a human embryonic derived neural stem cell that improved motor-sensory function in rodent stroke models, and was expanded to produce GMP cryopreserved Cell Lots (P18). The aim is to assess safety, tolerability and efficacy using intracerebral transplantation of NR1 cells in chronic stroke patients (NCT04631406).

Methods

Inclusion Criteria: 18-75 yo; 6-60 months post-ischemic subcortical MCA stroke; mRS 3-4. Subjects are transplanted with NR1 (2.5M, 5M, 10M or 20M). Primary Outcomes: Adverse events 0-6 mos; Change in Fugl-Meyer (FM) motor score (maximum FM 100) compared to baseline at 6 months (≥ 10 points improvement considered “clinically meaningful”). Exploratory outcomes: NIHSS, Gait Speed test, mRS, MRI DTI, FLAIR, Resting State fMRI and [18F] FDG PET.

Results

Four patients have been transplanted. Adverse events included headache and worsened speech, all resolving spontaneously. FM improved 13 points in Patients 1 and 2 at 6 months (both with faster gait), 9 points in Patient 3 at 3 months and 16 points in Patient 4 at 1 month. All 4 patients demonstrated a new transient

FLAIR signal in premotor cortex at d7, that resolved by 2 mos, which in prior studies was highly correlated with sustained neurologic recovery. Six additional patients are scheduled in the next 4 months.

Conclusion

Intraparenchymal transplantation with NR1 cells in chronic stroke patients appears safe and well tolerated. Early results suggest improved motor function at 1-6 months post-implant.

9:20 – 9:30	Does Adjunctive Middle Meningeal Artery Embolization Following Surgery for Chronic Subdural Hematomas Reduce Recurrence?
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Christopher S. Ogilvy, MD; Ajith J. Thomas, MD; Justin M Moore, MD, PhD; Rafael A. Vega, MD, PhD; Ron L. Alterman, MD; Martina Stippler, MD; Efstathios Papavassiliou, MD; MirHojjat Khorasanizadeh; Max Shutran, MD; Mira Salih

Introduction

Middle meningeal artery embolization (MMAE) is an emerging endovascular treatment for chronic subdural hematomas (cSDH). Some centers have been using adjunctive MMA embolization following surgery to reduce recurrence of cSDH. However, the efficacy of this approach is not yet established.

Objectives

To compare the outcomes of MMAE following open surgery versus open surgery alone.

Methods

Patients who underwent surgical evacuation alone or adjunctive MMAE for cSDH were identified at our institution. Two balanced groups were obtained through propensity score matching. Primary outcomes of recurrence risk and reintervention rate were compared between the matched groups.

Results

A total of 345 surgical and 52 adjunctive MMAE following surgical procedures were included. 42 pairs of cases were compared after propensity score matching for age, gender, comorbid conditions, mRS score on presentation, history of fall, SDH thickness, acute or subacute components, post-procedure anticoagulant and post-procedure antiplatelet use. Before matching, recurrence risk and reintervention rate in open surgery alone was significantly higher than open surgery plus MMAE (16.5% vs 5.8%, $p=0.04$; 14.2% vs 3.8%, $p=0.04$ respectively). No significant difference was seen in decrease in hematoma size and mRS score at last follow up. After propensity matching, recurrence risk trended lower (7.1% vs 21.4%, $p=0.06$), and overall reintervention rate was found to be significantly lower in MMAE following open surgery compared to open surgery alone (4.8% vs 19.1%, $p=0.04$).

Conclusion

After matching for potential confounders through propensity adjustment, it was found that MMAE following open surgery can lower recurrence risks and re-intervention rates for cSDH.

9:30 – 9:40	System of anatomical triangles defining dissection routes to brainstem cavernous malformations
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Michael T. Lawton, MD; Dimitri Benner; Benjamin Hendricks, MD; Joshua Catapano, MD

Introduction

A system of anatomical triangles defining dissection routes to brainstem cavernous malformations: definitions and application to a cohort of 183 patients.

Objectives

Anatomical triangles defined by intersecting neurovascular structures delineate surgical routes to pathological targets and guide neurosurgeons during dissection steps. Collections or systems of anatomical triangles have been integrated into skull base surgery to help surgeons navigate complex regions like the cavernous sinus.

We present a system of triangles specifically intended for resection of brainstem cavernous malformations (BSCM); this system of triangles is complementary to our BSCM taxonomy that defines dissection routes to these lesions.

Methods

The anatomical triangle through which a BSCM was resected microscopically was determined for patients treated during a 23-year period who had both brain MRI and intraoperative photographs or videos available for review.

Results

Of 183 patients who met the inclusion criteria, 50 had midbrain lesions (27%), 102 had pontine lesions (56%), and 31 had medullary lesions (17%). The craniotomies used to resect these BSCMs included the extended retrosigmoid (66 [36.1%]), midline suboccipital (46 [25.1%]), far lateral (30 [16.4%]), pterional/orbitozygomatic (17 [9.3%]), torcular (8 [4.4%]), and lateral suboccipital (8 [4.4%]) approaches. The anatomical triangles through which BSCMs were most frequently resected were the interlobular (37 [20.2%]), vallecular (32 [17.5%]), vagoaccessory (30 [16.4%]), supracerebellar-infratrochlear (16 [8.7%]), subtonsillar (14 [7.7%]), oculomotor-tentorial (11 [6.0%]), infragalenic (8 [4.4%]), and supracerebellar-supratrochlear (8 [4.4%]) triangles. New, but infrequently used, triangles included the vertebrobasilar junctional (1 [0.5%]), supratrigeminal (3 [1.6%]), and infratrigeminal (5 [2.7%]) triangles. Overall, 15 BSCM subtypes were exposed through 6 craniotomies, and the approach was redirected to the BSCM by one of the 14 triangles paired with the BSCM subtype.

Conclusion

A system of BSCM triangles, including 9 newly defined triangles, is introduced to guide dissection to these lesions. The use of an anatomical triangle better defines the pathway taken through the craniotomy to the lesion and refines the conceptualization of surgical approaches. The triangle concept and the BSCM triangle system increase the precision of dissection through subarachnoid corridors, enhance microsurgical execution, and potentially improve patient outcomes.

9:40 – 9:50	Triple Therapy versus Dual Antiplatelet Therapy for Dolichoectatic Basilar Fusiform Dolichoectasia
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Adnan Hussain Siddiqui, MD, PhD; Andre Monteiro; Ricardo A. Hanel, MD, PhD; Peter Kan, MD; Alina Mohanty; Gustavo Cortez; Margarita Rabinovich; Charles Christian Matouk, MD; Nanthiya Sujijantararat, MD; Charles Edward Romero; Jeremy Guy Stone, MD; Koji C. Ebersole, MD; Lane Fry; Sabareesh Kumar Natarajan, MD; Brittany Owusu-Adjei Thomson; Santiago Ortega-Gutierrez, MD; Juan Vivanco-Suarez; Ajay K. Wakhloo, MD, PhD; **Elad I. Levy, MD**

Introduction

Dolichoectatic vertebrobasilar fusiform aneurysms (DVBFAs) have poor natural history when left untreated and high morbimortality when treated with microsurgery. Flow diversion with dual-antiplatelet therapy (DAPT) is feasible but carries high risk of perforator occlusion and progression of brainstem compression. Elaborate antithrombotic strategies are needed to preserve perforator patency while vessel remodeling occurs. We compared triple therapy (TT [DAPT plus oral anticoagulation]) and DAPT alone in patients with DVBFAs treated with flow diversion (FD).

Objectives

To compare the efficacy and safety of triple therapy and DAPT in patients with DVBFAs treated with flow-diverters.

Methods

Retrospective review of the endovascular databases of 8 US neurosurgical centers. Only dolichoectatic aneurysms involving at least one segment of the basilar artery were included in this study. Baseline demographics (age, sex), clinical presentation (symptoms and degree of disability), aneurysm characteristics

(diameter, length, location, presence of thrombus), procedural details (access site, number of flow diverters used, adjunctive coiling used), complications (intraprocedural, in-hospital, and after discharge), and clinical (degree of disability) and angiographic (occlusion grade) follow-up were recorded. The modified Rankin Scale (mRS) was used to measure the degree of disability preprocedure, at discharge, and at last-follow-up. Patients with an mRS score ≥ 3 were considered to have moderate-to-severe disability. Overall decline in mRS score was based on preprocedure to last follow-up available, secondary to any complications, and development of symptoms or progression of the initial ones. Angiographic occlusion grade was assessed on digital subtraction angiography (DSA), computed tomography (CT) angiography, or magnetic resonance (MR) angiography and categorized as complete occlusion (no filling) and residual filling (any degree). Acute ischemic stroke was defined as neurological deficits resulting in an increase of ≥ 4 points on the National Institutes of Health Stroke Scale (NIHSS), with evidence of ischemia on noncontrast CT or MR diffusion-weighted imaging. Any type of bleeding events that were reported were considered hemorrhagic complications.

Results

The groups (DAPT=13, TT=14) were similar in age, sex, clinical presentation, baseline disability, and aneurysm characteristics. Radial access use was significantly higher in the TT group (71.4% vs. 15.3%, $P=0.006$). Median number of flow diverters and adjunctive coiling use were not significantly different between groups. The acute ischemic stroke rate during the oral anticoagulation period was lower in the TT group than the DAPT group (7.1% vs. 30.8%, $P=0.167$). Overall rate of modified Rankin Scale score decline was significantly lower in the TT group (7.1% vs. 69.2%, $P=0.001$). Overall rate of hemorrhagic complications was numerically higher in the TT group (28.6% vs. 7.7%, $P=0.162$). The rate of moderate-to-severe disability at last follow-up was significantly lower in the TT group (21.4% vs. 76.9%, $P=0.007$).

Conclusion

Patients with DVBFAs treated with FD in the TT group had less ischemic strokes, less symptom progression, and overall better outcomes at last follow-up than similar patients in the DAPT group.

9:50 – 9:55	Wrap-up/ Transition
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9:55 – 10:10	Break
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10:10 – 11:05	Peer Reviewed Abstract Session III: Clinical Science of Brain Tumors Moderators: Shenandoah Robinson and Randy Jensen
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10:10 – 10:20	The genomic profiles and clinical manifestations of meningiomas vary amongst different races
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Shaurey Vetsa; Sagar Vasandani; Muhammad Ibrahim Jalal; Neelan Joseph Marianayagam, MD, PhD; Kanat Yalcin; Mark W Youngblood, MD, PhD; Aladine A. Elsamadicy, MD; Ketu Mishra Gorur, PhD; Declan McGuone; Robert Fulbright; Lan Jin; Zeynep Erson-Omay; Murat Gunel, MD; **Jennifer A. Moliterno, MD**

Introduction

While socioeconomic factors for racial disparities amongst sporadic meningioma patients have been explored, other potential influences are poorly understood.

Objectives

We sought to identify whether the genomic make-up is different amongst meningioma patients of different races and how they correlate with clinical variables.

Methods

Patients who underwent surgery for sporadic meningioma and consented for whole exome sequencing were eligible. Genomic and clinical data were reviewed and analyzed.

Results

537 intracranial meningiomas from 483 patients were included. Whites were older at the time of diagnosis ($p=0.038$) and surgery ($p=0.015$). Black and Latinx patients more commonly presented with vision abnormalities ($p=0.006$). Whites were more likely to have convexity meningiomas ($p=0.003$), while Blacks harbored more anterior fossa meningiomas ($p=0.002$) with associated somatic Hedgehog (HH) mutations ($p=0.008$). Both Black and Latinx patients were more likely to have TRAF7 mutated meningiomas ($p=0.006$). Blacks were more likely to harbor genomically unstable, high-grade meningiomas ($p=0.011$), followed by Whites, Asians, and then Latinx ($p=0.020$). Black patients trended toward decreased progression-free survival than others (median survival: 57 vs. 130 months; $p=0.06$) despite similar extent of resection.

Conclusion

Overall, Black patients are more likely to have anterior skull base meningiomas with somatic HH and TRAF7 mutations. With regards to tumor grade, Blacks harbor a higher prevalence of high-grade meningiomas with underlying chromosomal instability. These findings have implications for meningioma care especially in minority populations, and form the basis of further genomic, epigenomic and environmental studies focusing on the inherited versus somatic basis of these observed differences.

10:20 – 10:30 Developing and validating new prognostic epigenetic subtypes of chordoma that are detectable with liquid biopsy

Jeffrey Alexander Zuccato, MD; Vikas Patil; Sheila Mansouri; Jeffrey Liu; Farshad Nassiri, MD; Yasin Mamatjan; Ankur Chakravarthy; Shirin Karimi; Joao Paulo Cavalcante de Almeida; Anne-Laure Bernat; Mohammed Ahmed Hasen, MD; Olivia Singh; Shahbaz Khan; Thomas Kislinger; Namita Sinha; Sebastien Froelich, MD; Homa Adle-Biasette; Kenneth Aldape, MD; Daniel de Carvalho; Gelareh Zadeh, MD, PhD

Introduction

Chordomas are skull-base and spine tumors that comprise 2-4% of aggressive primary bone cancers. Clinically, aggressive and benign patient subsets are observed but cannot be reliably distinguished using existing clinical factors, limiting stratification of treatment decisions.

Objectives

To identify prognostic DNA methylation-based subgroups of chordomas in tissue that are detectable in patient plasma.

Methods

Chordoma samples from a multi-institutional 20-year surgical series of 68 patients underwent tissue DNA methylation profiling. Matched plasma methylomes were obtained where available.

Results

Two chordoma subgroups were identified by consensus clustering with different disease-specific survivals (median 6.0 vs. 17.3 years, log rank $p=0.0062$) that were independent of clinical factors (multivariable Cox: HR=14.2, 95%CI: 2.1-94.8, $p=0.0063$). The poorer performing “Immune-infiltrated” subtype had immune-related gene-sets with hypomethylated promoters and increased tumor immune cell abundance. The better performing “Cellular” subtype had cell-to-cell/extracellular matrix interaction pathway hypomethylation and higher tumor cellularity. These subtypes were validated in an external DNA methylation dataset, pathways were validated in external gene expression data, and immune infiltration was validated with immunohistochemistry. Plasma methylome differentially methylated regions (DMRs) in fifty random 80% training sets accurately differentiated chordomas from representative clinical differential diagnoses in independent 20% testing sets (mean AUROC=0.84, 95%CI: 0.52-1.00). Leave-one-out models trained on DMRs between subtypes accurately identified the subtype of all left-out samples.

Conclusion

Here we present the first robust prognostic molecular subtypes of chordoma that are detectable in plasma to guide preoperative decision making, allowing the extent of resection and adjuvant therapy to be matched with patient prognosis.

10:30 – 10:40 Intraoperative microdialysis for glioma metabolic reconnaissance and biomarker discovery

Cecile Riviere-Cazaux; Lucas Carlstrom, MD, PhD; Desmond Brown, MD, PhD; **Terry C Burns, MD, PhD**

Introduction

Gliomas are genomically heterogeneous tumors that may harness convergent and therapeutically targetable metabolic pathways. At present, the metabolic landscape of in situ human gliomas remains poorly characterized, hampering translational progress.

Objectives

We sought to leverage the previously untapped potential of high molecular weight microdialysis during standard-of-care glioma surgeries to elucidate the global extracellular metabolic profiles of live human gliomas.

Methods

Under an investigational device exemption, High molecular weight (HMW) microdialysis (< 100 kDa) was performed at 2.0 μ L/min in an initial discovery cohort of five patients in glioma and adjacent brain during neurosurgical resection; a subsequent cohort of five patients was independently analyzed to critically evaluate results from the discovery group. Untargeted metabolomics via ultra-performance liquid chromatography-tandem mass spectrometry revealed over 300 named metabolites and five drugs from only 20 μ L of microdialysate, representing a short and feasible 10 minutes of intraoperative collection time.

Results

Enrichment analysis of each patient's tumor vs. brain ranked extracellular metabolome highlighted marked metabolic convergence within the most aggressive regions of molecular diverse tumors (FDR = 0.000). Pathway analysis revealed significant enrichment for large neutral amino acid pathways, including valine, leucine, and isoleucine biosynthesis ($p=1.6E-9$) and degradation ($p=0.001$) as well as glycine, serine, and threonine metabolism ($p=4.7E-5$). Notably, this amino acid signature was not as abundantly present in non-enhancing tumor when compared to enhancing tumor (Average tumor/brain: 1.9x vs. 4.3x, respectively), suggesting preferential upregulation of neutral amino acids within regions of more aggressive disease. Interestingly, guanidinoacetate (GAA) was the most highly conserved and upregulated metabolite (128.9x in tumor vs. brain). Given its co-production with ornithine, the precursor to protumorigenic polyamines, we posit that GAA may serve as a biomarker of increased ornithine decarboxylase (ODC) activity in live human gliomas. Indeed, we found that dual blockade of polyamine synthesis with a novel agent AMXT 1501 in combination with the ODC antagonist DFMO, improved survival in GBM xenografts. We have now secured an IND for a pharmacodynamically guided phase 0 clinical trial of AMXT 1501+DFMO that will utilize GAA and polyamine measurements via HMW microdialysis.

Conclusion

Intraoperative HMW microdialysis offers potentially important opportunities for mechanistic glioma discovery. Leveraging this typically untapped access to human glioma biology in situ may help guide development of rational early phase studies guided more directly by human disease biology than mouse experimentation.

10:40 – 10:50 Integrated single-cell epigenomic analysis identified a core vulnerability despite glioblastoma heterogeneity

Clark C. Chen, MD, PhD

Introduction

In 2021, the World Health Organization (WHO) reclassified glioblastoma, the most common form of adult brain cancer, into isocitrate dehydrogenase (IDH) wild-type glioblastomas and grade IV IDH mutant (G4 IDHm) astrocytomas. For both tumor types, intra-tumoral heterogeneity forms the basis for cancer evolution and fundamentally dictates therapeutic response.

Objectives

To provide single-cell epigenomic landscape of IDHwt glioblastomas and G4 IDHm astrocytomas as well as identified shared vulnerabilities.

Methods

We performed integrated genome-wide chromatin accessibility (snATACseq) and transcription (snRNAseq) profiling of clinical specimens derived from isocitrate dehydrogenase wild type (IDHwt) glioblastomas and grade 4 IDH mutant (IDHm) astrocytomas. A shared vulnerability was identified and validated using patient derived xenograft (PDX) models.

Results

The integrated analysis achieved resolution of intra-tumoral heterogeneity not previously possible, providing a molecular landscape of extensive regional and cellular variability. snATACseq delineated focal amplification down to an ~40 KB resolution. The snRNA analysis elucidated distinct cell types and cell states (neural progenitor/oligodendrocyte cell-like or astrocyte/mesenchymal cell-like) that were superimposable onto the snATACseq landscape. Paired-seq (parallel snATACseq and snRNAseq using the same clinical sample) provided high resolution delineation of extrachromosomal circular DNA (ecDNA), harboring oncogenes including CCND1 and EGFR. Importantly, the copy number of ecDNA genes correlated closely with the level of RNA expression. Integrated analysis across all specimens profiled suggests that IDHm grade 4 astrocytoma and IDHwt glioblastoma cells shared a common chromatin structure defined by open regions enriched for Nuclear Factor 1 transcription factors (NF1A and NF1B). Silencing of NF1A or NF1B suppressed in vitro and in vivo growth of patient-derived IDHwt glioblastomas and G4 IDHm astrocytoma models that mimic distinct glioblastoma cell states.

Conclusion

Our findings suggest despite distinct genotypes and cell states, glioblastoma/G4 astrocytoma cells share dependency on core transcriptional programs, yielding an attractive platform for addressing therapeutic challenges associated with intra-tumoral heterogeneity.

10:50 – 11:00 Natural history of von Hippel-Lindau disease.

Alexander Ksendzovsky, MD; **Russell R. Lonser, MD**

Introduction

von Hippel-Lindau disease (VHL) is associated with visceral and central nervous system tumors. Despite the morbidity and mortality linked to VHL-related neoplasms, long-term prospective characterization of disease natural history and functional outcome/mortality have not been defined.

Objectives

To define the causes of morbidity and mortality in VHL.

Methods

Prospective serial longitudinal (10 years or more) assessment (clinical, laboratory and imaging) of VHL patients was performed. Clinical, imaging, laboratory and genetic findings were analyzed.

Results

Two-hundred seven patients (104 male, 103 females) (mean follow-up, 12.4±1.3 years) were included. Thirty-seven patients died (mean age death, 51.0±12.8 years). Karnofsky performance score (KPS) remained stable in 154 (91%) and worsened in 16 (9%) patients. KPS decline was due to neurological (13 patients; 81%),

renal (2; 13%) or endocrinologic manifestations (1; 6%). Functional independence measure (FIM) remained stable in (162; 95%), improved in (1; 1%) and worsened in (7; 4%) patients (all neurological disease related). Greater nervous system hemangioblastomas, neurosurgical procedures and retinal hemangioblastoma-related visual symptoms ($P<0.01$) were associated with KPS decline. Greater nervous system hemangioblastomas and neurosurgical procedures ($P<0.01$) were associated with FIM decline. Adjusted life expectancy was 7 years shorter in VHL patients than expected. Most common causes of death were neurologic (14 patients; 38% of patients that died) or renal (14; 38%) complications. Greater nervous system hemangioblastomas were associated with mortality ($P<0.05$). Protein truncating germline mutations were associated with neurological death ($P=0.01$).

Conclusion

While nervous system hemangioblastomas underlie functional decline in VHL, disease-associated shortened life expectancy is associated with neurologic and renal disease. Partial germline mutations are associated with neurologic dysfunction and mortality.

11:00 – 11:05 Wrap-up/ Transition

11:05 – 11:40 Peer Reviewed Abstract Session IV: AI Tools and Applications in Neurosurgery
Moderators: Frederick Barker and Constantinos Hadjipanayis

11:05 – 11:15 Assessing the Utility of Low Resolution Brain Imaging: Tolerating Noise vs Risks of Deep Learning

Steven J. Schiff, MD, PhD; Joshua Harper; Venkateswararao Cherukuri ; Tom O'Reilly; Mingzhao Yu; Edith Mbabazi Kabachelor; Ronald Mulondo; Kevin Sheth; Andrew Webb; Benjamin C. Warf, MD; Abhaya Vivek Kulkarni, MD; Vishal Monga, PhD

Introduction

As low-field MRI technology is being disseminated into clinical settings around the world, it is important to assess the image quality required to properly diagnose and treat a given disease and evaluate the role of machine learning algorithms, such as deep learning, in the enhancement of lower quality images.

Objectives

In this post-hoc analysis of an ongoing randomized clinical trial, we assessed the diagnostic utility of reduced-quality and deep learning enhanced images for hydrocephalus treatment planning.

Methods

CT images of post-infectious infant hydrocephalus were degraded in terms of spatial resolution, noise, and contrast between brain and CSF and enhanced using deep learning algorithms. Both degraded and enhanced images were presented to three experienced pediatric neurosurgeons accustomed to working in low- to middle-income countries (LMIC) for assessment of clinical utility in treatment planning for hydrocephalus. In addition, enhanced images were presented alongside their ground-truth CT counterparts in order to assess whether reconstruction errors caused by the deep learning enhancement routine were acceptable to the evaluators.

Results

Results indicate that image resolution and contrast-to-noise ratio between brain and CSF predict the likelihood of an image being characterized as useful for hydrocephalus treatment planning. Deep learning enhancement substantially increases contrast-to-noise ratio improving the apparent likelihood of the image

being useful; however, deep learning enhancement introduces structural errors which create a substantial risk of misleading clinical interpretation. We find that images with lower quality than is customarily acceptable can be useful for hydrocephalus treatment planning. Moreover, low quality images may be preferable to images enhanced with deep learning, since they do not introduce the risk of misleading information which could misguide treatment decisions.

Conclusion

These findings advocate for new standards in assessing acceptable image quality for clinical use.

11:15 – 11:25 Dissociation of Broca’s Area from Broca’s Aphasia in patients undergoing neurosurgical resections

John Andrews, MD; Nathan Cahn; Benjamin Speidel; Jason Chung, MD; Deborah Levy; Stephen Wilson; Mitchel S. Berger, MD; **Edward Chang, MD**

Introduction

Broca’s aphasia is a syndrome of impaired fluency with retained comprehension. We used an unbiased algorithm to examine which neuroanatomic areas are most likely to result in Broca’s aphasia following surgical lesions.

Objectives

Determine if Broca’s aphasia is associated with lesions to Broca’s area.

Methods

Patients were prospectively evaluated with standardized language batteries before and after surgery. Broca’s area was defined anatomically as the pars opercularis and triangularis of the inferior frontal gyrus. Broca’s aphasia was defined by the Western Aphasia Battery language assessment. Resections were outlined from MRIs to construct 3D-volumes of interest. These were aligned using a non-linear transformation to MNI brain space. A voxel-based lesion-symptom mapping (VLSM) algorithm was used to test for areas statistically associated with Broca’s aphasia when incorporated into a resection, as well as areas associated with deficits in fluency independent of Western Aphasia Battery classification. Post-operative MRIs were reviewed blindly to estimate percentage resection of Broca’s area compared to areas identified through the VLSM algorithm.

Results

289 patients had early language evaluations, of whom 19 had postoperative Broca’s aphasia. VLSM analysis revealed an area highly correlated ($P < 0.001$) with Broca’s Aphasia, spanning ventral sensory-motor cortex and supramarginal gyri, as well as extending into subcortical white matter tracts. Reduced fluency scores were significantly associated with an overlapping region of interest. Fluency score was negatively correlated with fraction of resected pre-central, post-central, and supramarginal components of the VLSM area.

Conclusion

Broca’s Aphasia does not typically arise from neurosurgical resections in Broca’s Area. When Broca’s aphasia does after surgery, it is typically in the early postoperative period, improves by one month and is associated with resections of ventral sensorimotor cortex and supramarginal gyri.

11:25 – 11:35 Historical report

Michael Schulder, MD

11:35 – 11:40 Wrap-up/ Transition

11:40 – 11:55 Break

11:55 – 12:45 Guest Keynote Speaker

11:55 – 12:00 Introduction of the Guest Speaker by Dr. Aviva Abosch

12:00 – 12:45 Guest Speaker: Tyler R. Lyson, PhD
Curator of Vertebrate Paleontology Department of Earth Sciences

1:30 – 4:30 Academy Spine Emerging Investigators' Program

Program Director: Dr. Gregory Zipfel

7:30 – 7:35 WELCOMING REMARKS

7:35 – 8:40 Peer Reviewed Abstract Session V: Basic Science
Moderators: E. Sander Connolly and E. Antonio Chiocca

7:35 – 7:45 Report on NIH Funding
Gregory Zipfel, MD

7:45 – 7:55 Functional specialization along the hippocampal longitudinal axis
Bradley Charles Lega, MD

Introduction

Multi-modality evidence in both humans and rodent models suggests that the anterior and posterior hippocampus participate in distinct cognitive networks and fulfill complementary roles in cognition, especially episodic memory. However, this emerging area of research remains unknown to many practicing neurosurgeons. I will present novel human electrophysiology and gene expression data explicating these differences.

Objectives

Attendees will learn evidence supporting models of functional differentiation along the longitudinal axis of the human hippocampus.

Methods

I will present electrophysiological data from human epilepsy patients demonstrating key differences in anterior vs posterior hippocampal activity during episodic memory processing. I will then link these findings with gene expression profiles from 5 human hippocampal specimens resected in an en bloc fashion at the time of temporal lobectomy that had no MTS or other pathology. This analysis included identification of differentially expressed genes and how these genes are linked with cognitive performance and cognitive disorders, especially MDD and ASD.

Results

The posterior hippocampus exhibits elevated oscillatory power in the 2~5 Hz slow theta frequency band, contributing to mounting evidence that oscillations in this frequency band fulfill a role in human memory analogous to theta oscillations in rodent models. We identified several sets of genes that exhibit differential expression along the hippocampal longitudinal axis, including those expressed in pyramidal neurons, inhibitory interneurons, and astrocytes. Interneuron differences specifically may underly theta generation. These genes represent obvious targets for further investigation and therapeutic development.

Conclusion

The anterior vs posterior hippocampus represent functionally distinct structures. Our human data support cognitive models and previous findings from rodent studies.

7:55 – 8:05 Neural mechanisms of human episodic memory formation
Kareem A. Zaghloul, MD, PhD

Introduction

Memory is critical to our everyday experience. We rely upon our memories not only to form our own sense of identity, but also to guide and plan our future actions and behaviors. Understanding the neural mechanisms that underlie human memory formation is therefore critical in order to effectively treat memory disorders which are present in some of the most debilitating yet poorly managed neurological diseases.

Objective

Our research efforts are focused on investigating the neural correlates of human episodic memory formation by leveraging the opportunities to directly record neural activity across multiple spatial scales from the human brain in patients receiving surgical treatment for drug resistant epilepsy.

Methods

We investigate intracranial EEG (iEEG) signals captured using standard subdural and sEEG electrodes implanted for clinical seizure mapping as well as local field potential and single unit spiking activity captured through microelectrode arrays implanted in the anterior temporal lobe cortex.

Results

At larger spatial scales, we find that both specific patterns of localized neural activity and dynamic connections between brain regions emerge as people encode individual items into memory, and similar patterns of activity and connectivity are reinstated when people retrieve those same items from memory. At the smallest spatial scale, we find that populations of individual neurons in the anterior temporal lobe exhibit temporally organized sequences of spiking activity that are specific to the individual items people are encoding into memory, and that similar sequences are replayed when people retrieve those items from memory. The sequences of spiking activity are ordered based on the semantic category of the individual items, suggesting that sequences of spiking activity may be a fundamental unit of information in the human brain. In addition, these sequences of spiking activity are distributed across spatially contiguous yet distinct functional modules that are approximately the same size as the cortical columns hypothesized to exist throughout the human brain, suggesting a functional organization to how information is encoded across neuronal populations.

Conclusion

Together, our results provide novel insights into how information specific to individual memories is represented in the brain, and how this information is accessed as people recall previous experiences from memory.

8:05 – 8:15	Projection-defined cortical pyramidal neurons drive functionally-distinct cortical dynamics during decision-making
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Xiaonan Richard Sun, MD, PhD; Simon Musall; Hemanth Mohan; Xu An; Shujing Li; Rhonda Drewes; Anne Churchland

Introduction

Cortical pyramidal neurons (PyNs) are critical in the transformation of cognitive processing into meaningful behavior. Exquisitely diverse, PyN identity may be classified by their projections to various cortical or subcortical regions. While functional differences between PyN subtypes have been reported in specific cortical regions, how these properties extend across the cortex is poorly understood. To investigate this question experimentally, we leveraged cell type-specific mesoscale calcium imaging and optogenetics in a mouse model of human decision behavior.

Objectives

To delineate the functional roles of three major PyN projection classes during cognitive behavior.

Methods

We used genetic and viral approaches to perform circuit-specific interrogation and manipulation by targeting pyramidal tract (PT), intratelencephalic (IT) and corticostriatal projection neurons. Cortex-wide neural activity was recorded using wide-field and two-photon calcium imaging. Network causality was tested through

optogenetic inhibition. Our quantitative analyses include atlas-based factorization techniques and encoding and decoding models.

Results

Each PyN subtype was defined by unique neural dynamics, both locally and cortex-wide. Cortical activity and optogenetic inactivation during an auditory two-alternative forced choice decision task also revealed distinct functional roles: parietal PyNs were consistently recruited during the auditory stimulus, while, surprisingly, PT neurons exhibited the largest causal role. In the frontal cortex, all PyN subtypes were required for accurate choice selection with subtype-specific choice-tuning.

Conclusion

Our results reveal perceptual decisions shaped by parallel computations on multiple scales with projection specificity, highlighting the functional heterogeneity accompanying molecular and anatomic diversity. Our work seeks to inspire new perspectives in precision circuit modulation for cognitive and neuropsychiatric disorders.

8:15 – 8:25	Mutations In AK9 Decrease Cilia Motility And Cause Idiopathic Normal Pressure Hydrocephalus
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Mark D. Johnson, MD, PhD; Hongwei Yang, MD, PhD

Introduction

Idiopathic normal pressure hydrocephalus (iNPH) usually develops after age 60 and is characterized by gait difficulty, dementia and incontinence. Until recently, the etiology of iNPH was unknown. We recently reported that heterozygous CWH43 deletions can cause iNPH.

Objectives

To identify additional genetic alterations that may contribute to the development of shunt-responsive iNPH.

Methods

We performed whole exome sequencing of DNA obtained from 53 unrelated iNPH patients in 3 independent cohorts. Mutation frequency in these cohorts was compared to that of the general population.

Results

We identified heterozygous damaging mutations affecting AK9 that are statistically enriched among iNPH patients. AK9 mutations were observed in 5 of the 53 iNPH patients (9.6%, $P < 0.0001$, X2 Test with Yates correction). AK9 encodes adenylate kinase 9, which is a nucleoside mono- and diphosphate kinase involved in nucleoside homeostasis. Ak9 was highly expressed in sperm and in ventricular multiciliated neuroepithelial cells. We generated mice carrying an iNPH-associated AK9 mutation that causes a frameshift and premature termination of the encoded protein. AK9^{-/-} mice displayed normal sperm structure and number, but males were infertile due to decreased sperm flagellar motility. Homozygous AK9^{-/-} mice also displayed decreased cilia beat frequency, early onset communicating hydrocephalus and balance impairment. Heterozygous AK9^{+/-} mice were fertile and displayed normal brain development and behavior until early adulthood, but subsequently developed communicating hydrocephalus as they aged.

Conclusion

Our finding of iNPH-associated mutations in AK9 and CWH43 suggest that iNPH can be caused by heterozygous damaging mutations in multiple genes that impair ventricular multiciliated neuroepithelial cell function.

8:25 – 8:35	Rare Coding Mutations Identify Pathological Reprogramming of Endothelial Cells in Intracranial Aneurysms
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Tanyeri Barak, MD; Adife Gulhan Ercan Sencicek, PhD; Emma Ristori; Danielle F Miyagishima, BA; Kanat Yalcin; Katsuhito Yasuno; Ketu Mishra Gorur, PhD; Stefania Nicoli; Murat Gunel, MD

Introduction

The genetic architecture of intracranial aneurysms (IAs) is complex with contributions from common as well as rare genomic alleles that act in combination with environmental risk factors.

Objectives

While common risk IA loci have been identified through genome-wide association studies, the discovery of genes that harbor rare coding mutations that increase the risk of IA several fold has proven to be challenging.

Methods

To discover these IA genes with rare mutations, we conducted whole exome sequencing analysis of >200 patients from 58 multigenerational families. We performed a gene-based case-control study between IA cases and population matched controls in gnomAD. We used knockout zebrafish and mouse models to validate the functional role of candidate genes.

Results

Overall, we identified accumulation of mutations in genes that play a role in normal cerebrovascular morphology and integrity, mainly through Wnt signaling. Mutations in two genes, PPIL4 and WBP11, explained the genetic basis of >10% of cases in our cohort. Using zebrafish and mouse models, we demonstrated cerebral hemorrhage and changes in brain vasculature due to ppil4 and wbp11 depletion. Using RNA-seq, we demonstrated depletion of these IA genes resulting in a pathological reprogramming towards a senescent cellular state in endothelial cells, leading to IA formation.

Conclusion

We have identified a novel WBP11-PPIL4 axis essential for endothelial senescence and pathological reprogramming that plays a fundamental role in IA formation and growth. The discovery of this novel mechanism of endothelial cell homeostasis forms the basis of novel therapeutic interventions for IA.

8:35 – 8:40	Wrap-up/ Transition
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8:40 – 9:35	Peer Reviewed Abstract Session VI: Functional Moderators: Aviva Abosch and Kim Burchiel
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8:40 – 8:50	DBS for Depression Informed by Intracranial Recordings
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Sameer Sheth, MD

Introduction

The success of DBS for movement disorders has fueled its application for a variety of other disorders including treatment-resistant depression (TRD). Whereas initial open-label studies were encouraging, two pivotal trials were aborted after interim analyses.

Objectives

We seek to understand the neurophysiological underpinnings of TRD to better deliver DBS therapy to dysfunctional brain networks regulating emotional regulation and cognition. To do so, we borrow an approach commonly used in epilepsy surgery but rarely in other fields - the use of inpatient intracranial recordings to individualize network understanding.

Methods

We implant TRD patients with permanent DBS leads targeting two commonly used regions for depression (ventral capsule / ventral striatum and sub-callosal cingulate), as well as with temporary stereo-EEG electrodes

targeting depression-relevant frontotemporal regions. Patients were monitored in the inpatient unit for 10 days during a variety of recording and stimulation activities.

Results

The data-driven stimulation parameters are delivered during an 8-month outpatient trial following the inpatient phase. The first subject in our trial achieved symptom remission. Relapse during the double-blind, randomized withdrawal phase with subsequent remission following reinstating DBS demonstrated that this response was a true response, not sham. The rich intracranial neural data also allowed us to apply machine learning decoding approaches. We fit regularized regression models to depression severity scores using neural activity recorded across prefrontal sites. We identified spatio-spectral features, most notably gamma power in anterior cingulate cortex, that predicted depression severity robust to cross-validation.

Conclusion

The intracranial platform allows individualized appreciation of network pathology and therapy delivery for challenging neuropsychiatric disorders.

8:50 – 9:00	Decoding Dynamically Shifting States of Parkinson's Disease: Tremor, Bradykinesia and Effective Motor Control
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Wael Asaad, MD, PhD; Peter M Lauro; Shane Lee; Umer Akbar; David D Liu

Introduction

Parkinson's Disease (PD) is a neurodegenerative disorder with distinct motor manifestations. Despite this, the approach to understanding the circuit basis of this disorder – as well as its potential treatment using closed-loop deep brain stimulation (DBS) – has typically not leveraged possibly distinct neurophysiological biomarkers for cardinal features such as tremor and bradykinesia.

Objectives

To identify symptom-specific neurophysiological biomarkers of PD.

Methods

27 subjects with PD performed an intra-operative, naturalistic, target-tracking task. Movement trajectories were decomposed into epochs of tremor, bradykinesia, or effective motor control (accurate tracking) by referencing these metrics to control (non-PD) behavior on the same task.

Results

Tremor and bradykinesia were distinct states, anti-correlated in time. We applied an explainable machine-learning approach to identify neural biomarkers from subcortical and cortical signals that reflected these distinct states. In the subthalamic nucleus (STN), we found that tremor and bradykinesia had nearly, though not completely, opposite spectral fingerprints. States of effective motor control were further distinguishable. Meanwhile, cortical ECoG signals were often more capable of supporting accurate decoding of symptomatic state.

Across subjects, tremor and bradykinesia were more optimally decoded from different regions of the STN. This was confirmed within-subjects using a novel, high-resolution, robotic STN survey in 5 subjects.

Conclusion

These results highlight 1) decoding of individual PD symptoms is feasible; 2) multi-spectral decoding PD states may be necessary for optimal closed-loop neuromodulation; and 3) effective motor control may be uniquely differentiable from pathologic motor states, potentially serving as a target for rational neuromodulation.

9:00 – 9:10	The Genomics of Trigeminal Neuralgia With and Without Neurovascular Compression
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Kim J. Burchiel, MD; Ashwin A Kamath, MD; Scott Diehl; Olga Korczeniewska

Introduction

The currently accepted pathophysiologic model of trigeminal neuralgia (TN) is that neurovascular compression (NVC) is typically required, and that microvascular decompression (MVD) can result in long-term alleviation of TN pain. However, increasingly it has been recognized that patients may develop a typical TN syndrome without NVC. We conducted a retrospective genome-wide association study (GWAS) to determine if there is a genetic predisposition to the development of TN with and without NVC.

Objectives

To determine if there is a genetic predisposition for the development of TN with (TNWNVC) and without (TNWONVC) NVC.

Methods

132 patients with Type 1 TN (TN1) were included in this study. Two neurosurgeons who were not involved in the surgical treatment of these patients reviewed high resolution T2 MRI (BFFE), operative videos, and operative note on all patients. The Sindou classification was used, and the patients were divided into two groups: Those with Gr 0-1 NVC [no NVC + simple contact] (TNWONVC) and those with Gr 2-3 [compression + distortion] (TNWNVC). These subjects submitted DNA samples and GWAS analysis was carried out. Comparison was made to controls without TN.

Results

89 patients were found to have Gr 2-3 NVC, and 43 patients had Gr 0-1 NVC. The TNWNVC group had five single nucleotide polymorphisms (SNPs) which were significantly different than controls. The TNWONVC group showed these same five SNPs, but in addition had two additional SNP variants which were significantly associated.

Conclusion

It appears that both TN with and without NVC occurs on a background of a genetic predisposition. TNWNVC requires five genetic variants, and TNWONVC requires these plus two additional variants. These findings need to be expanded and replicated, but suggest that our understanding of the pathologic basis of TN should be reconsidered.

9:10 – 9:20 Therapeutic mechanism of DBS in Tourette Syndrome

Aaron E. Rusheen; Abhinav Goyal; Jason Yuen; Juan Rojas Cabrera; Hojin Shin; Kevin Bennet; Charles Blaha; Yoonbae Oh; **Kendall H. Lee, MD, PhD**

Introduction

Deep brain stimulation (DBS) of the centromedian parafascicular complex (CM/Pf) is effective for medical refractory Tourette syndrome. The CM/Pf sends dense glutamatergic projections to the dorsal striatum. The dorsal striatum has high dopaminergic tone and its dysfunction has been proposed to underlie tic behavior.

Objectives

Test the hypothesis that CM/Pf DBS activates thalamostriatal glutamatergic neurons to evoke dopamine release and reduce tics in a rat model of Tourette syndrome.

Methods

A Tourette syndrome model was generated by striatal infusion of the GABA-A antagonist bicuculline in rats that underwent CM/Pf DBS with a concentric bipolar electrode. Tonic and phasic dopamine were recorded with voltammetry. Pharmacologic studies were performed with nicotinic cholinergic antagonist mecamylamine, dopamine1-Receptor antagonist SCH23,390, and D2-R antagonist sulpiride. A separate group of rats were injected with the excitatory viral vector AAV1::CaMKIIa-Chronos-eGFP and the inhibitory viral vector AAV1::CaMKIIa-eNpHR3.0-eYFP. Optogenetic stimulation was applied with simultaneous dopamine recording.

Results

DBS elicited 106.7 ± 9.3 nM phasic dopamine release and increased tonic dopamine by 10.6 ± 3.0 nM. DBS reduced tic frequency by $31.8 \pm 6\%$. Optogenetic activation elevated tonic dopamine by 14 ± 5 nM, and optogenetic inhibition had no effect. Mecamylamine reduced phasic dopamine release by 59 ± 13.2 nM, abated tonic dopamine elevation, and reversed the therapeutic effect of DBS. Sulpiride, but not SCH23,390, reversed the therapeutic effect of DBS, indicating dopamine activity at D2-Rs.

Conclusion

Our results demonstrate CM/Pf DBS therapeutically reduces tics by activation of thalamostriatal glutamatergic neurons, induction of striatal dopamine release via cholinergic interneurons, and resultant dopamine activity at D2-Rs.

9:20 – 9:30 Responsive nucleus accumbens deep brain stimulation restores eating control in severe obesity

Casey H. Halpern, MD

Introduction

The presence of loss of control (LOC) eating appears to predict treatment-resistance in obesity, including to gastric bypass surgery. Responsive deep brain stimulation guided by low frequency changes in the nucleus accumbens (NAc-rDBS) was previously found to block LOC eating-like behavior in mice. Following this novel preclinical work, the U.S. Food and Drug Administration approved a first-in-human study.

Objectives

Assess early insights of NAc-rDBS for LOC eating in obesity (NCT0388670).

Methods

Two female participants with binge-eating disorder (the severest form of LOC eating) and morbid obesity ($BMI > 45 \text{ kg/m}^2$) refractory to gastric bypass were implanted with bilateral NAc depth leads connected to a rDBS system. Field potentials were recorded in the clinic during eating tasks. Outside of the clinic, participants maintained a diary describing craving severity and triggered data storage to time-stamp NAc field potentials.

Results

In the clinic, the left ventral NAc region activity revealed increase delta (2-4Hz) and theta (4-8Hz)-band power immediately preceding bites of highly palatable food. In the ambulatory setting, we also observed increased bilateral ventral NAc delta band activity that appeared selective for states of food craving prior to LOC eating. NAc-rDBS was programmed to detect low-frequency activity and stimulate in response (125 Hz; 5-sec bursts $\times 2$, $0.5\text{--}1.5 \mu\text{C/cm}^2$). This paradigm resulted in a decrease in frequency and severity of LOC eating at 6-months (i.e. the primary endpoint) and decreased body weight (-5.9 kg ; -8.2 kg). Subjects exhibited substantially improved binge-eating disorder or no longer met criteria.

Conclusion

These findings provide early support for restoring inhibitory control with electrophysiologically-guided NAc DBS.

9:30 – 9:35 Wrap-up/ Transition

9:35 – 9:50 Break

9:50 – 10:55 Peer Reviewed Abstract Session VII: Technology and Translation
Moderators: Douglas Kondziolka and William Curry

9:50 – 10:00 Nanoshunt for hydrocephalus

Cargill H. Alleyne, MD; Kayyani Adiga

Introduction

Treatment of hydrocephalus requires the shunting of cerebrospinal fluid from the ventricle to the abdomen via a catheter tunneled underneath the skin. This can lead to pain and discomfort, disconnection, migration, occlusion, infection, bowel injury, and shunt revision. The technique was developed in the late 1800's and has not undergone significant change in over a century.

Objectives

The purpose of the study was to develop a device without a distal tubing for the treatment of hydrocephalus. The proposed NanoShunt would convert CSF into ultrafine mist droplets which would diffuse through the scalp, obviating the need for distal tunneling of a catheter thus decreasing the surgical risk.

Methods

A prototype implant was developed whereby CSF is transferred to the implant via a catheter and is atomized into ultrafine mist. The fine mist dissipates through the scalp in vitro and may be absorbed by the scalp's rich vascular supply in vivo. Alternatively, the mist may be diverted into an air-cavity (e.g. mastoid air cells). The implant is powered by a battery.

Results

The implant was tested in vitro for concept demonstration. It consists of the implant (which consists of a mesh atomizer, wick, and receiver coil), a Band-Aid-like patch with a transmitter coil, and an external driver and battery pack.

Conclusion

We have developed a prototype device designed to convert CSF in ultra-fine mist particles. Future animal experiments are planned using an animal model of hydrocephalus.

10:00 – 10:10 Personalization of Tumor Treating Fields: A Glioblastoma Organoid Model for In Vitro Efficacy

Benjamin Hendricks, MD; Jayati Chakrabarti; Jennifer Eschbacher; Yana Zavros

Introduction

Tumor treating field (TTF) therapy is the newest addition to the glioblastoma (GBM) standard of care. TTF for GBM involves delivery of a 200kHz alternating electric field at a $>0.7\text{V/cm}$ field strength, as determined by investigation within multiple cell lines and patient derived cultures.

Objectives

Given the well-defined presence of tumor heterogeneity within in vivo GBM, a patient-specific treatment parameter should optimize TTF efficacy. To test this hypothesis, we generated a patient-specific organoid model of GBM for TTF assessment.

Methods

Three patient-derived GBM organoid lines (GBMOs) were cultured from newly diagnosed tumor samples. Each was grown 72-hours with 1 of 24 TTF frequency (150 - 275kHz) and electric field strength (0.7 - 2V/cm) combinations. Microscopic quantification of surface area at 0-, 24-, 48-, and 72-hours was conducted. Changes in cell populations were identified by spectral flow cytometry and confocal microscopy.

Results

Across the parametric spectrum, GBMOs demonstrated the largest surface area reduction at 275kHz but with line specific differences in susceptibility ($p < 0.0001$). Compared to the 200kHz standard of care, 275kHz provided a line-dependent 22-46% enhanced efficacy. Universally, increasing field strength was associated with an increased efficacy ($p < 0.0001$). Flow cytometry revealed both tumor cells and glioma stem cell sub-populations have differing susceptibility to treatment ($p < 0.0001$).

Conclusion

Differing susceptibility to TTF at various parameters within the 3 GBMOs is supportive of the impact tumor heterogeneity has on in vivo efficacy of TTF. Although 275kHz remained the optimized frequency for all GBMOs, greater resolution within the frequency testing spectrum may isolate a line specific optimized frequency.

10:10 – 10:20 Deep phenotyping of drug responses in patients with gliomas using tumor-embedded microdevices

Pier Paolo Peruzzi, MD, PhD; Christine Dominas; Patrick Wen; E. Antonio Chiocca, MD PhD; Oliver Jonas

Introduction

The lack of reliable predictive biomarkers is a major obstacle for the advancement of therapy for high grade gliomas (HGG), and particularly glioblastoma (GBM).

Objectives

To demonstrate the safety and feasibility of integrating drug-releasing intratumoral microdevices (IMD) into standard neurosurgical practice for glioma resection, as a novel method to predict responses and guide selective pharmacological therapies in a personalized fashion.

Methods

This is a non randomized phase 1 clinical trial enrolling patients with known or suspected supratentorial glioma, for which a craniotomy for tumor resection was indicated. Each tumor was implanted with two IMDs which remained indwelled into the tumor for the entire duration of surgery, allowing time for drug release. At the end of the procedure, the IMDs were retrieved with a cuff of surrounding specimen and sent to the lab for molecular analysis.

Results

Six patients were enrolled in this study. The application of IMD did not result in significant changes in the surgical procedure and its aftermath. Twelve out of 12 inserted IMD (100%; 90% CI (61%-100%)) were successfully retrieved and none was lost or abandoned in the patient. There were no immediate (<48 hours after surgery), nor delayed (<30 days) adverse events (AEs). Eleven out of 12 (92%; 90% CI (66%-100%)) total implanted IMD provided specimens which could be successfully processed for downstream molecular analysis. Nine different drugs per IMD were successfully analyzed, and robust correlations could be made between the IMD readout and clinico-radiological responses after systemic therapy.

Conclusion

The use of IMD is safe and can be seamlessly integrated into neurosurgical-oncological practice. The amount of information obtained with IMD allows unprecedented characterization of tissue effects of any drugs of interest, within the physiological context of the intact tumor.

10:20 – 10:30 Photoacoustic and Epigenetic Nerve Scaffolds for Nerve Regeneration

Elias Boulos Rizk, MD

Introduction

Autograft remains the gold standard for PN injury repair. Still, various disadvantages remain.

Objectives

Bioimaging of peripheral nerves has a great potential to contribute to the diagnosis, operative treatment, and monitoring of postoperative outcomes. Herein, we report an epigenetic and photoacoustic citrate-polymer based NGC (EPC-NGC) for the optimum repair and functional recovery of critical-sized PN gaps (15 mm in rats).

Methods

EPC-NGCs facilitate local delivery of an inexpensive and stable (half-life of over 100 days) folate (also known as vitamin B9) directly to the peripheral injury site at a critical concentration to enhance nerve regeneration and functional recovery through an intriguing epigenetic modulation and enables photoacoustic imaging (PAI) in the tissue transparent near-infrared (NIR) window for potential non-invasive, real-time, in-situ monitoring of nerve scaffold degradation and nerve regeneration.

Results

Specifically, we have developed multifunctional multi-channeled biodegradable elastic NGCs with compelling data to support that biologically stable folate displayed intriguing dose-dependent epigenetic and biomechanical effects to promote neuronal differentiation migration and proliferation of both rat Schwann and neuron cells, and the regeneration and functional recovery of 20 mm sciatic nerve defects in rats as early as 4 weeks post-implantation. The NGCs also displayed unexpected strong absorption in near-infrared-I (NIR-I, 700-1000 nm) for PAI.

Conclusion

This work represents a new direction for the optimal design of imageable NGCs with suitable epigenetic, biomechanical, and topographical cues for the regeneration and functional recovery of critically sized nerve defects.

10:30 – 10:40 MRgFUS-enhanced delivery of chemotherapeutics for Diffuse Intrinsic Pontine Glioma: Phase I Clinical Trial

James T. Rutka, MD, PhD

Introduction

DIPG is a fatal brainstem tumor in early childhood. Despite radiation therapy (XRT) and countless chemotherapy trials, the prognosis for this disorder has not changed. Failure of drug therapy may relate to an intact blood-brain barrier (BBB), and penetration of targeted agents within the pons. Our preclinical studies have shown that BBB disruption in animal models can lead to increased concentrations of drugs to therapeutic levels within the brainstem. Here, we sought to determine if MRgFUS in conjunction with simultaneously administered intravenous (IV) microbubbles could lead to enhanced concentration of drug within the brainstem of children harboring DIPG.

Objectives

Initiation of a Phase I, dose escalation clinical trial for children with radiologically/pathologically confirmed DIPG using Exablate BBB disruption, Doximity (TM) microbubbles, and IV doxorubicin.

Methods

Children diagnosed with DIPG are treated with standard therapy comprised of fractionated XRT over 6 weeks. Following XRT, Exablate BBB disruption is performed under general anesthesia with IV administered doxorubicin. Pre- and post-procedural MRI scans are performed. Blood biomarkers are drawn during the procedure. Recovery is facilitated in the intensive care unit. Following an observation period of 24 hr, children are discharged. Three cycles of Exablate therapy are performed at monthly intervals. MRI scans are performed at 1 and 3 months following the final treatment session. Physical, neurological, and radiological exams are performed to assess for feasibility and safety.

Results

This Phase I clinical trial is now FDA and Health Canada Approved. Enrollment has begun at the Hospital for Sick Children, Toronto, and the Children's National Hospital, Washington. Recruitment is scheduled for 20 children with DIPG to receive the Exablate strategy for BBB disruption of the brainstem. Preliminary results of the trial will be presented.

Conclusion

DIPG is a fatal tumor for which there are no good treatment options. This Phase I clinical trial is founded on the premise that treatment failures to date relate to inability of targeted chemotherapeutic agents to penetrate the intact BBB of the pons. It is hoped that novel drug delivery strategies such as this will improve the prognosis of children with DIPG.

10:40 – 10:50 Adeno-Associated Viral Directed Evolution for Targeted Gene Editing of Plexiform Neurofibromas

Nicholas M. Boulis, MD; David Schafer

Introduction

Neurofibromatosis involves the development of neurofibromas throughout the body as a result of mutations to the neurofibromin gene on chromosome 17. Neurofibromin is a tumor suppressor protein that inhibits the activity of the Ras oncogene. Recent advances in gene editing suggest the possibility of correcting this mutation using Adeno-associated viral vectors. Plexiform neurofibromas present a unique challenge for the management of neurofibromatosis patients, as resection is often impossible without creating severe neurological deficits.

Objectives

For such a strategy to work, a high percentage of the tumor's cells would need to receive the gene editing transgenes. The present set of experiments attempt to use directed evolution to select for novel AAV serotypes capable of enhanced binding to human nerve sheath tumor cells. It further attempts to validate this targeting by using patient derived in vitro models of nerve sheath tumors.

Methods

Directed Evolution is a technique that biopans for novel AAV clones with particular binding and biodistribution properties. Starting with a library containing millions of AAV, clones were selected and amplified based on binding to rodent Schwann cells. In a final step, a human schwann cell line was used to select for AAV binding.

To assess the potential of these clones to enhance gene delivery to human nerve sheath tumors, primary cell cultures were made from human Schwann cells recovered from residual sural nerves harvested for nerve repairs, as well as tumor cells harvested from human Schwannomas and Plexiform Neurofibromas resected from patients with isolated or syndromic masses. The resulting AAV clones were compared to AAV2, AAV5, and AAV6 for comparative potency for gene delivery to human tumors.

Results

Initial results demonstrate marked enhancement of transgene delivery to human tumor derived 2D and spheroid cultures using the C5 clone derived from directed evolution on mixed rodent schwann cell and a human schwann cell line. Progress toward a directed evolution AAV library panned directly on patient derived cells will be presented.

Conclusion

Convection enhanced delivery of AAV vectors may provide a means to halt the growth of plexiform neurofibromas without compromising neurological function. Novel AAV mutants derived from directed evolution will facilitate the development of this therapeutic approach.

10:50 – 10:55 Wrap-up/ Transition

10:55 – 11:50 Peer Reviewed Abstract Session VIII: Other and Education

Moderators: Shelly Timmons and Howard Riina

10:55 – 11:05 Promoting Diversity in Neurosurgery - a Multi-Institutional Scholarship Based Approach

Allan D. Levi, MD, PhD; Nicholas Theodore, MD; Gregory J. Zipfel, MD; Nelson M. Oyesiku, MD, PhD; Linda M. Liao, MD, PhD; John G. Golfinos, MD; Brenton Henry Pennicooke, MD; Anthony Frempong-Boadu, MD; Langston T. Holly, MD; Stephanie Chen; Michael E. Ivan, MD; Aviva Abosch, MD, PhD

Introduction

Diversity in organized medicine is slowly improving, however disparities remain in both racial and gender composition. Surgical subspecialties such as orthopedics and neurosurgery have some of the largest gaps. The reasons for these disparities are multifactorial.

Objectives

In the current presentation we outline a pipeline approach using summer scholarships to encourage medical students to seek careers in neuroscience and neurosurgery thereby reducing these disparities.

Methods

We describe a multi-institutional approach to create summer scholarship funds and opportunities for black students. Fundraising, social media awareness, ranking and selection process of medical students, salary costs, housing, and creation of rotations are discussed.

Results

This scholarship program is in its fourth year and has grown from a single institution - one student scholarship to seven institutions (UM, JHU, WashU, UNC, UN, UCLA, NYU) with a minimum of 13 scholars per year. The 9-week program targets students in cities all over the U.S., and many from schools without parent neurosurgical programs.

Conclusion

This grass roots approach can have considerable impact on future generations of neurosurgeons. Mentorship and recruitment of black medical students early in their careers is only one mechanism of many to reduce inequities. Success of this scholarship program will be assessed prospectively by determining the percentage of these scholars who eventually match in neuroscience, neurology or neurosurgery.

11:05 – 11:15 Inspiring Translational Neuroscience Inclusion through an NIH R-25

Richard G. Ellenbogen, MD; Jeffrey Ojemann; Christine MacDonald; James Pridgeon

Introduction

Over the past 15 years, we have placed 171 college undergraduate students age 18 and older from 90 different schools in our Training Grant, Neurological Surgery Summer Student Program. Approximately 50% of the students are under-represented minority students (URM). In 2016, our program was awarded 5-year NIH NINDS R25 funding which was renewed in 2021 for an additional 5-year period. The NIH funding allowed us to expand the program and pay stipends allowing a wider range of participation by underrepresented minorities and economically disadvantaged students.

Objectives

The principal aim of the program is to provide modeling and mentorship which provides transformative experience for all students and especially URM students. The hope is that this experience engenders them to pursue medical or neuroscience research careers.

Methods

We have had over 1000 applicants from 43 states and US territories. Students have participated in 20 different laboratories hosted by 24 faculty basic science members. They have attended over 140 unique Neurological Surgery Grand Rounds presentations and observed over 500 surgical procedures and clinical shadowing opportunities by following 23 different attending neurological surgeons at 3 UW-affiliated hospitals. Students have also participated in 92 weekly student group presentations delivered by 27 different neuroscience faculty members. Longitudinal mentorship extends to medical school, residency and PhD programs, and professional and career advancement.

Results

Although still a very young program, over 25 students from earlier classes have gone on to medical school or Ph.D. neuroscience programs. A commitment to long term mentorship has been achieved by the full compliment of students, neurosurgeons and neuroscientists.

Conclusion

Post program longitudinal review, coupled with surveys and evaluations suggest that diversity modeling and inclusion bolsters the principal aim that this process is effective in inspiring and mentoring URM students to pursue the fields of biomedical research or clinical careers.

11:15 – 11:25 NYUMets: a massive, open-source, longitudinal dataset of metastatic brain cancer with clinical and imaging annotations

Douglas S. Kondziolka, MD; Eric Oermann

Introduction

The development of accurate and generalizable machine learning algorithms requires sufficient quantities of diverse data. This poses a challenge in healthcare due to the sensitive and siloed nature of biomedical data. Furthermore, the longitudinal nature of cancer necessitates the assembly of longitudinal, multimodal datasets which are challenging to build and access.

Objectives

To build the world's largest, longitudinal dataset of real world tumor imaging with multimodal annotations describing the clinical care of patients with metastatic brain tumors.

Methods

The clinical registry of the NYU Center for Advanced Radiosurgery (CAR) was converted into a SQL database. Each time point in the dataset was augmented with all available image studies from the hospital PACS, and with all available medication prescriptions from the EHR. MRI studies were co-registered at each time point, resampled to 1mm isotropic dimensions, and pre-processed using standard protocols. The final dataset was de-identified of structured data, skull stripped, and uploaded to Amazon S3. Naïve out-of-domain transfer learning was assessed on tumor segmentation with vanilla U-nets using the Brain Tumor Segmentation Challenge (BraTS) 2021 dataset.

Results

1,293 patients with 3,449 radiosurgery high-resolution MRI studies were identified in the CAR registry. These were augmented by 27,006 diagnostic MRI studies from PACS matched on patient MRN. After excluding studies for incomplete sequences, failed registration, or duplication we obtained a final dataset of 2,148 patients, 13,381 MRI studies, and 2,115 expert tumor segmentations derived from gamma knife radiosurgery plans. A total of 490,096 prescriptions were written for 19,083 unique medications and dosages. A vanilla U-Net using simple supervised pre-training obtained a mean DICE score of 0.78 on the BraTS 2021 validation set compared to baseline performance of 0.76 with training only on BraTS.

Conclusion

NYUMets is the world's largest publicly available dataset of annotated tumor imaging, brain metastases, and longitudinal multi-modal medical data. Opening this data to the scientific research community has the

potential to substantially advance the state of the art in medical computer vision, and to potentially unlock new insights into metastatic brain tumor science and care. The dataset can be accessed at <https://nyumets.org/> after registration with the NYUMets team and creation of an Amazon Web Services account.

11:25 – 11:35 Stroke in Native Americans: A Treatable Scourge

Robert J. Dempsey, MD; Umadevi Wesley; Stephanie Wilbrand; Carol Mitchell

Introduction

Stroke is a neurosurgically treatable disease if we embrace epidemiology, prevention and rehabilitation as a cohesive program. Stroke will soon become the number one cause of death and disability in the developing world. The U.S. population with the greatest risk factors for stroke are reservation-based Native Americans.

Objectives

In response, we have initiated a program to identify risk factors contributing to this disparity within the Wisconsin reservation-based tribes.

Methods

In a partnership with the Oneida Nation Health Division, a program was initiated to identify risk factors in tribal members, using tribal coaches to change risk factors.

Results

In 81 tribal elders, we found a striking percentage of 76.2% having atherosclerotic changes in their carotid bulb, known risk factors for stroke and vascular cognitive decline. 50% were diabetic, 52.4% had a BMI greater than 30 and 75% had elevated A1c levels greater than 5.6. We further studied proteomic screening of inflammatory adipokines associated with increased fat cells, capable of causing accelerated atherosclerosis and multisystem damage. 20 of 58 potentially inflammatory proteins were found to be elevated in tribal members compared to controls. These novel blood markers are associated with stress, atherosclerosis, cognitive impairment and stroke risk factors.

Conclusion

Stroke risk factors heavily associated with atherosclerosis are extremely prevalent in at risk tribal members. Decreased stroke will only come about by combining prevention with acute intervention therapies with the assistance of the Tribal Health Council utilizing their health interventions for targeted individuals.

11:35 – 11:45 A New Disease-Specific Quality of Life Instrument for Sporadic Vestibular Schwannoma: The VSQOL

Michael J. Link, MD; Matthew L Carlson; Christine Lohse; Nicole Tombers; Devin McCaslin; Aniket Saoji; Melanie Hutchins; Kathleen Yost

Introduction

Facial nerve function, hearing preservation, and tumor control have been the primary benchmarks used to assess vestibular schwannoma outcomes. Acknowledging the frequent discrepancy between what physicians prioritize and what patients value, there has been increasing prioritization of patient-reported outcome measures when evaluating the impact of being diagnosed with a vestibular schwannoma and its treatment. The current study describes the development and validation of a new disease-specific quality of life measure: the Vestibular Schwannoma Quality of Life (VSQOL) Index. This valid and reliable instrument builds upon previous work and seeks to overcome potential limitations of prior instruments including omission or underrepresentation of domains that frequently impact well-being such as pain, cognition, and fatigue, as well as satisfaction or regret surrounding treatment decisions. Furthermore, inclusion of other overlapping

features that are often overlooked, such as occupational limitations and need for permanent disability, reflect a more global picture of disease impact.

Objectives

Motivated by past studies reporting that general instruments assessing quality of life in patients with vestibular schwannoma (VS) omit or underrepresent key factors that impact well-being, we describe the development and validation of a new disease-specific quality of life measure: the Vestibular Schwannoma Quality of Life (VSQOL) Index.

Methods

The content development phase comprised the creation of a measurement framework to identify clinically important domains and the identification and prioritization of feelings or concerns that people with VS may experience. The validation phase encompassed further item reduction through exploratory factor analysis. During both phases, we leveraged data from cross-sectional and longitudinal surveys, expertise from a multidisciplinary working group, and a broad range of experiences from patient focus groups (N=43 during content development, N=264 during validation).

Results

The VSQOL Index consists of 40 items that evaluate the impact of VS diagnosis and its management on quality of life, treatment satisfaction, and employment. Domain scores range from 0 (worst) to 100 (best). Cronbach's alphas measuring internal consistency of items within each domain were high, ranging from 0.83 to 0.91, as were the correlation coefficients measuring test-retest reliability of domain scores, ranging from 0.85 to 0.94.

Conclusion

The VSQOL Index is a valid and reliable measure that overcomes limitations of prior instruments including omission or underrepresentation of domains that frequently impact well-being such as pain, cognition, fatigue, regret surrounding treatment decisions, and occupational limitations to comprehensively evaluate the impact of VS diagnosis or its treatment on quality of life.

11:45 – 11:50 Wrap-up/ Transition

11:50 – 12:00 Break

12:00 – 12:45 <u>Presidential Address</u>

12:00 – 12:05 Introduction of the Academy President: Dr. Daniel Yoshor

12:05 – 12:45 Presidential Address: Dr. James Markert
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1:30 – 4:30 <u>Joint Academy Emerging Investigator's Program</u>
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Program Directors: Dr. Gregory Zipfel

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: Mark Johnson and Linda Liao

7:30 – 7:35 Session Introduction
Frederick Barker, MD

7:35 – 7:45 AI-based molecular classification of diffuse gliomas using rapid, label-free optical histology

Todd C. Hollon, MD; John G. Golfinos, MD; Daniel A. Orringer, MD; Mitchel Berger; Georg Widhalm; Shawn L. Hervey-Jumper, MD; Christian Freudiger; Karin M. Muraszko, MD; Wajd Al-Holou, MD; Oren Sagher, MD; Volker Neuschmelting; Sandra Camelo-Piragua

Introduction

Molecular classification has transformed the management of brain tumors by enabling more accurate prognostication and personalized treatment. Access to timely molecular diagnostic testing for brain tumor patients is limited, complicating surgical and adjuvant treatment and obstructing clinical trial enrollment.

Objectives

We aim to develop a rapid (<90 seconds), AI-based diagnostic screening system that can provide molecular classification of diffuse gliomas and report its use in a prospective, multicenter, international clinical trial of diffuse glioma patients (n = 153).

Methods

By combining stimulated Raman histology (SRH), a rapid, label-free, non-consumptive, optical imaging method, and deep learning-based image classification, we are able to predict the molecular features used by the World Health Organization (WHO) to define the adult-type diffuse glioma taxonomy. We developed a multimodal training strategy that uses both SRH images and large-scale, public diffuse glioma genomic data in order to achieve optimal molecular classification performance.

Results

Four institutions (NYU, UCSF, Medical University of Vienna, University Hospital Cologne) were included for prospective patient enrollment. Using our system, called DeepGlioma, we were able to achieve an average molecular genetic classification accuracy of 93.2% and identify the correct diffuse glioma molecular subgroup with 91.5% accuracy. DeepGlioma outperformed conventional IDH1-R132H immunohistochemistry (94.2% versus 91.4% accuracy) as a first-line molecular diagnostic screening method for diffuse gliomas.

Conclusion

Our results demonstrate how artificial intelligence and optical histology can be used to provide a rapid and scalable alternative to wet lab methods for the molecular diagnosis of brain tumor patients during surgery.

7:45 – 7:55 ARID1A Mutation Associated with Recurrence and Shorter Progression-free Survival in Atypical Meningiomas

Raj K Shrivastava, MD; Russell McBride; Robert Sebra; Melissa Umphlett

Introduction

The oncologic outcomes for atypical meningiomas are not monolithic and range from favorable to grim. Generally, patients that have had a prior recurrence have a substantially elevated risk of a future recurrence. Additionally, certain tumor genomic profiles have been shown as markers of poor prognosis.

Objectives

We sought to characterize the genomic differences between primary and recurrent tumors as well as assess if those differences had implications on recurrence.

Methods

Through a review of our institutional cohort of meningiomas with accompanying targeted next generation sequencing data, we identified primary and recurrent gross totally resected WHO grade II meningiomas with > 30 days of post-surgical follow-up. For genes with a prevalence of > 5% in the cohort, we compared the mutational prevalence in primary and recurrent tumors. For a gene of interest, we assessed the time to radiographic recurrence using adjusted cox-regression.

Results

We identified 88 meningiomas (77 primary, 16 recurrent) with a median follow-up of 5.33 years. Mutations in ARID1A found in association with recurrent tumors (7/16 recurrent tumors vs 5/72 primary tumors, $p < 0.001$). In the whole cohort, mutations in ARID1A were not associated with alterations in time to recurrence after adjusting for recurrence status ($p = 0.713$). When restricted to primary tumors, ARID1A is associated with a 625% increase in the hazard of recurrence (HR = 7.26 [1.42-37.0]; $p = 0.017$).

Conclusion

We demonstrate mutations in ARID1A, a chromatin remodeling gene, in a higher prevalence in recurrent tumors. We further demonstrate that when mutations in ARID1A are present in primary atypical meningiomas, these tumors tend to have worse prognosis. Further prospective study may validate ARID1A as a prognostic marker. Additionally, this finding may have implications for the treatment of select meningiomas with HDAC inhibitors that specifically target the alterations in chromatin structure as has been done in other ARID1A mutant neoplasms.

7:55 – 8:05 Epigenetic suppression allows GBM to maintain p53 wild-type status
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Jung Park, MD, PhD; Michael Schulder, MD; John A. Boockvar, MD

Introduction

Glioblastoma (GBM) is the most common primary brain malignancy in adults. The vast majority of GBM cases maintain wild-type status of p53, the protein considered to be the most critical tumor suppressor. How GBM displays such a malignant phenotype despite retaining normal p53 protein is unknown.

Objectives

Establish the molecular mechanism through which p53 is permitted to maintain wild-type status in GBM. We then aim to translate these molecular findings into clinical therapeutics via a randomized clinical trial.

Methods

A variety of molecular biological techniques were employed. These include, but are not limited to, CRISPR-CAS, next generation sequencing, stereotactic mouse brain injections, and histological analyses of human GBM tumors. Approval for a small, single site, blinded, randomized clinical trial for newly diagnosed GBM is underway. Overall survival, recurrence-free survival, immunohistochemical analyses will be assessed.

Results

Human p53 wild-type GBM cell lines express Brd at higher levels compared to p53 mutant cell (~10 fold, $p < 0.05$). Cell viability assays show that deletion of Brd leads to a ~50% decrease ($p < 0.05$) in cell survival. Critically, this effect is abrogated upon deletion of p53 itself, showing that Brd functions through the inhibition of p53's tumor suppressive effects. Furthermore, immunoprecipitation of Brd shows that it is a member of a complex of proteins that bind to p53 target genes. The importance of these findings are corroborated in vivo, as mice stereotactically injected with human GBM cells that lack Brd survive ~2-fold longer ($p < 0.05$) than mice that retain Brd. We have begun the process to start clinical trials with an already FDA-approved drug (currently used for the treatment of lymphoma and sarcoma) that blocks the Brd pathway.

Conclusion

Brd is the key suppressor of p53 target genes in GBM. Brd binds to a multimeric protein complex, which inhibits p53 target genes. Thus, the tumor suppressive effects of p53 are prevented in GBM. Inhibition of this pathway in GBM patients is underway.

8:05 – 8:15	A Novel Technique for Chronic Convection-Enhanced Delivery Provides Unlimited Drug Regimens and is Effective for GBM
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Jeffrey N. Bruce, MD; Eleonora Francesca Spinazzi, MD; Michael G Argenziano; Pavan S Upadhyayula, MD; Matei A Banu, MD; Justin Neira, MD; Dominique Higgins, MD; Peter Wu; Nathalie Y.R. Agar, PhD; Peter Sims; Mary Welch; Andrew Lassman, MD; Fabio Iwamoto; Randy D'Amico, MD; Jack Grinband; Peter D. Canoll, MD, PhD

Introduction

Compared to systemic delivery, convection-enhanced delivery (CED) provides a sizeable advantage for achieving therapeutic drug concentrations without systemic toxicities. A major CED shortcoming has been restriction to a single treatment of limited duration due to infection risks from external pumps. Therefore, we engineered a subcutaneously implanted catheter-pump system capable of repeated, unlimited, chronic local drug delivery into the brain.

Objectives

To test the feasibility, effectiveness and safety of chronic CED in glioma patients using a subcutaneous pump/catheter construct.

Methods

Five recurrent GBM patients had catheters stereotactically implanted into the tumor/surrounding brain and connected to subcutaneously implanted pumps that infused topotecan over a 30-day period, after which pump was removed and tumor resected. Multiple MRI-localized biopsies taken immediately pre- and post-treatment were analyzed with advanced histopathologic and molecular techniques. Drug distribution was non-invasively monitored in real-time using MRI of co-infused gadolinium.

Results

Chronic CED of topotecan eliminated proliferating tumor cells without brain toxicity. MRI of co-infused gadolinium demonstrated large, stable drug distribution volumes. Analysis of tissue taken before and after treatment (integrated for the first time into a human glioma trial) facilitated an unprecedented tissue-based assessment which demonstrated decreased proliferating tumor signature without neuronal toxicity.

Conclusion

Chronic CED of topotecan is safe, effective and clinically feasible for recurrent glioblastoma. This novel drug delivery strategy and innovative clinical trial paradigm overcomes current limitations in delivery and treatment response assessment, as shown here for glioblastoma, and is potentially applicable for other anti-glioma agents as well as other CNS diseases.

8:15 – 8:20	Wrap-up/ Transition
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8:20 – 9:10	Academy Award Presentation and Lecture
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8:20 – 8:25	Introduction of Academy Award Winner
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Michael Vogelbaum, MD, PhD

8:25 – 8:35	A population-normalized tractographic fiber atlas of the anterior limb of the internal capsule
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Garrett Banks, MD; Sarah Heilbronner, PhD; Wayne Goodman, MD; Sameer A. Sheth, MD, PhD

Introduction

The anterior limb of the internal capsule (ALIC) is a white matter highway that connects several subcortical structures to the prefrontal cortex. Although there have been many surgical interventions in the ALIC for psychiatric illnesses, there is still significant debate regarding how to target this area due to an incomplete understanding of connectivity in the region.

Objectives

We aim to use a diffusion tensor imaging analysis to study how thalamic and subthalamic pathways traveling to the prefrontal cortex are organized in the ALIC.

Methods

Public imaging data from 100 random subjects from the WU-Minn Human Connectome Project were used to analyze tractographic fiber patterns from the subthalamic nucleus, the medial dorsal nucleus, anterior nucleus, and ventral anterior nucleus of the thalamus. We used the FMRIB Software Library (FSL) to perform probabilistic tractography and study the variance of fibers between the 100 subjects in order to build an ALIC atlas.

Results

The results showed that posteriorly there is an organizational gradient of thalamic fibers medially and STN fibers laterally in the ALIC that fades more anteriorly. Also, while posteriorly fibers organize more strongly by their subcortical connectivity, more anteriorly in the ALIC fibers better organize by their cortical connectivity. (Figure 1) This shift occurs approximately around the anterior commissure.

Conclusion

These results are important for understanding the differences in therapeutic effects observed in different areas of the ALIC. An improved understanding of how fibers shift their primary organizing principal from their subcortical connectivity to their cortical connectivity may help in the improvement of current and future therapies in the ALIC.

8:35 – 8:40	Introduction of NREF Academy Winners (3)
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8:40 – 8:55	American Academy Young Clinician Investigator & Research Fellowship Grant Recipients
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8:55 – 9:05	Emerging Investigator Program
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Gregory Zipfel, MD

9:05 – 9:10	Wrap-up/Transition
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9:10 – 9:25	Break
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9:25 – 10:10 Peer Reviewed Abstract Session IX: Brain Tumor
Moderators: Michael Schulder

9:25 – 9:35 Subclonal evolution and expansion of spatially distinct glioma stem cells is associated with recurrence in glioblastoma

Wajid Al-Holou, MD; Hanxiao Wang; Visweswaran Ravikumar; Sunita Shankar; Ziad Fehmi; Morgan Oneka; Roel Verhaak; Hoon Kim; Drew Pratt; Sandra Camelo-Piragua; Corey Speers; Daniel Wahl; Todd Charles Hollon, MD; Oren Sagher, MD; Jason Heth, MD; Ana de Carvalho; Tom Mikkelsen, MD; Arvind Rao; Karin M. Muraszko, MD; Alnawaz Rehemtulla

Introduction

Glioblastoma (GBM) is a lethal disease characterized by inevitable recurrence.

Objective

The objective of this study was to investigate the molecular pathways mediating resistance, in hopes of identifying novel therapeutic targets.

Methods

We developed a longitudinal in vivo recurrence model utilizing patient-derived explants to produce paired specimens(pre- and post-recurrence) following temozolomide(TMZ) and radiation(IR). These specimens were evaluated for treatment response and to identify gene expression pathways driving treatment resistance. Findings were clinically validated using spatial transcriptomics of human GBMs.

Results

These studies reveal in replicate cohorts, a gene expression profile characterized by upregulation of mesenchymal and stem-like genes at recurrence. Analyses of clinical databases revealed increased expression of this transcriptional profile to be significantly associated with worse median overall survival (248 days vs 430 days, $p=0.0004$), and upregulation of this profile at recurrence. Most notably, we identified upregulation of TGF β signaling, and more than one-hundred-fold increase in THY1 levels at recurrence. Utilizing cell sorting, we observed that THY1-positive cells represented <10% of cells in the treatment-naïve tumors and 75-96% in the recurrent tumors. We then isolated THY1-positive cells from treatment-naïve patient samples and determined that they were inherently resistant to chemoradiation in orthotopic models. Additionally, using image-guided biopsies from treatment-naïve human GBM, we conducted spatial transcriptomic analyses. This revealed rare THY1+ regions characterized by mesenchymal/stem-like gene expression, analogous to our recurrent mouse model samples, which co-localized with macrophages within the perivascular niche. Since TGF β signaling contributes to a mesenchymal/stem-like phenotype, we inhibited TGF β RI activity in vivo which resulted in decreased mesenchymal/stem-like protein levels, including THY1, and restored sensitivity to TMZ/IR in recurrent tumors.

Conclusion

These findings reveal that GBM recurrence may result from tumor repopulation by pre-existing, therapy-resistant, THY1-positive, mesenchymal/stem-like cells within the perivascular niche. Furthermore, our data demonstrate the promise of targeting upregulated pathways in resistant subclones as a novel mechanism to achieve therapeutic response, and specifically that THY1 expression may represent a biomarker of response to TGF β inhibition.

9:35 – 9:45 A Phase 0/2 Clinical Trial of PARP Inhibition plus Radiotherapy in Newly-Diagnosed and Recurrent Glioblastoma

Nader Sanai, MD; Yu-Wei Chang; Tigran Margaryan; Jocelyn Harmon; John E. Wanebo, MD; Igor Barani; Wonsuk Yoo; Artak Tovmasyan; An-Chi Tien; Shwetal Mehta

Introduction

Poly (ADP-ribose) polymerase (PARP) mediates DNA damage response and a Phase 1 study of the PARP inhibitor, olaparib, suggests it is a brain-penetrant radiosensitizer in glioblastoma (GBM).

Objectives

We evaluated tumor pharmacokinetics (PK), tumor pharmacodynamics (PD), and clinical efficacy of three first-generation PARP inhibitors (niraparib, pamiparib, and olaparib) in newly-diagnosed (nGBM) and recurrent GBM (rGBM) patients.

Methods

Presumed nGBM patients received 4 days of niraparib (300 mg QD) or pamiparib (60 mg BID) and rGBM patients received 4 days of pamiparib (60mg BID) or olaparib (200 mg BID) prior to tumor resection. In all cases, enhancing and nonenhancing tumor, CSF, and plasma were collected. Total and unbound drug concentrations were measured using validated LC-MS/MS methods. PARP inhibition was assessed via 10 Gy ex vivo tumor irradiation and quantification of PAR compared to control. Patients with nonenhancing tumor tissue exceeding the PK threshold (unbound drug > 5-fold IC50) were continued to therapeutic dosing plus radiotherapy.

Results

In nonenhancing regions of nGBM, mean unbound concentrations of niraparib (n=20) and pamiparib (n=20) were 18-fold IC50 and 26-fold IC50, respectively. In nonenhancing regions of rGBM, mean unbound concentrations of pamiparib (n=14) and olaparib (n=4) were 25-fold IC50 and 2-fold IC50, respectively. 100% of niraparib/pamiparib patients, compared to 25% of olaparib, exceeded PK criteria. PAR suppression was observed in 69%, 61%, and 25% of niraparib, pamiparib, and olaparib patients, respectively.

Conclusion

Niraparib and pamiparib achieved pharmacologically-relevant concentrations in GBM and suppressed ex vivo PAR induction post-radiation. Olaparib's PK/PD profile does not support its continued development.

9:45 – 9:55	Blocking a noncoding RNA overcomes single immune checkpoint inhibition's failure to improve GBM immunotherapy
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E. Antonio Chiocca, MD PhD; Shkha Saini; Genaro Villa, MD, PhD; Marco Mineo

Introduction

We have recently completed and published a phase 1 surgical trial of interleukin 12 (IL12) immunogene therapy in recurrent GBM (Chiocca et al, Science TM, 2019) where tumor evasion was observed to be possibly due to PD-1/ PD-L1 immune checkpoint. However, subsequent surgical phase 1 and 2 trials failed to show that PD-1 inhibition improved IL12 immunogene therapy (Chiocca et al., Neuro-oncology, 2021). One reason for this failure is that GBM evasion from immunotherapy involves more than up-regulation of PD1 immune checkpoint signaling. We have discovered a novel long noncoding RNA (INCR1) that functions upstream of several immune checkpoint signals (Mineo et al, Mol. Cell, 2020) and showed that INCR1 inhibition with an antisense oligonucleotide (ASO) against INCR1 leads to improved IL12 immunotherapy in models of human GBM.

Objectives

We thus hypothesize that this INCR1 ASO may be a more efficacious means to improve IL12 and other immunotherapy approaches in GBM.

Methods

Transcriptomic, immunologic, and GBM models were employed for methods.

Results

We find that the INCR1 ASO leads to more T cell activation and cytotoxicity against GBM than PD-1 immune checkpoint signaling. The mechanism for this superiority is that INCR1 inhibition leads to down-regulation of multiple immune checkpoint signals, including PD-L1, IDO, CSF-1, TGFbeta and others.

Analysis of human tumors from the phase 1 clinical trial shows that there was up-regulation of INCR1 in tumors that escaped IL12 immunogene therapy, providing a clinical rationale for using INCR1 inhibition via the ASO to improve immunotherapy.

Conclusion

Based on these findings, we are now preparing an investigational new drug (IND) application to the FDA to perform a novel phase 1 trial combining surgical IL12 immunogene therapy with INCR1 ASO delivered via an Ommaya in patients with recurrent GBM.

9:55 – 10:05 Resting State fMRI Accurately Predicts Survival Outcomes in Glioblastoma Multiforme Patients

Eric C. Leuthardt, MD; KiYun Park; Bidhan Lamichhane; Michael O Olufawo; John Lee; Peter H Yang, MD; Albert H. Kim, MD, PhD; Joshua Shimony; Patrick Lockett

Introduction

Glioblastoma multiforme (GBM) is the most common brain malignancy in adults, and has a poor overall survival. Techniques capable of predicting survival outcomes could lead to improved clinical decision making.

Objectives

To use machine learning models to predict survival using clinical and imaging data in GBM patients.

Methods

Cross sectional clinical and neuroimaging (volumetric and resting state functional MRI, rsfMRI) data were acquired in 141 GBM patients (Table 1). Random forest models were used to classify length of survival (<1, 1-2, 2-3, >3 years) using clinical and neuroimaging features. Estimates of feature importance were calculated using out-of-bag predictor permutations. All models were optimized with Bayesian optimization, and validated with 10 fold cross validation. Model results were further evaluated in the context of extent of resection and genetic mutations.

Results

The random forest model was able to classify survival with 98% accuracy. The strongest predictive features in the model were resting state network correlations involving subcortical (thalamus and basal ganglia) regions. When evaluating genetic features, IDH1 mutation and MGMT methylation showed significant differences based on classification results, with longer survival associated with these features. Participants who received gross total resections also had significantly higher rates of long-term survival.

Conclusion

Techniques capable of predicting survival outcomes in GBM patients could lead to improved pre-surgical planning and post-surgical care. Our findings suggest machine learning is capable of highly accurate survival predictions based predominantly on rsfMRI network correlations.

10:05 – 10:10 Wrap-up/ Transition

10:10 – 10:55 Peer Reviewed Abstract Session X: Pediatrics

Moderators: Gerald Grant and Richard Ellenbogen

10:10 – 10:20 Multi-omics analysis elucidates the genetic basis of hydrocephalus

Andrew T. Hale, MD, PhD; Lisa Bastarache; Diego Morales; John C. Wellons, MD; Steven J. Schiff, MD, PhD; David Delmar Limbrick, MD, PhD; Eric R. Gamazon, PhD

Introduction

The genetic basis of hydrocephalus remains largely unknown. Using whole-genome genetic information, genetic analysis linked to neuroimaging phenotypes, proteomic data from CSF of hydrocephalic infants, and single-cell sequencing of neonatal brains across timescales, we perform the largest genetic study of hydrocephalus to date (Hale et al., Cell Reports, 2021).

Objectives

To define the genetic basis of hydrocephalus.

Methods

We perform a transcriptome-wide association study to identify genes associated hydrocephalus and brain-structural phenotypes derived from neuroimaging studies. We perform rare-variant exome analysis, unbiased proteomic analysis by mass spectrometry in CSF isolated from infants undergoing permanent CSF diversion, and single-cell analysis of the neonatal brain across timescales from the Allen Brain Atlas to validate our findings.

Results

We identify MAEL (a regulator of DNA transposons and methylation), as a transcriptome-wide predictor of hydrocephalus. Genetic analysis of neuroimaging phenotypes in the UK Biobank revealed that MAEL expression in the cortex is among the top genes regulating white matter and total brain volumes. Across the top differentially expressed genes associated with hydrocephalus in brain, we observed a significant enrichment for genes regulating white matter and total brain, but not CSF, volume. Additional support for MAEL is provided through rare exome variant analysis. We compare protein expression in CSF from hydrocephalic infants to differentiate disease-induced vs. disease-causing genetic mechanisms, with implications for future development of hydrocephalus biomarkers. Finally, we analyze single-cell RNA sequencing data to understand the temporal, spatial, and evolutionary origin of MAEL expression in the neonatal brain.

Conclusion

Our findings provide convergent evidence underscoring the importance of genetic mechanisms broadly underlying hydrocephalus.

10:20 – 10:30 Posterior Fossa Decompression with or without Duraplasty for Chiari Type I Malformation with Syringomyelia
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David Delmar Limbrick, MD, PhD

Introduction

Children with Chiari type I malformation (CM) and syringomyelia (SM) may suffer intractable headaches, neuropathic pain, sensorimotor deficits, and spinal deformity with lifelong disability. While neurosurgical treatment may reverse the progression of CM+SM, the best surgical approach [posterior fossa decompression with (PFDD) or without (PFD) duraplasty] is unknown.

Objectives

Our objective was to conduct a multicenter cluster randomized controlled trial of CM+SM with the following Specific Aims: SA1: Determine if PFD is associated with fewer surgical complications and less harm to patients than PFDD. SA2: Determine if PFD provides non-inferior clinical improvement and syrinx regression compared to PFDD. SA3: Compare treatment durability (surgical revision rate) between PFD and PFDD.

Methods

We conducted a multicenter cluster randomized controlled trial to compare posterior fossa decompression without (PFD) or with intradural microdissection and duraplasty (PFDD). Individuals ≤ 21 years with ≥ 5 mm cerebellar tonsillar ectopia and SM 3-9 mm in diameter were enrolled at 38 centers of the Park-Reeves

Syringomyelia Research Consortium. Centers were cluster randomized such that all enrollees at each center underwent the same intervention. Outcomes included surgical complications ≤ 6 months post-operatively (primary outcome) and clinical improvement (non-inferiority analysis), syrinx regression, and revision decompression at 12 ± 2 months post-operatively.

Results

162 participants were randomized (84 PFD, 78 PFDD) with an average cluster size of 4.26/site. Age at surgery was 10.34 ± 5.48 years, tonsillar ectopia was 13.80 ± 5.00 mm, and syrinx diameter was 5.66 ± 2.10 mm. Per Treatment analysis demonstrated no difference in odds of surgical complications for PFDD:PFD [1.29 (0.48-3.44; $p=0.62$)]. PFD was non-inferior to PFDD in clinical improvement [0.58 (1.22, 95% 1-sided upper CI limit)]. Syrinx regression was superior following PFDD [3.04 ± 2.47 mm versus 1.07 ± 1.75 mm, $p < 0.0001$]. Treatment durability was lower for PFD, which had a higher rate of revision decompression (log-rank $p=0.045$).

Conclusion

Compared with PFD, PFDD was more effective in treating SM with better treatment durability with no increase in surgical risk. PFD was non-inferior to PFDD in symptomatic clinical improvement.

10:30 – 10:40 Systems-level elucidation of the pathogenesis of cerebral arachnoid cysts

Kristopher Kahle, MD, PhD

Introduction

Arachnoid cysts (ACs), the most common space-occupying intracranial lesion in humans, are leptomeningeal-lined, cerebrospinal fluid-filled sacs that interdigitate between the major sulcal folds of the developing brain. Gaps in our understanding of AC pathogenesis impede the development of improved diagnostic, prognostic, and therapeutic measures for patients.

Objectives

Here, we sought to elucidate the cellular and molecular pathogenesis of pediatric cerebral ACs, and devise a new AC classification system with prognostic value. We hypothesized that: (i) multiple novel AC genes harboring de novo variants (DNVs) will be discovered using trio-based exome sequencing; (ii) AC genes will spatiotemporally converge in co-expression modules, cell types, and biological pathways pertinent to the regulation of fetal brain and meningeal development; and (iii) the systematic comparison of phenotypic data from individual AC cases will assist gene discovery by clustering cases with similar endophenotypes, thereby defining clinically-relevant disease subclasses.

Methods

We performed an integrated systems-level analysis of exome sequencing data from 617 proband-parent trios (1,851 individuals), single-cell RNAseq data of 152,898 cells of the developing brain and meninges, and phenomic data from artificial intelligence-mined patient medical records.

Results

We identified marked enrichment of damaging de novo variants (DNVs) in genes highly intolerant to loss-of-function variation ($pLI \geq 0.9$) in AC cases but not controls ($P = 1.57 \times 10^{-33}$). Seven genes, each a critical regulator of gene transcription in the developing brain and implicated in an OMIM disease, harbored an exome-wide significant burden of protein-damaging or -altering DNVs. Two of these had recurrent DNVs at identical amino acid residues. 21 other high- pLI genes had ≥ 2 two damaging DNVs. In all, damaging DNVs accounted for $\sim 20\%$ of AC cases. AC risk genes are enriched for chromatin modifiers, including three interacting components of the neural-specific ATP-dependent BAF (SWI/SNF) chromatin remodeling complex, and multiple regulators of histone-3 lysine-4 (H3K4) methylation. AC genes converge in co-expression modules, cell types, and pathways in the midgestational brain pertinent to the function of neural networks and the integrity of the arachnoid membrane. Unsupervised clustering of phenotype data identified four clinical AC subtypes that correlated with genomic results.

Conclusion

These findings provide novel insight into the genetic coordination of human cortical and leptomeningeal development and implicate epigenomic dysregulation due to germline DNV in AC pathogenesis. In the appropriate clinical context, some ACs may be considered radiographic harbingers of neurodevelopmental pathology warranting genetic follow-up and early referral for speech, neurobehavioral, and physical therapies.

10:40 – 10:50 Intraoperative C5 Stimulation during Obstetrical Brachial Plexus Injury Surgery Improves Functional Outcomes in Children

P. David Adelson, MD; Javier Figueroa; Randa Jarrar; Jorge Arango

Introduction

Obstetrical brachial plexus injury (OBPI) occurs in 1/1000 live births. The vast majority will have spontaneous recovery of function but surgical intervention with neurolysis/neurectomy of neuroma, primary neurotization, and nerve grafting results in good outcomes when there is limited return of functionality.

Objective

Short term intraoperative neurostimulation (IONS) in animal and limited adult human studies have resulted in improved functional outcome but there have no studies of IONS in children undergoing OBPI surgery.

Methods

In a retrospective analysis of patients with OBPI undergoing primary repair, C5 was stimulated at 2 milliamps, 20 Hz, for 60 minutes concurrent with standard surgical management and compared to previously treated cohort without stimulation at least 1 year postoperatively. Outcomes were assessed using intraoperative motor evoked potentials (MEP), British Medical Research Council (BMRC) and Mallet scale functional assessments.

Results

Fifty-one patients were included; IONS 27 patients; no IONS 24 patients. There were no differences in demographic, preoperative neurologic status, or operative technique. Both surgical groups demonstrated significant improvements in their intraoperative MEP and their functional assessments at 1-year postoperatively ($p \leq 0.02$). There were no differences in outcomes between groups except those who underwent IONS of C5 had a significantly greater improvement in shoulder external rotation on Mallet functional assessment ($p = 0.023$).

Conclusion

Primary nerve repair including neuroplasty and neurorraphy in pediatric OBPI improves neurophysiological, strength and complex functional outcome. C5 IONS resulted in improvement in external rotation. Further study though is necessary to optimize parameters for IONS and potential stimulation of other nerve roots to improve outcome.

10:50 – 10:55 Wrap-up/ Transition

10:55 – 11:10 Break

11:10 – 12:15 Peer Reviewed Abstract Session XI: Clinical Science **Moderators: Alexandra Golby**

11:10 – 11:20 Vagus Nerve Stimulation to Mitigate Inflammation After Subarachnoid Hemorrhage: A Pilot Randomized Control Trial

Anna Huguenard, MD; Gabbie Johnson; Gansheng Tan; Markus Adameck; Andrew Coxon; Gregory J. Zipfel, MD; Peter Brunner; Eric C. Leuthardt, MD

Introduction

Inflammation plays an important role in morbidity following subarachnoid hemorrhage (SAH). Vagus nerve stimulation (VNS) is a non-pharmacologic approach to immunomodulation, and a potential target for post-SAH intervention.

Objectives

To assess safety and feasibility of transauricular branch VNS (taVNS) in modulating the deleterious inflammatory response following SAH.

Methods

Preliminary data is reported from a prospective, triple-blinded, randomized controlled trial. Patients with SAH were randomized to taVNS or Sham stimulation. Blood and cerebrospinal fluid (CSF) were collected every three days to quantify inflammatory markers. Rates of cerebral vasospasm and hydrocephalus were assessed, and functional outcomes via modified Rankin Scale (mRS) scores were collected.

Results

12 SAH patients were randomized, with 6 in each arm. No adverse events related to the intervention were encountered. Levels of TNF- α both in plasma and CSF were significantly lower in the taVNS group by day 10 ($p=0.02$ for both). Radiographic vasospasm was observed in 33.3% and 100% of patients in taVNS and Sham arms, respectively ($p=0.06$). Permanent CSF diversion was required in 0% and 33.3% of patients in taVNS and sham arms, respectively ($p=0.46$). The average change in mRS between admission and first follow-up was -1.7 for taVNS and -0.3 for sham patients ($p=0.12$). Hospital-acquired infections were diagnosed in 16.7% and 66.7% of patients in taVNS and Sham arms, respectively ($p=0.24$).

Conclusion

taVNS is a non-invasive, non-pharmacologic method of neuro- and systemic immunomodulation. Preliminary data from this ongoing trial supports that taVNS following SAH can mitigate the inflammatory response, and potentially improve outcomes.

11:20 – 11:30 Transgenerational inheritance of a folate-driven axon regeneration trait is mediated by sperm DNA methylation

Bermans J Iskandar, MD; Joyce Koueik; Andy Madrid; Roy Chebel; Nithya Hariharan, MD; Ligia Papale; Reid Alisch

Introduction

Discovery of the molecular events underpinning epigenetic gene regulatory mechanisms has recently revealed how inherited conditions may be triggered by environmental stimuli without modifications in genomic sequence. While such inheritance is well established in human epidemiologic studies, and experimentally in plants, drosophila, and nematodes, mammalian investigations that show transgenerational inheritance of a non-genetic trait (i.e., beyond the F2 generation) are limited. We've shown that administration of folic acid and related methyl donors enhances post-injury axon regeneration in peripheral nerve grafts transplanted into the spinal cord in vivo, and in cultured neurons in vitro, and that the responses are mediated by DNA methylation.

Objectives

Here, we will extend on previously reported data indicating that the effect of folic acid is inherited by several consecutive generations of progeny, and show new data indicating that the phenotype and corresponding DMRs are transmitted through the gametes.

Methods

Mouse lineages were initiated in which F0 animals were treated with either folic acid or DDI control, producing 3 generations of untreated progeny (F1-F3). Sperm and oocytes from F3 generation mice were co-incubated and transferred into pseudopregnant naïve females to recover live IVF-generated F4 offspring. At adulthood, F1 to F3 mice as well as IVF-generated F4 offsprings were phenotyped for enhanced post-injury spinal cord axonal regeneration in vivo. In parallel, DNA methylation patterns and levels spanning the entire mouse methylome (i.e., >25 million sites) in F0-F3 and IVF-generated F4 animals' sperm were examined.

Results

The beneficial effects of methyl supplementation on post-injury axon regeneration are not confined to the treated F0 ancestor, but are transmitted to untreated F1-F4 progeny in parallel with alterations in DNA methylation and RNA transcription in spinal cord tissue, including at least 70 genes known to participate in axon regeneration. IVF-generated F4 animals from a lineage of mice in which F0 was treated with folic acid (vs. DDI control) have enhanced ability to regrow sensory spinal axons into a grafted segment of peripheral nerve in vivo ($p < 0.001$) in parallel with DNA methylation alterations in sperm.

Conclusion

Ancestral methyl donor-induced modifications of germ cell DNA methylation levels are sufficient to sustain the inheritance of augmented axonal regeneration after injury. This confirms that the heritable regenerative potential of tissues is dynamic over the lifespan rather than fixed at conception, and is responsive to ancestral environmental conditions and stimuli.

11:30 – 11:40 Replicating retroviral delivery of an IL-15 superagonist improves survival and lymphocyte infiltration in glioblastoma

Alexander F Haddad, MD; Jordan Spatz, PhD; Megan Montoya; Sara Collins; Sabraj Gill; Elaina Wang; Pavlina Chuntova; Jacob Young, MD; Noriyuki Kasahara; **Manish Kumar Aghi, MD, PhD**

Introduction

While glioblastoma has an immunosuppressed microenvironment, systemic immunotherapies have had limited success.

Objectives

We evaluated the efficacy of RLI, a superagonist of T-cell activator IL-15, delivered to tumor cells using a tumor-selective retroviral replicating vector (RRV).

Methods

RRV-RLI was studied in murine SB28 and Tu2449 glioblastoma models, which are engineered to be poorly immunogenic with low-mutational burden and known immunotherapy resistance, and hence more biomimetic models of human GBM.

Results

RRV-RLI replicated in cultured SB28/Tu2449 cells with robust production of functional RLI (165.4±5.3 ng/mL). Stereotactic injection of RRV-RLI into pre-established intracerebral SB28 tumors significantly reduced tumor growth on bioluminescence imaging, and increased median survival compared to mice receiving RRV (55 vs. 19 days, $p = 0.002$), leading to long-term survival in 12% of treated mice. In the Tu2449 model, imaging showed complete eradication of intracerebral tumors after RRV-RLI treatment, with long term survival in >85% of treated mice, compared to 12.5 day median survival in mice receiving RRV ($p = 0.001$). RRV-RLI treated tumors showed increased CD8 T-cell infiltration, without altering immunosuppressive cell populations. Anti-tumor inflammatory changes, including increased expression of T-cell activation and killing genes, were observed in the NanoString nCounter platform using a 770-gene panel representing different immune cell types. RLI was not detected in the blood of treated mice and intratumoral RRV-RLI caused no adverse systemic immune effects.

Conclusion

RRV-RLI immunotherapy caused immunostimulatory and pro-inflammatory changes to the glioblastoma microenvironment and prolonged survival in two poorly-immunogenic syngeneic murine models of GBM. This tumor-localized immunomodulatory gene therapy could safely reverse the T-cell depleted immunophenotype of GBM.

11:40 – 11:50 Cellular localization of protoporphyrin IX in glioblastoma

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Introduction

Fluorescence guidance is widely utilized to improve the precision of cancer surgery. 5-aminolevulinic acid, the most widely used fluorophore in glioma surgery, is thought to cause selective accumulation of fluorescent protoporphyrin IX (PpIX) in tumor cells. 5-aminolevulinic acid is indeed highly specific for densely tumor-infiltrated tissue but less effective for visualizing the tumor periphery.

Objective

To develop and validate an imaging system to improve detection of PpIX in the operating room, using paired stimulated Raman histology and two-photon excitation fluorescence microscopy.

Methods

Using the first paired stimulated Raman histology/two-photon fluorescence microscope, we imaged 175 fresh tumor specimens from 75 high-grade glioma patients at three institutions. Paired stimulated Raman histology and two-photon fluorescence images were quantitatively analyzed for cellularity and fluorescence. Conventional histology and immunohistochemistry (GFAP, CD163 and SMA) was performed on specimens after fresh tissue imaging.

Results

Here, we identify and define five distinct microscopic patterns of PPIX accumulation in glioblastoma. There was no correlation between the degree of tumor cellularity and the concentration of PpIX across all imaged specimens ($R=-0.21$). We further demonstrate that intracellular PpIX accumulation occurs most prominently in histiocytic, rather than neoplastic, appearing cells, and that the abundance of cells concentrating PpIX and CD163 positive cells is directly correlated ($p<0.02$).

Conclusion

Our findings encourage reconsideration of the existing theory of 5-ALA-induced glioma cell fluorescence and demonstrate how 5-ALA imaging can provide a window into the immune microenvironment of human gliomas.

11:50 – 12:00 Sustained ICP elevation in the setting of intraventricular hemorrhage leads to synaptic engulfment by microglia

Chloe Puglisi; Bradley Ander; Catherine Peterson, MD; Janet Ann Keiter; Heather Hull; Cameron Hawk; Venina Kalistratova; Ali Izadi, BS; Gene G Gurkoff, PhD; Frank Sharp; **Ben Waldau, MD**

Introduction

Intraventricular hemorrhage (IVH) in the setting of a ruptured brain aneurysm or hypertensive bleed is associated with long-term memory loss in survivors. Several mechanisms of memory decline after intraventricular hemorrhage have been investigated in animal models including but not limited to

hydrocephalus, neuro-inflammation, oxidative stress and iron toxicity. However, there has been a paucity of studies examining the influence of elevated intracranial pressure (ICP) on long-term memory decline after intraventricular hemorrhage. I have developed an intraventricular hemorrhage model with ICP recordings in which rodents develop a long-term spatial memory deficit if there is a sustained ICP elevation to 50 mm Hg for 2 hours.

Objectives

The objective is to understand the mechanism of long-term memory deficits after IVH+ elevated IC.

Methods

Four groups of rodents were compared and analyzed with RNAseq: IVH + elevated ICP (IVH+ICP), IVH, volume control and sham control. Animals underwent Morris water maze 2 weeks after the injury. Animals were then euthanized, hippocampi removed and analyzed with RNAseq. Taking each list of DEGs and running them through ontological databases using the WebGestalt interface, functional pathways and processes related to these gene sets were identified with overrepresentation in the curated gene sets above the cutoff of $FDR < 0.05$ ($FDR = \text{Benjamini Hochberg False Discovery Rate for multiple comparisons}$). We also analyzed microglial activation with regards to fractal dimensions and lacunarity. Finally, we investigated classical complement activation and microglial engulfment of synapses in the dentate gyrus.

Results

There was no group effect on swim speed during the probe trial ($F(3, 40)=0.6285$, $p=0.6009$). In the probe trial, we found a main effect of the group on latency to reach the platform ($F(3, 42)=3.976$, $p=0.0140$). Additionally, one-way ANOVA revealed a significant effect of group when evaluating time spent in the 10% ring ($F(3, 42)=3.467$, $p=0.0244$) and 5% ring ($F(3, 42)=4.765$, $p=0.0060$) during the probe trial. A post hoc Dunnett's analysis revealed that the IVH+ICP group spent significantly less time in the 10% ring than the IVH ($p=0.0216$) and the volume control groups ($p=0.0240$), and the IVH+ICP group spent significantly less time in the 5% ring than the IVH group ($p=0.0016$).

RNAseq analysis showed that members of the C1 complex C1qa (1.4-fold, $p=0.04$), C1qb (1.5-fold, $p=0.01$), as well as C1r (1.8-fold, $p=0.04$) were significantly increased in the IVH+ICP group compared to the IVH group. C1qc was increased 1.4-fold, but just outside the significance cutoff ($p=0.051$). C3, C2, and C4a expression were increased 3.5-fold ($p=0.001$), 2.1-fold ($p=0.0009$), and 2.2-fold ($p=0.04$), respectively, in the IVH+ICP hippocampus compared to IVH.

Fractal analysis showed the strongest microglial activation with IVH+ICP compared to the other groups. Immunohistological analysis showed colocalization of C1q with synaptophysin and engulfment of synapses by microglia in the dentate gyrus in the IVH+ICP group.

Conclusion

Only introduction of ICP into the IVH model led to spatial memory deficits, microglial activation and enrichment of the classical complement cascade signaling pathway. Therefore, the mechanism of long-term memory decline after IVH may be due to ICP-induced microglial activation leading to aberrant synaptic engulfment and elimination.

12:00 – 12:10 Prediction calculator for LOS, readmission, and reoperation in patients with intramedullary spinal cord tumors

Daniel Sciubba, MD; Andrew Hersh

Introduction

Intramedullary spinal cord tumors (IMSCTs) are rare tumors associated with significant morbidity and mortality. Surgical resection is often indicated for symptomatic lesions but may result in new neurological deficits and decrease quality of life. Identifying predictors of these adverse outcomes may help target interventions designed to reduce their occurrence. Nonetheless, most prior studies have employed population-level datasets with limited granularity.

Objectives

To determine independent predictors of nonroutine discharge, prolonged length of stay (LOS), and 30 day readmission and reoperation, and to deploy these results as a web-based calculator.

Methods

Retrospective cohort study
PATIENT SAMPLE: A total of 235 patients who underwent resection of IMSCTs at a single comprehensive cancer center. Nonroutine discharge, prolonged LOS, 30 day readmission, and 30 day reoperation
METHODS: Patients who underwent surgery from June 2002 to May 2020 at a single tertiary center were included. Data was collected on patient demographics, clinical presentation, tumor histology, surgical procedures, and 30 day readmission and reoperation. Functional status was assessed using the Modified McCormick Scale (MMS) and queried preoperative neurological symptoms included weakness, urinary and bowel dysfunction, numbness, and back and radicular pain. Variables significant on univariable analysis at the $\alpha \leq 0.15$ level were entered into a stepwise multivariable logistic regression model.

Results

Of 235 included cases, 131 (56%) experienced a nonhome discharge and 68 (29%) experienced a prolonged LOS. Of 178 patients with ≥ 30 days of follow-up, 17 (9.6%) were readmitted within 30 days and 13 (7.4%) underwent reoperation. Wound dehiscence (29%) was the most common reason for readmission. Nonhome discharge was independently predicted by older age (OR=1.03/year; $p < .01$), thoracic location of the tumor (OR=2.36; $p = .01$), presenting with bowel dysfunction (OR=4.09; $p = .03$), and longer incision length (OR=1.44 per level; $p = .03$). Independent predictors of prolonged LOS included presenting with urinary incontinence (OR=2.65; $p = .05$) or a higher preoperative white blood cell count (OR=1.08 per $10^3/\mu\text{L}$; $p = .01$), while GTR predicted shorter LOS (OR=0.40; $p = .02$). Independent predictive factors for 30 day unplanned readmission included experiencing ≥ 1 complications during the first hospitalization (OR=6.13; $p < .01$) and having a poor (A-C) versus good (D-E) baseline neurological status on the ASIA impairment scale (OR=0.23; $p = .03$). The only independent predictor of unplanned 30 day reoperation was experiencing ≥ 1 inpatient complications during the index hospitalization (OR=6.92; $p < .01$). Receiver operating curves for the constructed models produced C-statistics of 0.67-0.77 and the models were deployed as freely available web-based calculators (<https://jhuspine5.shinyapps.io/Intramedullary30day>).

Conclusion

We found that neurological presentation, patient demographics, and incision length were important predictors of adverse perioperative outcomes in patients with IMSCTs. The calculators can be used by clinicians for risk stratification, preoperative counseling, and targeted interventions.

12:10 - 12:15 **Wrap-up/ Transition**

12:15 **Closing Remarks & Meeting Adjourn**



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EBEN ALEXANDER, JR.	1950	2004
JOAO (JOHN) L. 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
GILLES P. BERTRAND	1967	2019
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
JERALD S. BRODKEY	1977	2014
HOWARD BROWN	1939	1990
KARLAUGUST BUSHE	1972	1999
FERNANDO CABIESES	1966	2009

LUC CALLIAUW	1988	2021
JUAN Y. 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 S. CONNOLLY	1972	2014
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 L. 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 G. FISHER	1955	2003
ELDON L. FOLTZ	1960	2013
RICHARD A. R. FRASER	1976	2017
JOHN FRENCH	1951	1989
LYLE A. FRENCH	1954	2004

JAMES GALBRAITH	1947	1997
HENRY GARRETSON	1973	2007
F. JOHN GILLINGHAM	1962	2020
SIDNEY GOLDRING	1964	2004
PHILIP GORDY	1968	2014
EVERETT G. GRANTHAM	1942	1997
JOHN WILLIS GREEN	1953	1990
JAMES GREENWOOD, JR.	1952	1992
ROBERT G. GROSSMAN	1984	2021
WESLEY A. GUSTAFSON	1942	1975
WALLACE B. HAMBY	1941	1999
HANNIBAL HAMLIN	1949	1982
JOHN WILLIAM HANBERY	1959	1996
JOHN HANKINSON	1973	2007
GRIFFITH R. HARSH, III	1980	2019
GEORGE HAYES	1962	2002
MARK PETER HEILBRUN	1984	2010
E. BRUCE HENDRICK	1968	2001
JESS D. HERRMANN	1938	1944
HENRY L. HEYL	1951	1975
JULIAN T. HOFF	1975	2007
HAROLD J. HOFFMAN	1982	2004
EDGAR M. HOUSEPIAN	1976	2014
WILLIAM E. HUNT	1970	1999
OLAN HYNDMAN	1942	1966
FABIAN ISMAT	1989	2019
SHOZO ISHII	1975	2012
KENNETH JAMIESON	1970	1976
JOHN A. JANE, SR.	1982	2015
PETER J. JANNETTA	1994	2016
SIR GEOFFREY JEFFERSON (Honorary)	1951	1961

HANS-PETER JENSEN	1980	2000
RICHARD JOHNSON	1974	1997
ELLIS B. KEENER	1978	2021
WILLIAM KEITH, Founder	1938	1987
GLENN W. KINDT	1977	2022
ROBERT B. 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	2014
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
STEPHEN MAHALEY	1972	1992
LEONARD MALIS	1973	2005
GEORGE MALTBY	1942	1988
FRANK MARGUTH	1978	1991
DONALD MATSON	1950	1969
ROBERT E. MAXWELL	1992	2022

FRANK MAYFIELD, Founder	1938	1991
AUGUSTUS McCRAVEY	1944	1989
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	1992
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 W. PORTER	1962	2021
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	1998
WILLIAM SCOVILLE	1944	1984
EDWARD L. SELJESKOG	1992	2022
R. EUSTACE SEMMES (Honorary)	1955	1982
C. HUNTER SHELDEN	1941	2003
FREDERICK A. SIMEONE	1981	2022
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

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