



THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

86<sup>TH</sup> ANNUAL MEETING

THE RITZ-CARLTON, HALF MOON BAY, CALIFORNIA

OCTOBER 16-19, 2024



Congress of  
Neurological  
Surgeons

Jointly Provided by the CNS

## FUTURE MEETINGS

October 22-25, 2025

The Hotel Grande Bretagne  
Athens, Greece

September 27-30, 2026

Ojai Valley Inn  
Ojai, CA

*Mark your calendars now!*

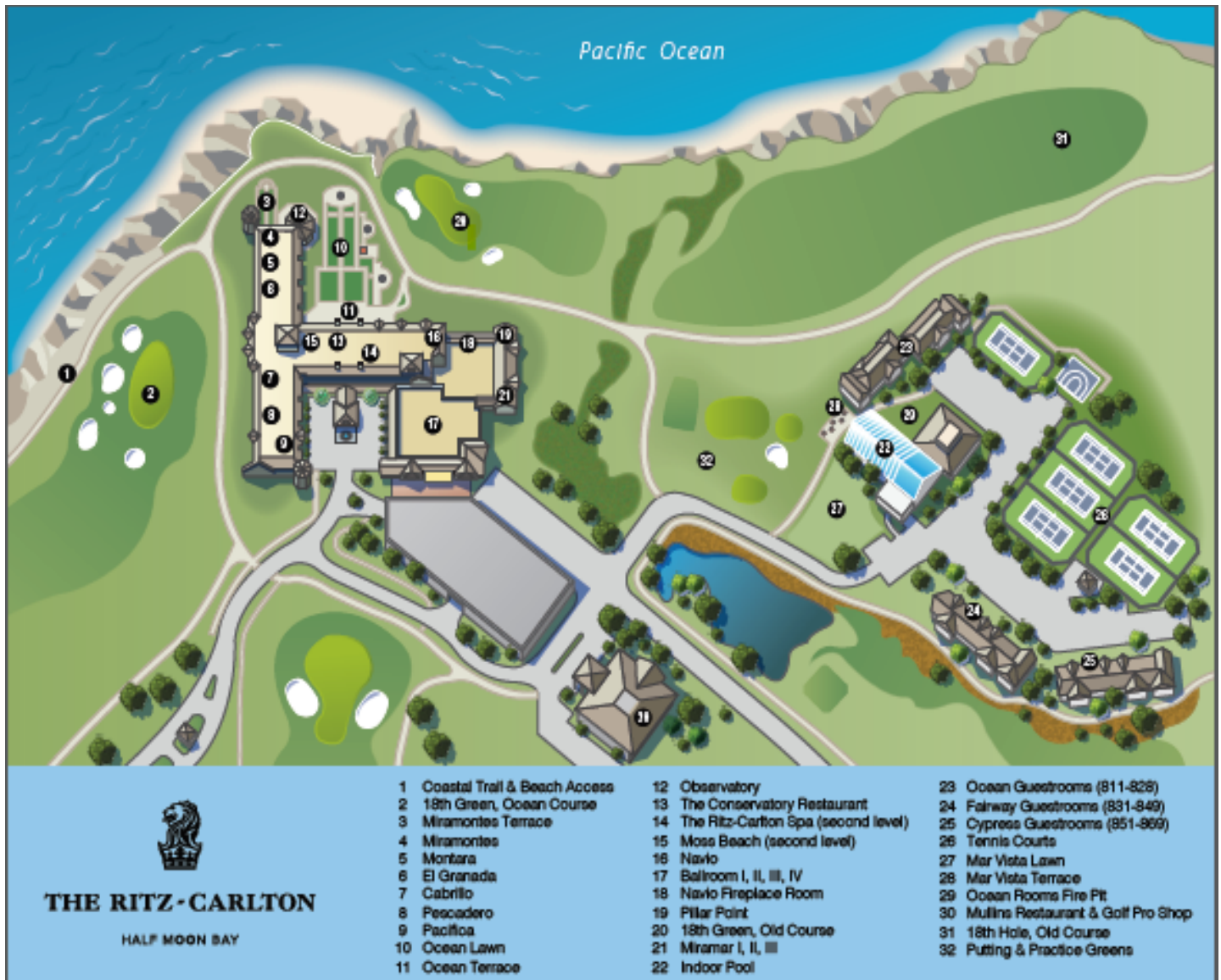
# GENERAL INFORMATION

## HOTEL INFORMATION

### THE RITZ-CARLTON

1 Miramontes Point Rd, Half Moon Bay, CA 94019

855-868-1373



REGISTRATION LOCATION:

[WWW.AMERICANACADEMYS.ORG](http://WWW.AMERICANACADEMYS.ORG)

REGISTRATION:

On-site registration is currently open.

Complete form on website. Email inquiries directly to [shellbey@voilameetings.com](mailto:shellbey@voilameetings.com)

*A special thanks to the following exhibitors supporting the*

**THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY  
86<sup>TH</sup> ANNUAL SCIENTIFIC MEETING**

*Please take time to visit with them during the break.*

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- Clearpoint Neuro
- Integra LifeSciences
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THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY  
86<sup>TH</sup> ANNUAL SCIENTIFIC MEETING

**WEDNESDAY, OCTOBER 16**

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1:00 – 5:30 pm	<b>Registration</b>	Location
2:00 – 4:30 pm	<b>Executive Committee Meeting</b>	Observatory
6:00 – 6:30 pm	<b>New Members Reception</b>	Ocean Lawn
6:30 – 8:30 pm	<b>Opening Reception</b>	Ocean Lawn

**THURSDAY, OCTOBER 17**

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6:30 am – 12:30 pm	<b>Registration</b>	Pre Function of Ritz-Carlton Ballroom
6:30 – 7:30 am	<b>Members Breakfast &amp; Business Meeting</b> (Voting Membership Only)	Miramar
7:00 – 9:00 am	<b>Guest &amp; Spouse/Companion Breakfast</b>	Miramontes Room & Terrace
7:30 – 7:35 am	<b>Welcoming Remarks</b>	Ritz-Carlton Ballroom
7:35 – 7:45 am	<b>Historian's Report</b>	Ritz-Carlton Ballroom
7:45 – 9:00 am	<b>Peer Reviewed Abstract Session I: Tumor 1</b>	Ritz-Carlton Ballroom
9:00 – 10:10 am	<b>Peer Reviewed Abstract Session II: Spine 1</b>	Ritz-Carlton Ballroom
10:10 – 10:30 am	Break	Pre Function of the Ritz-Carlton Ballroom
10:30 – 10:55 am	<b>Special Debate Session: Regulation of Innovation: How Much Is Enough?</b>	Ritz-Carlton Ballroom
10:55 – 12:00 pm	<b>Peer Reviewed Abstract Session III: Pediatrics, Trauma, and Other</b>	Ritz-Carlton Ballroom
12:05 – 12:50 pm	<b>Special Session: Presidential Address</b>	Ritz-Carlton Ballroom
1:30 – 4:30 pm	<b>Academy Emerging Investigators' Program</b>	Miramar
6:30 – 9:30 pm	<b>Reception</b>	Mar Vista Lawn

## FRIDAY, OCTOBER 18

6:30 am - 12:00 pm	<b>Registration</b>	Pre Function of Ritz-Carlton Ballroom
6:30 - 7:30 am	<b>Members Breakfast &amp; Business Meeting</b> (Voting Membership Only)	Miramar
7:00 - 9:00 am	<b>Guest &amp; Spouse/Companion Breakfast</b>	Miramontes Room & Terrace
7:30 - 7:35 am	<b>Welcoming Remarks</b>	Ritz-Carlton Ballroom
7:35 - 8:50 am	<b>Peer Reviewed Abstract Session IV:</b> Functional/Epilepsy	Ritz-Carlton Ballroom
8:50 - 10:00 am	<b>Peer Reviewed Abstract Session V:</b> Cerebrovascular	Ritz-Carlton Ballroom
10:00 - 10:20 am	Break	Pre Function of Ritz-Carlton Ballroom
10:20 - 11:00 am	<b>Special Session:</b> Guest Speaker	Ritz-Carlton Ballroom
11:10 - 12:45 pm	<b>Peer Reviewed Abstract Session VI:</b> Tumor	Ritz-Carlton Ballroom
1:30 - 4:30 pm	<b>Academy Emerging Investigators' Program</b>	Miramar
6:00 - 7:00 pm	<b>Cocktail Reception</b>	Mar Vista Lawn
7:00 - 10:00 pm	<b>Gala Dinner</b> (Black Tie Optional)	Ritz-Carlton Ballroom

## SATURDAY, OCTOBER 19

7:00 am - 12:00 pm	<b>Registration</b>	Pre Function of Ritz-Carlton Ballroom
7:00 - 9:00 am	<b>Members, Guests, &amp; Spouse/Companion Breakfast</b>	Miramar
7:30 - 8:20 am	<b>The Oldfield Session</b>	Ritz-Carlton Ballroom
8:20 - 9:30 am	<b>Peer Reviewed Abstract Session VII:</b> Spine and Other	Ritz-Carlton Ballroom
9:30 - 9:50 am	<b>Break</b>	Pre Function of Ritz-Carlton Ballroom
9:50 - 11:05 am	<b>Peer Reviewed Abstract Session VIII:</b> Cerebrovascular	Ritz-Carlton Ballroom
11:05 - 11:25 am	<b>Special Sessions:</b> Academy Award Presentation and Lecture	Ritz-Carlton Ballroom
11:25 am - 12:35 pm	<b>Peer Reviewed Abstract Session IX:</b> Functional and Epilepsy	Ritz-Carlton Ballroom
12:35 - 12:45 pm	<b>Closing Remarks &amp; Meeting Adjourn</b>	Ritz-Carlton Ballroom



# THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

## 2023 – 2024 OFFICERS

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### PRESIDENT

Shenandoah Robinson, MD

### PRESIDENT - ELECT

E. Sander Connolly Jr., MD

### VICE PRESIDENT

Anil Nanda, MD

### SECRETARY

Sepideh Amin-Hanjani, MD

### TREASURER

Russell Lonser, MD (2025)

### HISTORIAN

Michael Schulder, MD (2025)

### PAST PRESIDENT

Fred G. Barker II, MD

### EXECUTIVE COMMITTEE

Shenandoah Robinson, MD

Fred G. Barker II, MD

E. Sander Connolly Jr., MD

Sepideh Amin-Hanjani, MD

Russell Lonser, MD

Michael Schulder, MD

Anil Nanda, MD

Aviva Abosch, MD

## 2023 – 2024 COMMITTEES

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### ACADEMY AWARD COMMITTEE

Michael Vogelbaum, MD, PhD – Chair (2024)  
Praveen Mummaneni, MD (2025)  
Christopher Ogilvy, MD (2026)

### AUDITING COMMITTEE

Gerald Grant, MD – Chair (2024)  
Praveen Mummaneni, MD (2025)  
Shelly Timmons, MD (2026)

### BYLAWS COMMITTEE

Linda Liao, MD, PhD – Chair  
Fred G. Barker II, MD  
Shenandoah Robinson, MD  
E. Sander Connolly Jr., MD

### FUTURE SITES COMMITTEE

Howard Riina, MD (2026)

### MEMBERSHIP ADVISORY COMMITTEE

James M. Markert, MD, MPH – Chair  
Fred G. Barker II, MD  
E. Sander Connolly Jr., MD  
Shenandoah Robinson, MD  
Russell Lonser, MD  
Sepideh Amin-Hanjani, MD  
Linda Liao, MD, PhD  
Aviva Abosh, MD  
Zoher Ghogawala, MD

### SUBCOMMITTEE ON CORRESPONDING MEMBERSHIP

Jacques Morcos, MD – Chair (2024)  
Christopher Loftus, MD (2025)



NOMINATING COMMITTEE

Fred G. Barker II, MD - Chair (ex officio)  
Shenandoah Robinson, MD (ex officio)  
E. Sander Connolly Jr., MD (ex officio)

SCIENTIFIC PROGRAM COMMITTEE

Daniel Resnick, MD - Chair (2024)  
Zohar Ghogawala, MD (2025)  
Gerald Grant, MD (2026)  
Judy Huang, MD (2027)

COMMUNICATIONS & ROUND ROBIN COMMITTEE

QUARTERLY NEWSLETTER

Mark N. Hadley, MD  
Gerald Grant, MD

LOCAL ARRANGEMENTS

Michael Lim, MD - Chair (2024)

CNS JOINT SPONSORSHIP EDUCATION REPRESENTATIVE

Judy Huang, MD

WFNS DELEGATES

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

RESEARCH ADVISORY COMMITTEE

Gregory Zipfel, MD - Chair (2025)  
Mark Johnson, MD, PhD (2025)  
Sameer Sheth, MD, PhD (2025)  
Eric Leuthardt, MD (2026)  
Melanie Gephardt Hayden, MD (2026)  
Zohar Ghogawala, MD (2026)

## PAST-PRESIDENTS

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Dean H. Echols	1938 - 39	Sidney Goldring	1983
Spence Braden	1940	Russel H. Patterson, Jr.	1984
Joseph P. Evans	1941	Thomas Langfitt	1985
Francis Murphey	1942	Phanor L. Perot, Jr.	1986
Frank H. Mayfield	1943	Shelley N. Chou	1987
A. Earl Walker	1944	James T. Robertson	1988
Barnes Woodhall	1946	Thoralf M. Sundt, Jr.	1989
William S. Keith	1947	Robert Ojemann	1990
Howard A. Brown	1948	Nicholas Zervas	1991
John Raaf	1949	Henry Garretson	1992
E. Harry Botterell	1950	George Tindall	1993
Wallace B. Hamby	1951	William A. Buchheit	1994
Henry G. Schwartz	1952	David L. Kelly, Jr.	1995
J. Lawrence Pool	1953	John M. Tew, Jr.	1996
Rupert B. Raney	1954	Julian T. Hoff	1997
David L. Reeves	1955	Edward Connolly	1998
Stuart N. Rowe	1956	J. Charles Rich	1999
Arthur R. Elvidge	1957	George A. Ojemann	2000
Jess D. Herrmann	1958	Roberto C. Heros	2001
Edwin B. Boldrey	1959	Donald O. Quest	2002
George S. Baker	1960	David G. Piepgras	2003
C. Hunter Shelden	1961 - 62	Volker K.H. Sonntag	2004
Samuel R. Snodgrass	1963	Martin B. Camins	2005
Theodore Rasmussen	1964	L. Nelson Hopkins	2006
Edmund J. Morrissey	1965	Richard Morawetz	2007
George Maltby	1966	Robert F. Spetzler	2008
Guy L. Odom	1967	Ralph G. Dacey, Jr.	2009
James G. Galbraith	1968	Steven Giannotta	2010
Robert H. Pudenz	1969 - 70	Robert A. Solomon	2011
William B. Scoville	1971	James T. Rutka	2012
Robert L. McLaurin	1972	Griffith R. Harsh	2013
Lyle A. French	1973	Fredric B. Meyer	2014
Benjamin B. Whitcomb	1974	Mitchel S. Berger	2015
John R. Green	1975	Mark N. Hadley	2016
William H. Feindel	1976	William T. Couldwell	2017
William H. Sweet	1977	Daniel L. Barrow	2018
Arthur A. Ward	1978	E. Antonio Chiocca	2019
Robert B. King	1979	M. Sean Grady	2020
Eben Alexander, Jr.	1980	Douglas Kondziolka	2021
Joseph Ransohoff II	1981	James M. Markert	2022
Byron C. Pevehouse	1982	Fred Barker	2023

## PAST VICE-PRESIDENTS

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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	Daniel Yoshor	2022
Hugo V. Rizzoli	1983	Bob S. Carter	2023
James W. Correll	1984		
E. Bruce Hendrick	1985		

## PAST SECRETARY-TREASURERS

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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

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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. Piegras	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
E. Sander Connolly, Jr.	2021 - 2023

## PAST TREASURERS

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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
Shenandoah Robinson	2020 - 2022

## OLDFIELD AWARD

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Russell Lonser	2018
Amy Heimberger	2019
Fred G. Barker II	2021
Todd Hollon	2022
Kim Burchiel	2023

## MEETINGS OF THE ACADEMY

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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
The Broadmoor, Colorado Springs, Colorado	September 28 - October 1, 2022
The Cloister, Sea Island, Georgia	October 4 - October 7, 2023





## 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



Congress of  
Neurological  
Surgeons

## LEARNING OBJECTIVES

- Describe the inherent conflict between innovation and regulation as it pertains to the development of new technologies in neurosurgery.
- Discuss new cutting-edge technologies to improve resection margins in glial tumors.
- Identify opportunities for enhancing diversity and scientific exploration through emphasis on vision and perseverance.
- 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 Congress of Neurological Surgeons (CNS) and the American Academy of Neurological Surgery. The CNS is accredited by the ACCME to provide continuing medical education for physicians.

## DESIGNATION STATEMENT

The CNS designates this live activity for a maximum of 14.50 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.

The Congress of Neurological Surgeons controls the content and production of this CME activity and attempts to assure the presentation of balanced, objective information. In accordance with the Standards for Integrity and Independence in Accredited Continuing Education established by the Accreditation Council for Continuing Medical Education (ACCME), speakers are asked to disclose all relationships they have with ineligible companies\* over the previous 24 months, which may be related to the content of their lecture. Speakers who have disclosed a relationship with an ineligible company whose products may have relevance to their presentation will be listed for viewing prior to the event.

A list of financial disclosures relevant to the meeting will be posted prior to the meeting on the meeting's web page and app.

**Any planner, reviewer, or faculty member not on the disclosure list has reported they have nothing to disclose.**

*\*Ineligible companies are those whose primary business is producing, marketing, selling, re-selling, or distributing healthcare products used by or on patients. An ineligible company is not eligible for ACCME accreditation or participation in Joint Partnership.*

## INTENDED AUDIENCE/BACKGROUND REQUIREMENT

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

## CNS JOINT PROVIDERSHIP DISCLAIMER STATEMENT

The material presented at the 86th Annual Meeting of the American Academy of Neurological Surgery has been made available by the American Academy of Neurological Surgery and the Congress of Neurological Surgeons (CNS) 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 CNS, or its Committees, Commissions, or Affiliates.

Neither the CNS nor the American Academy of Neurological Surgery makes any statements, representations or warranties (whether written or oral) regarding the Food and Drug Administration (FDA) status of any product used or referred to in conjunction with any course, seminar or other presentation being made available as part of the 85th Annual Meeting of the American Academy of Neurological Surgery. Faculty members shall have sole responsibility to inform attendees of the FDA status of each product that is used in conjunction with any course, seminar or presentation and whether such use of the product is in compliance with FDA regulations.

## RELEVANT CONFLICT OF INTEREST DISCLOSURES

### CNS DISCLOSURE POLICY

The Congress of Neurological Surgeons controls the content and production of this CME activity and attempts to assure the presentation of balanced, objective information. In accordance with the Standards for Integrity and Independence in Accredited Continuing Education established by the Accreditation Council for Continuing Medical Education (ACCME), speakers are asked to disclose all relationships they have with ineligible companies\* over the previous 24 months which may be related to the content of their lecture. Speakers who have disclosed a relationship with an ineligible company whose products may have a relevance to their presentation are listed below.

**Any planner, reviewer, or faculty member not on the disclosure list has reported they have nothing to disclose.**

**All relevant financial relationships listed for these individuals have been mitigated.**

\***Ineligible companies** are those whose primary business is producing, marketing, selling, re-selling, or distributing healthcare products used by or on patients. An ineligible company is not eligible for ACCME accreditation or participation in Joint Providership.

### DISCLOSURE LISTING – SPEAKERS, PLANNERS AND EXECUTIVE COMMITTEE MEMBERS

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.

PLANNERS		
Individual's Name	Nature of Relationship(s)	Name(s) of Ineligible Company
Zoher Ghogawala	Receipt of IP/Patent	NidusAI
Daniel Resnick		Nothing to Disclose

FACULTY		
Individual's Name	Nature of Relationship(s)	Name(s) of Ineligible Company
Aviva Abosch	Consulting Fee, Contracted Research	Medtronic
Wael Asaad	Contracted Research	Functional Neuromodulation Inc. Enspire Inc.
Issam Awad	Consulting Fee	Neurelis, Ovid Rx
Chetan Bettgowda	Consulting Fee	Haystack Oncology Bionaut Labs Privo Technologies Depuy-Synthes
Nicholas Boulis	Consulting Fee	Trames, LifeEdit, UCB, Jupiter Fund, Kriya, UniQure
Samuel Browd	Own Stock	Proprio Balt, Medtronic, Siemens Healthineers, Microvention, Q'apel
Kevin Cockroft	Fees for Non-CME/CE Services	Intersocietal Accreditation Commision

James Elder	Consulting Fee	Medtronic, Icad
Benjamin Elder	Consulting Fee	Depuy Synthes, SI Bone, Iota Bioscience
	Contracted Research	SI Bone, Stryker
	Royalty	SI Bone
	Stock Options	Injectsense
Dario Englot	Consulting Fee	NeuroPace, Boston Scientific
Peter Fecci	Consulting Fee	Monteris Medical
Justin Fraser	Stock Options	Lets Get Proof, Stream Biomedical
	Consulting Fee	Penumbra, Medtronic
Melanie Hayden Gephart	Consulting Fee	Telix, Midatech/Biodexa, Robeaute
	Receipt of IP/Patent	SensoBrain SmartLens
	Contracted Research	Quadriga
Zoher Ghogawala	Receipt of IP/Patent	NidusAI
Constantinos (Costas) Hadjipanayis	Consulting Fee	Stryker corporation, Synaptive Medical Hemerion Therapeutics, Integra True Digital Surgery NICO Corp
Todd Hollon	Future Stock Options	Invenio Imaging, Inc.
Wajd Al-Holou	Consulting Fee	Servier
Peter Kan	Consulting Fee	Stryker International, Imperative Care
Alexander Khalessi	Consulting Fee	Medtronic
Albert Kim	Consulting Fee	Monteris Medical
	Contracted Research	Stryker
Vibhor Krishna	Consulting Fee	Medtronic Inc
Fredrick Lang	Receipt of IP/Patent	CV Bio
Michael Levitt	Own Stock	Proprio, Apertur, Hyperion Surgical, Synchron, Stroke Diagnostics
	Future Stock Option	Stereotaxis, Fluid Biomed
Linda Liau	Own Stock	ClearPoint Neuro, Inc.
	Consulting Fee	Northwest Biotherapeutics
Andre Machado	Receipt of IP/Patent	Enspire, Ceraxis
	Stock Options	Enspire
	Contracted Research	Abbott, Enspire
	Consulting Fee	Abbott
Ian McCutcheon	Fees for Non-CME/CE Services	Merck Inc.
Michael McDermott	Stock Options	Zap-X, Deinde Medical, Light Helmets
	Consulting Fee	Viseon, Endostream, RIST, Synchron, Perflow, Viz.ai, CVAid

J. Mocco	Stock Options	Investor-Imperative Care, Endostream, Echovate, Viseon, BlinkTBI, Serenity, NTI Managers, RIST, Viz.ai, Synchron, Songbird, Tulavi, Vastrax, Neuroolutions, Radical, E8, Brovo, Spinaker
	Contracted Research	Grants Awards- PCORI, Stryker, Penumbra, Microvention
Praveen Mummaneni	Consulting Fee	Depuy Synthes, Globus, BK Medical, Brainlab, SI Bone, Thieme Publisher
	Contracted Research	NREF, ISSG, NIH, DoD, AO Spine, PCORI, SLIP II, Pacire (Fellowship grant)
	Royalty	Springer Publisher
	Own Stock	Discgenics
Joseph Neimat	Consulting Fee	Inc., Monsteris Inc
Eric Oermann	“Employee/Executive”	Eikon Therapeutics
Daniel Orringer	Consulting Fee	NXDC, Stryker Instruments, Medexus
	Stock Options	Invenio Imaging
John O’Toole	Consulting Fee	Globus Medical, Cerapedics
	Royalty	Globus Medical
	Own Stock	Viseon, Inc
Ali Rezai	Future Stock Options	Neurotechnology Innovation Management, Aviation Medical, Realeve
Daniel Sciubba	Consulting Fee	Depuy, Medtronic, Stryker, NuVasive, Baxter, Pacira, SI-Bone
Sameer Sheth	Consulting Fee	Boston Scientific, Zimmer Biomet, Neuronpace, Koh Young, Sensoria Therapeutics, Varian
Vincent Traynelis	Royalty	Medtronic, NuVasive
	Consulting Fee	Medtronic, NuVasive
Corey Walker	Fees for Non-CME/CE Services	Globus
	Consulting Fee	Alphatec
Doris Wang	Consulting Fee	Boston Scientific, Iota Biosciences, Medtronic
Robert Whitmore	Consulting Fee	Depuy Synthes, Intrinsic Therapeutics
	Own Stock	On Point Surgical

## FACULTY

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Faculty	Institution   University	City
Aviva Abosch, MD, PhD	University of Nebraska	Omaha, NE
Manish K. Aghi, MD, PhD	University of California	San Francisco, CA
Felipe Albuquerque, MD	Barrow Brain and Spine	Phoenix, AZ
Wajd Al-Holou, MD	University of Michigan	Ann Arbor, MI
Sepideh Amin-Hanjani, MD	Case Western Reserve University	Cleveland, OH
Wael Asaad, MD	Brown University	Providence, RI
Issam Awad, MD	University of Chicago	Chicago, IL
Tej Azad, MD	Johns Hopkins Hospital	Baltimore, MD
Fred G. Barker II, MD	Harvard University	Boston, MA
David Baskin, MD	Houston Methodist	Houston, TX
Bernard Bendok, MD	Mayo Clinic	Phoenix, AZ
Mitchel Berger, MD	University of California, San Francisco	San Francisco, CA
Chetan Bettgowda, MD, PhD	Johns Hopkins University	Baltimore, MD
John A. Boockvar, MD	Hofstra-Northwell School of Medicine	New York, NY
Nicholas Boulis, MD	Emory University	Atlanta, GA
Samuel Browd, MD	Seattle Children's Hospital	Seattle, WA
Jan-Karl Burkhardt, MD	University of Pennsylvania	Philadelphia, PA
Bob Carter, MD, PhD	Harvard University	Boston, MA
Edward F. Chang, MD	University of California, San Francisco	San Francisco, CA
E. Antonio Chiocca, MD, PhD	Harvard University	Boston, MA
Bryan Choi, MD	Harvard University	Boston, MA
Omar Choudhri, MD	University of Pennsylvania	Philadelphia, PA
Kevin Cockroft, MD	Penn State University	Hershey, PA
E. Sander Connolly, Jr., MD	Columbia University	New York, NY
Andrew Dailey, MD	University of Utah	Salt Lake City, UT
Benjamin Elder, MD, PhD	Mayo Clinic	Rochester, MN



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Faculty	Institution   University	City
James Elder, MD	Ohio State University	Columbus, OH
Dario Englot, MD	Vanderbilt University	Nashville, TN
Peter Fecci, MD, PhD	Duke University	Durham, NC
Michael Fehlings, MD, PhD	Toronto Western Hospital	Toronto, ON Canada
Justin Fraser, MD	University of Kentucky	Lexington, KY
Melanie Gephart Hayden	Stanford University	Stanford, CA
Zoher Ghogawala, MD	Tufts University	Burlington, MA
Jorge González-Martínez, MD, PhD	University of Pittsburgh	Pittsburgh, PA
Oren Gottfried, MD	Duke University	Durham, NC
Gerald Grant, MD	Duke University	Durham, NC
Constantinos Hadjipanayis, MD, PhD	University of Pittsburgh	Pittsburgh, PA
D. Kojo Hamilton MD	University of Pittsburgh	Pittsburgh, PA
Odette Harris, MD	Stanford University	Stanford, CA
Todd Hollon, MD	University of Michigan	Ann Arbor, MI
Judy Huang, MD	Johns Hopkins Hospital	Baltimore, MD
Bermans Iskandar, MD	University of Wisconsin	Madison, WI
Andrew Jea, MD	Indiana University School of Medicine	Indianapolis, IN
Peter Kan, MD	University of Texas Medical Branch	Galveston, TX
Alexander Khalessi, MD	University of California San Diego	San Diego, CA
Albert Kim, MD, PhD	Washington University in St. Louis	St. Louis, MO
Douglas S. Kondziolka, MD	NYU Langone Medical Center	New York, NY
Vibhor Krishna, MD	University of North Carolina	Chapel Hill, NC
Michael T. Lawton, MD	Barrow Neurological Institute	Phoenix, AZ
Eric C. Leuthardt, MD	Washington University in St. Louis	St. Louis, MO
Nancy Levenson, PhD	Space Telescope Science Institute	Baltimore, MD

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<b>Faculty</b>	<b>Institution   University</b>	<b>City</b>
Michael Levitt, MD	University of Washington	Seattle, WA
Linda M. Liau, MD, PhD	University of California, Los Angeles	Los Angeles, CA
Michael Lim, MD	Stanford University	Palo Alto, CA
Michael Link, MD	Mayo Clinic	Rochester, MN
Russell R. Lonser, MD	Ohio State University	Columbus, OH
Andre Machado, MD	Cleveland Clinic	Cleveland, OH
Luigi Mariani, MD	University of Basel	Basel, Switzerland
Ian McCutcheon, MD	University of Texas MD Anderson	Houston, TX
Michael McDermott, MD	Miami Neuroscience Institute	Miami, FL
J. Mocco, MD	Mount Sinai	New York, NY
Jennifer Moliterno, MD	Yale University	New Haven, CT
Praveen V. Mummaneni, MD	University of California San Francisco	San Francisco, CA
Karin Muraszko, MD	University of Michigan	Ann Arbor, MI
Brian Nahed, MD	Harvard University	Boston, MA
Anil Nanda, MD	Rutgers University	Newark, NJ
Joseph Neimat, MD	University of Louisville	Louisville, KY
David Newell, MD	University of Seattle	Seattle, WA
Eric Oermann, MD	NYU Langone	New York, NY
Christopher S. Ogilvy, MD	Harvard University	Boston, MA
Daniel Orringer, MD	NYU Langone	New York, NY
John O'Toole, MD	Rush University Medical Center	Chicago, IL
Ann Parr, MD	University of Minnesota	Minneapolis, MN
Akash, Patel, MD	Baylor College of Medicine	Houston, TX
Matthew Pease, MD	Indiana University	Indianapolis, IN
Alfredo Quinones-Hinojosa, MD	Mayo Clinic Jacksonville	Jacksonville, FL
Ganesh Rao, MD	Baylor College of Medicine	Houston, TX

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Faculty	Institution   University	City
Zach Ray, MD	Washington University in St. Louis	St. Louis, MO
Daniel Resnick, MD	University of Wisconsin	Madison, WI
Ali Rezai, MD	West Virginia University	Morgantown, WV
Laurence Rhines, MD	University of Texas MD Anderson	Houston, TX
Howard Riina, MD	NYU Langone	New York, NY
Shenandoah Robinson, MD	Johns Hopkins University	Baltimore, MD
Marie Roguski, MD	Tufts Medical Center	Boston, MA
Steven J. Schiff, MD, PhD	Penn State University	University Park, PA
Michael Schulder, MD	North Shore University Hospital	Manhasset, NY
Theodore Schwartz, MD	Weill Cornell Medical College	New York, NY
Daniel Sciubba, MD	Hofstra-Northwell School of Medicine	Manhasset, NY
Sameer Sheth, MD, PhD	Baylor University Medical Center	Houston, TX
Adnan Siddiqui, MD, PhD	University of Buffalo	Buffalo, NY
Dennis Spencer, MD	Yale University	New Haven, CT
Robert Spinner, MD	Mayo Clinic	Rochester, MN
Jennifer Strahle, MD	Washington University in St. Louis	St. Louis, MO
Nicholas Theodore, MD	Johns Hopkins University	Baltimore, MD
Vincent Traynelis, MD	Rush University	Chicago, IL
Peter Vajkoczy, MD	Charité - Universitätsmedizin Berlin	Berlin, Germany
Fernando Vale, MD	Augusta University	Augusta, GA
Ashwin Viswanathan, MD	Baylor University	Houston, TX
Michael Vogelbaum, MD	Moffitt Cancer Center	Tampa, FL
Corey Walker, MD	Cedars Sinai	Los Angeles, CA
Doris Wang, MD	University of California San Francisco	San Francisco, CA
Robert Whitmore, MD	Lahey Clinic	Burlington, MA
Ziv Williams, MD	Harvard University	Boston, MA

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Faculty	Institution   University	City
Graeme Woodworth, MD	University of Maryland Medical Center	Baltimore, MD
Risheng Xu, MD	Johns Hopkins University	Baltimore, MD
Gregory Zipfel, MD	Washington University in St. Louis	St. Louis, MO

## GUESTS, LOCATIONS & HOSTS

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Guest	City	Host
Wajd Al-Holou, MD	Ann Arbor, MI	Guest of the Academy
Mohammed Alshareef, MD	Aurora, CO	Gregory Zipfel
Tej Azad, MD	Baltimore, MD	Nicholas Theodore
Nicholas C. Bambakidis, MD	Cleveland, OH	Sepideh Amin-Hanjani
Carolina Benjamin, MD	Miami, FL	Guest of the Academy
Chetan Bettegowda, MD	Baltimore, MD	Henry Brem
Samuel Browd, MD	Seattle, WA	Kim Burchiel
Joseph Cheng, MD	Cincinnati, OH	Shelly Timmons
Omar Choudhri, MD	Philadelphia, PA	M. Sean Grady
Daniel Cleary, MD	Portland, OR	Gregory Zipfel
Ashley Dunbar, MD	St. Louis, MI	Gregory Zipfel
Benjamin Elder, MD, PhD	Rochester, MN	Robert Spinner
Brad Elder, MD	Columbus, OH	Russell Lonser
Aladine Elsamadicy, MD	New Haven, CT	Wilson Z. Ray
Dario Englot, MD	Nashville, TN	James Rutka
Justin Fraser, MD	Lexington, KY	Phillip A. Tibbs
Jorge González-Martínez, MD, PhD	Pittsburgh, PA	Guy McKhann
Oren Gottfried, MD	Durham, NC	Gerald Grant/ Christopher Shaffrey
Christopher Graffeo, MD	Oklahoma City, OK	Bruce Pollock
Andrew Grande, MD	Minneapolis, MN	Raj Narayan
Andrew Hale, MD	Birmingham, AL	Guest of the Academy
D. Kojo Hamilton MD	Pittsburgh, PA	Robert M. Friedlander
Odette Harris, MD	Stanford, CA	Michael Lim
Todd Hollon, MD	Ann Arbor, MI	Karin Muraszko
Peter Konrad, MD	Morgantown, WV	Ali Rezai
Vibhor Krishna, MD	Charlotte, NC	Nelson Oyesiku

<b>Guest</b>	<b>City</b>	<b>Host</b>
Shekar Kurpad, MD	Milwaukee, WI	Guest of the Academy
Wendell Lake, MD	Madison, WI	Daniel Resnick
Dimitrios Mathios, MD	St. Louis, MO	Gregory Zipfel
Jonathan P. Miller, MD	Syracuse, NY	Warren R. Selman
Jennifer Moliterno Gunel, MD	New Haven, CT	Guest of the Academy
Ziev Moses, MD	Boston, MA	Wilson Z. Ray
Joseph Neimat, MD	Louisville, KY	Guest of the Academy
Eric Oermann, MD	New York, NY	Douglas Kondziolka
John O'Toole, MD	Chicago, IL	Vincent Traynelis
Aditya Pandey, MD	Ann Arbor, MI	Guest of the Academy
Ann Parr, MD	Minneapolis, MN	Guest of the Academy
Harold Phillips, MD	Stanford, CA	Gregory Zipfel
Marie Roguski, MD	Boston, MA	Carl Heilman
Cameron Sadegh, MD	Davis, CA	Gregory Zipfel
Anthony Schulien, MD	Pittsburgh, PA	Guest of the Academy
Alfred Pokmeng See, MD	Boston, MA	Gregory Zipfel
Jennifer Strahle, MD	St. Louis, MI	Guest of the Academy
Jignesh Tailor, MD	Indianapolis, IN	Alan Cohen
Philip Theodosopoulos, MD	San Francisco, CA	Michael McDermott
Craig van Horne, MD	Lexington, KY	Kendall H. Lee
Doris Wang, MD	San Francisco, CA	Edward Chang
Robert Whitmore, MD	Burlington, MA	Zoher Ghogawala
Ziv Williams, MD	Boston, MA	Tina Duhaime
Jon Willie, MD	St. Louis, MO	Albert Kim
Stacey Wolfe, MD	Winston-Salem, NC	Guest of the Academy
Henry Woo, MD	Rego Park, NY	Brian Hoh
Risheng Xu, MD	Baltimore, MD	Guest of the Academy
Mohamed Zaazoue, MD	St. Louis, MO	Wilson Z. Ray



THE AMERICAN ACADEMY OF NEUROLOGICAL SURGERY  
86<sup>TH</sup> ANNUAL SCIENTIFIC MEETING  
SCIENTIFIC PROGRAM

WEDNESDAY, OCTOBER 16, 2024

REGISTRATION AND RECEPTION

THURSDAY, OCTOBER 17, 2024

7:30 – 7:35 WELCOMING & INTRODUCTION

Shenandoah Robinson, MD and Daniel Resnick, MD

7:35 – 7:45 Historian's Report

Michael Schulder, MD

7:45 – 9:00 Peer Reviewed Abstract Session I: Tumor I

Moderators: Luigi Mariani, Manish Aghi, and Ted Schwartz

7:45 – 7:55 Ultra-rapid droplet digital PCR enables IDH mutation detection and quantification of tumor burden at surgical margins – Oldfield Award

Daniel Orringer, MD

Introduction

The vast majority of adult diffuse low-grade gliomas harbor oncogenic gain of function mutations in the IDH1 gene. Detection of IDH1 mutations during glioma surgery would facilitate diagnosis and shape operative strategy. The fastest reported molecular diagnostic methods for IDH1 mutation detection rely on a complex workflow with turnaround times that preclude swift and iterative use during surgery.

Objectives

Here, we introduce an ultra-rapid droplet digital PCR (UR-ddPCR) workflow that profoundly reduces the time from tissue biopsy to molecular diagnosis and serves as a highly accurate means of quantifying residual tumor infiltration at tumor margins.

Methods

We developed and validated a 15 minute UR-ddPCR assay for the detection of IDH1 R132H hotspot mutations in 31 specimens collected from 12 patients in the operating room.

## Results

UR-ddPCR allele fraction predicted in 15 minutes was virtually identical to the allele fraction predicted by the standard 3-hour ddPCR workflow (allele fraction range: 0.14-94.4%;  $p=5.8e-53$ ,  $R^2=0.9958$ ). The UR-ddPCR assay enabled accurate quantification of tumor cell concentration, ranging from >1100 IDH mutant cells/mm<sup>2</sup> within tumor core to <1 IDH mutant tumor cell/mm<sup>2</sup> at the tumor margins.

## Conclusion

The UR-ddPCR workflow developed here represents the fastest and most accurate intraoperative molecular genetic assay reported to date. We anticipate that our method, along with its planned automation, will further reduce turnaround time from tissue to mutational detection and facilitate molecular guidance to inform intraoperative diagnosis and decision-making in neurosurgical oncology.

<b>7:55 – 8:05</b>	<b>Microbubble-enhanced Focused Ultrasound and Temozolomide for High Grade Gliomas</b>
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**Graeme Woodworth, MD**

## Introduction

Microbubble-enhanced focused ultrasound (MB-FUS) has been shown to be safe, feasible, and repeatable in multiple clinical studies of HGGs and to have multi-modal therapeutic effects including temporary opening of the blood brain barrier (BBB), increased drug delivery and activity, and immunomodulation.

## Objectives

We report the first combined results and outcomes of a prospective, single arm, multi-center Phase 1 clinical trial of MB-FUS in HGG patients combined with standard monthly adjuvant temozolomide chemotherapy.

## Methods

Thirty-four patients receiving standard temozolomide (TMZ) chemotherapy underwent MB-FUS treatments each month. The study outcomes included clinical and radiologic endpoints of safety and feasibility, brain imaging (MRI) measurements of BBB opening, assessments of progression-free survival (PFS) and overall survival (OS). PFS/OS were assessed using a 1:10 Coarsened Exact Matching cohort design with Cox proportional hazards regression analysis. MB-FUS-enabled sono-liquid biomarker analyses of cell-free DNA were performed and correlated with tumor responses.

## Results

The 34 patients completed an average of 4 monthly transcranial MB-FUS plus TMZ cycles with no device-related SAEs. BBB opening was visualized in 99% of treatments covering 92% of the targeted volume. Median PFS in the treatment groups was 14.3 months compared to 10.4 months in the control group (relative risk reduction of 41%,  $p=0.02$ ). Median OS was 36.4 months compared to 17.4 months in the control group (relative risk reduction of 53%,  $p<0.001$ ). Sono-liquid cell-free DNA ratios from the peak level to the final level showed a significant correlation with survival outcomes ( $p=0.04$ ).

## Conclusion

The combined results of this study demonstrate that monthly transcranial, localized MB-FUS with TMZ is a practical and safe combination therapeutic approach for HGG patients, with a high percentage of temporary BBB opening achieved. The results also provide evidence of therapeutic and diagnostic benefits of MB-FUS including the potential to improve PFS and OS and enhance tumor monitoring through novel sono-liquid biomarker analyses.



**8:05 – 8:15 Radiographic and Radiomic Features Differentiate the Aggressive Molecular Group from the Benign Groups of Meningioma**

**Akash Patel, MD**

Introduction

Meningiomas are the most prevalent primary intracranial neoplasms. We pioneered a departure from the WHO system, utilizing multiplatform profiling to identify three molecular groups (MenG A, B, and C) that predict recurrence better than histopathology. However, current profiling methods require tissue samples, highlighting the need for non-invasive preoperative identification of molecular groups.

Objectives

We used semantic (radiographic) and quantitative (radiomic) features on preoperative MRI to differentiate benign (MenG A and B) and aggressive (MenG C) meningioma.

Methods

We examined preoperative MRIs of patients with molecularly classified tumor samples from 2012-2020 (n=178, training set). We used standardized radiographic variables and radiomic features extracted from T1 post-contrast sequences. Features were selected using non-parametric analyses and refined with a recursive feature elimination algorithm. Random forest and neural network algorithms were trained on selected features to classify molecular subtypes. Models were validated on unseen data from tumors resected from 2021-2022 (n=66, validation set). Finally, a model was trained on the entire dataset to create the best predictive model.

Results

Both classification algorithms achieved similar optimal accuracies (79-80%, AUC 0.84-0.85) for predicting benign vs. aggressive meningioma within the training set. Predicting the molecular status of tumors from the validation set using training data yielded higher accuracies (83-89%, AUC 0.88-0.89). Overall, we predicted molecular status for all tumors (n=244) with an accuracy of 82% and an AUC of 0.86. Key predictors included tumor location and sex. Additionally, the random forest algorithm had zero false negative errors when assessing meningiomas without edema. Consequently, we could definitively classify 38% of our patients as having benign meningiomas, allowing for a more conservative treatment approach.

Conclusion

Preoperative imaging can differentiate benign and aggressive molecular status in meningiomas, aiding in more accurate preoperative counseling and treatment planning.

**8:15 – 8:25 Intracranial Tumors Elicit Systemic Sympathetic Hyperactivity that Limits Immunotherapeutic Responses**

**Peter Fecci, MD**

Introduction

Tumors situated intracranially (GBM, brain metastases), elicit unique combinations of local and systemic immune dysfunction whose mechanistic underpinnings are incompletely understood.

### Objectives

Determine how tumors confined to the intracranial compartment elicit systemic immune derangements, including T cell dysfunction, lymphopenia, splenic and thymic atrophy, and bone marrow T cell sequestration.

### Methods

Given its capacity for driving brain-immune reflexes in other disease states, the sympathetic nervous system was investigated. Systemic levels of catecholamines were assessed in mice with intracranial glioma, melanoma, lung, and breast cancers, as well as in patients with newly diagnosed or recurrent GBM. Single-cell RNA-sequencing determined expression levels of adrenergic receptors on lymphocytes and tumors in mice and patients with GBM and brain metastases. Pumps delivering catecholamines or various adrenergic agonists were implanted into mice to assess sufficiency for reproducing tumor-imposed immune dysfunction. Survival was evaluated in glioma-bearing mice administered immunotherapy and/or beta-adrenergic blockade. Large scale analysis of SEER-Medicare data evaluated the association between beta-adrenergic blockade and survival (+/- immunotherapy) in patients with GBM (n=8743), metastatic lung cancer with and without brain involvement (n=25,711; 68,041), or metastatic melanoma with and without brain involvement (n=2332; 1218).

### Results

Tumors harbored intracranially elicit systemic increases to circulating catecholamine levels, driving immune dysfunction and limiting immunotherapeutic success. Conversely, beta-blockade increases immune cell NF-KB activity, restores T cell polyfunctionality, modifies the tumor microenvironment, and extends survival to immune-based therapies in murine models of GBM. Extended survival is also observed in GBM patients receiving beta-blockade, as well as in patients with melanoma and lung cancer brain metastases receiving beta-blockade and immune checkpoint inhibition. While beta-blockade likewise impacts outcomes in the setting of extracranial disease, benefits are especially pronounced with intracranial disease burdens.

### Conclusion

This suggests sympathetic hyperactivity facilitates immune dysfunction in the setting of intracranial tumors and advances a role for beta-blockade in licensing immunotherapeutic responses within the intracranial compartment.

<b>8:25 – 8:35      Ventricular Entry, Tumor Contiguity, and Leptomeningeal Disease after Resection of Supratentorial Glioblastoma</b>
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**Ian McCutcheon, MD**

### Introduction

Ventricular entry (VE) can maximize extent of resection (EOR) in patients with glioblastoma (GBM), but it remains unclear whether tumor contiguity to the ventricles or VE (or both) increases the risk of leptomeningeal disease (LMD) and/or worsens overall survival (OS).

### Objectives

We sought to clarify the role of VE and tumor location in influencing the incidence of post-operative LMD and OS.

### Methods

We retrospectively reviewed patients who underwent first resection of supratentorial GBM or gliosarcoma between 1993-2021 at a single center. OS and time to LMD diagnosis were estimated using the Kaplan-

Meier method; their associations with patient and treatment variables were assessed via Cox regression analysis.

### Results

Of 884 patients, 390 (44%) had VE and 444 (50%) had ependymal contact (EC) tumors. EC occurred in 82% of patients with VE; only 25% of those without VE had EC ( $p < 0.0001$ ). On multivariate analysis, VE did not significantly predict LMD (HR 1.42 (0.69-2.9);  $p = 0.34$ ). EC significantly increased LMD risk (HR 2.63 (1.13-6.12);  $p = 0.02$ ). VE was not associated with worse OS (HR 1.03 (0.87-1.22);  $p = 0.744$ ), unlike EC (HR 1.33 (1.1-1.6);  $p = 0.003$ ). Although patients with VE had lower complete resection rates than those without VE (63% vs 72%,  $p = 0.005$ ), VE improved EOR among EC tumors (58% had complete resection) vs. 47% among EC tumors without VE.

### Conclusion

Tumor proximity to ventricles predicts higher LMD risk and shorter OS; VE during resection does not increase LMD risk or worsen OS. Surgeons can still use VE for resection of supratentorial gliomas without increasing the risk of subsequent LMD.

<b>8:35 – 8:45      Sensitive Detection of Central Nervous System Tumors Using a Sequencing Based Cerebrospinal Fluid Test</b>
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**Chetan Bettgowda, MD**

### Introduction

The current approach to diagnosing and monitoring tumors of the central nervous system relies almost exclusively on radiographic imaging and neurosurgical procedures. Cerebrospinal fluid (CSF) is appealing for diagnosis because it is already part of the standard of care for the diagnosis or management of several types of CNS disease, including cancer, and the tumor DNA is more highly concentrated inside the blood brain barrier than in plasma or other bodily fluids. Even though cytology is widely used, sensitivity remains low ranging from 2% to 50%, depending on cancer type.

### Objectives

To develop a minimally invasive, next generation based molecular assay to aid in the diagnosis and monitoring of brain cancers by identifying tumor derived DNA in cerebrospinal fluid.

### Methods

We report an analytic technique that efficiently introduces identical molecular barcodes to both strands of CSF template DNA molecules for the identification of cancer specific genetic alterations. Our assay is able to identify molecules of tumor derived DNA as rare as 1 mutant molecule in a background of 100,000 wild type molecules. Tumor derived DNA is identified by detecting mutations in canonical cancer driver genes from 121 different amplicons and whole genome sequencing to identify chromosomal copy number alterations.

### Results

We apply this approach to 126 CSF samples obtained from individuals with known primary or metastatic tumors involving the brain and 36 CSF samples obtained from individuals with non-neoplastic neurological conditions such as multiple sclerosis. We correctly detect 91% of cancers at a specificity of 94.5% (Table 1). In cases ( $n = 20$ ) where standard of care cytology was available, cytology detected 25% of cancers while our molecular assay detected 90%.

### Conclusion

Our molecular approach has the capacity to be used in combination with other clinical, radiologic, and laboratory-based data to inform the diagnosis and management of patients with suspected cancers of the brain.

### **8:45 – 8:55 Expert Panels Can Identify Variation and May Help Guide Care among Patients with CNS Neoplasms: a Survey-based Study**

Marie Roguski, MD

### Introduction

Treatment variation in the care of patients with central nervous system (CNS) neoplasms is prevalent. Unwarranted variation can lead to increased costs of care with limited benefits.

### Objectives

To identify sources of variation in the treatment of brain tumors and determine whether expert review of standardized vignette-based surveys can be used to identify and reduce unwarranted treatment variation.

### Methods

We administered 203 de-identified clinical vignette-based surveys to a panel of 18 experienced brain tumor-focused neurosurgeons. Consensus was defined as at least 80% consensus with at least 10 experts providing responses on treatment. Chart review was performed to obtain baseline demographic, treatment, and outcomes data on submitted cases. Statistical analysis was performed with SAS enterprise.

### Results

Consensus was observed in 54.7% of surveys. Gross total resection (GTR) was the most commonly recommended treatment among glioma patients when consensus was met (73.8%). Biopsy and GTR were the most commonly recommended treatments when 80% consensus was not met (45.2% and 41.9%, respectively). When recommended extent of resection was further subdivided into supramaximal resection, GTR, or 5-ALA-guided, consensus decreased to 80 of 203 surveys (39.4%). Consensus was more likely among metastasis patients (OR 1.78, 95% CI 1-3.2, p=0.05). When consensus was met, the actual treatment aligned with the recommended treatment in 91.2% of patients. There was no difference in progression free survival between patients whose treatment was aligned and those whose treatment was not aligned or did not achieve consensus (p=0.44). There was no difference in overall survival in these groups (p=0.98). If consensus definition was liberalized to at least 70%, consensus improved significantly to 71.4%.

### Conclusion

Expert panels can aid in identifying and reducing treatment variation among patients with CNS neoplasms. There is significant disagreement among experts regarding degree of resection among resectable gliomas

### **8:55 – 9:00 Wrap up and Transition**

### **9:00 – 10:10 Peer Reviewed Abstract Session II: Spine I** Moderators: Andrew Jea and Nicholas Theodore

### **9:00 – 9:10 The Subparaneurial Compartment: A New Concept in the Clinicoanatomic Classification of Peripheral Nerve Lesions**

Robert Spinner, MD

### Introduction

Nerve lesions and tumors have historically been considered as being localized to intraneural or extraneural compartments. There are obvious surgical implications if lesions are contained within or outside of the dense connective tissue layer, the epineurium. The paraneurium is a loose layer of connective tissue that surrounds the epineurium; the subparaneurial compartment is a potential space that can expand and host various pathologies.

### Objective

To illustrate a spectrum of peripheral nerve pathologies localized to the subparaneurial compartment

### Methods

From our experience with 50 cases of lesions in the subparaneurial compartment, we will present examples to demonstrate the existence, imaging and operative appearance, and surgical implication of different peripheral nerve pathologies occurring in this subparaneurial compartment (by itself or together with an intraneural component).

Four different categories of pathologies will be discussed including vascular lesions (hemangiomas); adipose tumors (lipomas/lipomatosis of nerve); intraneural ganglion cysts; and the most common, hematologic malignancies (such as 'tumefactive' neurolymphomatosis, neuroleukemiosis or neuroplasmacytoma).

### Results

In all cases, high resolution MRI depicts characteristic patterns that can not only establish the anatomic localization, but also the diagnosis (pathology); in other cases, it provides insight into formation/propagation patterns as well. This information can help surgeons predict the resectability of the lesion (without a neurologic deficit) or determine the safest place to biopsy; in other cases, the entity (a subparaneurial cyst, a rare variant of an intraneural ganglion) can be treated by disconnecting the articular branch-joint connection and decompressing the cyst (rather than attempting to resect it).

### Conclusion

The subparaneurial compartment is becoming known to anesthesiologists who are injecting local anesthesia within it to allow circumferential spread around major nerves (e.g., sciatic nerve and brachial plexus). Suffice it to say, there is little knowledge about the relevance and the importance of this compartment amongst neurosurgeons.

<b>9:10 – 9:20      An Economic Analysis of the Cervical Spondylotic Myelopathy Surgical Trial; Cost-Effectiveness of Surgical Approaches</b>
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**Robert Whitmore, MD**

### Introduction

Surgery for cervical spondylotic myelopathy (CSM) improves quality of life but surgical approaches might differ by cost.

### Objectives

To evaluate cost-effectiveness of anterior cervical discectomy/fusion (ACDF), posterior cervical decompression/fusion (PCDF), and laminoplasty (LP).

## Methods

We conducted a cost-effectiveness analysis of a prospective randomized trial comparing surgical approaches for CSM across 15 sites in North America. Patients were randomized (2:3) to either anterior (ACDF) or posterior surgery (LP or PCDF, at surgeons' discretion). A cost analysis was performed from a societal perspective with a one-year time horizon, including only patients from the United States. Direct costs were estimated using 2022 Medicare reimbursement rates for professional fees and cost-to-charge ratios. Indirect costs were estimated using a human capital approach based on patient surveys. Effectiveness was measured in quality-adjusted life-years (QALYs) using the Euro-Qol-5-Dimensions (EQ-5D) at one year.

## Results

153 patients were included as-treated in a three-way cost analysis by surgical approach. Index hospitalization costs were higher after PCDF than ACDF and LP (\$32,507 vs. \$24,991 vs. \$24,574;  $p < 0.0001$ ). 34 patients (22.2%) had complications. Complication costs and lost wages did not differ between groups. One-year total costs were higher after PCDF than ACDF and LP (\$49,590 vs. \$39,678 vs. \$40,716;  $p = 0.0072$ ). For 71 patients with one-year costs and EQ-5D outcomes available, PCDF was associated with lower QALY gains than ACDF (0.687 vs. 0.786,  $p = 0.029$ ) and LP (0.687 vs. 0.791,  $p = 0.062$ ).

## Conclusion

Among patients in the CSM-S Trial, LP and ACDF had similar cost-utility. PCDF was less cost-effective, yielding worse outcomes with higher costs, driven by index hospitalization.

<b>9:20 – 9:30      Use of Expert Panel for Patients with Grade I Degenerative Lumbar Spondylolisthesis: A Randomized Clinical Trial</b>
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Zoher Ghogawala, MD

## Introduction

Recent published RCTs have created uncertainty around the appropriate utilization of lumbar fusion for symptomatic lumbar degenerative spondylolisthesis.

## Objectives

To perform an RCT to determine if a surgical expert review panel recommending fusion might improve patient satisfaction and reduce surgical failures.

## Methods

14 sites randomized patients to receive an expert panel review (10-15 surgeons' review of key images and history) or not. Patients had surgical treatment at the discretion of the treating surgeon. Analysis was focused upon whether a strong majority (>80% consensus) of spinal experts recommending fusion might reduce operative failures. Outcomes (EQ-5D and ODI) were assessed pre-operatively and at 3, 6, 12, and 24 months. NASS patient satisfaction was assessed at 1 year.

## Results

Fourteen sites randomized 662 patients. Mean age was 65.6 years (59.5% female). Overall, fusion was performed on 78% of patients. Follow-up was 79% at 1 year. Super majority recommendation for fusion was associated with -0.296 change in vs. 0.239 change in EQ5D ( $p = 0.035$ ). With super majority recommendation recommending fusion, 7.5% of patients failed to improve EQ-5D score vs. 18.6% in the non-review group ( $p = 0.016$ ). With super majority favoring fusion, NASS grade I patient satisfaction was 68% compared to 55% patients in non-review group ( $P = 0.042$ ).

### Conclusion

There is evidence that direct electrical stimulation of the motor thalamus augment motor output in the upper limb segment, both in non-human and human primates. We hope to use these outcomes to implement DBS of motor thalamus as a potential therapeutic approach to treat post-stroke motor deficits.

<b>9:30 – 9:40</b>	<b>Pre- Operative Anemia is an Unsuspecting Driver of Machine Learning Prediction of Adverse Outcomes after Spinal Fusion</b>
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**Corey Walker, MD**

### Introduction

Predicting post-surgical outcomes in spinal fusion patients is crucial for pre-operatively assessing procedure feasibility and success.

### Objectives

This study investigates the use of novel automated machine learning models to predict the adverse outcomes.

### Methods

The study is based on electronic records from a single institution of elective spinal fusions performed over roughly one decade. Employing the automated machine learning tool TPOT, we construct, optimize, and select classification predictive models for our outcomes. TPOT utilizes genetic programming to select optimal pipelines in a process inspired by evolution. We derive Shapley values to identify the major features influencing the predictive power of optimal models.

### Results

We analyzed 5,248 operations from 4,952 patients. We observed adverse outcome rates of 24.8% for discharge to a non-home facility, 21.1% for extended hospital stays, and 15.5% for readmission within 90 days. The best-performing models achieved a balanced accuracy of 0.75 for discharge disposition, 0.73 for length of stay, and 0.64 for readmission. Notably, preoperative hemoglobin emerges as a consistently strong predictor in the models. Patients with levels of severe anemia demonstrated higher associations with adverse outcomes. Additionally, metabolic, and weight-related comorbidities significantly influence post-surgical outcomes.

### Conclusion

This study demonstrates the effectiveness of automated machine learning in constructing predictive models and its power in identifying key variables associated with outcomes. The persistent significance of preoperative hemoglobin as a top predictive feature across various models and outcomes suggests its critical role in pre-surgical assessment. Age, BMI, insurance type, and specific comorbidities also demonstrate notable effects on outcomes, but hemoglobin emerges as a prominent single major contributor, independent of age. These findings underscore the potential of enhancing patient care through predictive modeling and highlight the critical role of thorough preoperative assessments in improving surgical outcomes.

<b>9:40 – 9:50</b>	<b>Complications Affecting Patient Satisfaction and Outcomes Following Adult Spinal Deformity Surgery</b>
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**D. Kojo Hamilton, MD**

### Introduction

Surgical management of adult spinal deformity (ASD) has a well-cited complication profile, including medical, neurologic, and implant-related complications. A subset of patients who experience perioperative complications still express post-surgical satisfaction with surgical management.

### Objectives

To identify the characteristics of complications that influence levels of satisfaction.

### Methods

Data was obtained from a multicenter cohort of patients with ASD, who developed postoperative complications within two years of follow-up. Satisfaction was determined by the SRS-22 satisfaction subscore. Demographic and spinopelvic parameters at baseline and two years were recorded. Complication-related variables included latency, frequency, subcategory, and severity (high = major/requiring reoperation and low = minor). The associations between the variables were evaluated with multiple linear regression. Post-hoc analysis was conducted of the complication profiles significantly related to satisfaction ( $p$ -value < 0.05).

### Results

Of the initial 795 patients meeting inclusion criteria, 533 (67.0 %) had at least one complication. The demographic, spinopelvic, and satisfaction parameters are shown in Table 1. On regression, patients with implant-related ( $\beta$ = 0.39,  $p$  = 0.006) and infection-related ( $\beta$ = 0.42,  $p$  = 0.017) complications were associated with greater satisfaction at two years. Complication frequency was negatively associated with satisfaction at two years ( $\beta$ = -0.14,  $p$  = 0.018). Among patients who had an infectious complication, those who developed sepsis were more likely to have worse satisfaction at two years ( $\beta$ = -1.66,  $p$  = 0.047). The subcategory of neurologic or implant-related complication was not associated with satisfaction at two years.

### Conclusion

Sustaining an adverse event with a permanent deficit yields poorer satisfaction, when compared with recoverable adverse events. Greater complication frequency, due to the cumulative effect of multiple complications, yields poor long-term satisfaction. Patients with implant-related or infectious complications tended to have better long-term satisfaction compared to patients with other categories of complications (Table 2).

## **9:50 – 10:00 Surgical Resection of Spinal Chordoma: Overall Survival and Local Recurrence**

Daniel Sciubba, MD

### Introduction

Spinal chordomas are slow-growing primary bone tumors where surgery represents the primary treatment modality. However, their low incidence, lack of evidence, and late disease presentation make them challenging to manage.

### Objectives

We investigate the postoperative outcomes of a large cohort of patients after definitive surgical resection, the predictors for overall survival (OS) and local recurrence-free survival (LRFS). We also trend functional outcomes over multiple time periods.



### Methods

A retrospective review of patients with spinal chordomas were followed at a single institution. Data was collected regarding demographics, preoperative treatment, perioperative management, and follow-up since initial definitive surgery. Primary outcomes were mortality, local tumor recurrence, and functional outcomes.

### Results

101 patients had an average follow-up of  $5.9 \pm 4.2$  years. At time of census, 25/101 (24.8%) had experienced a recurrence and 10/101 (9.9%) had died. After surgery, patients experienced a significant decrease in pain over time, but rates of sensory deficits, weakness, and bowel/bladder dysfunction remained static. Decreased LRFS was significantly associated with tumor volume  $>100\text{cm}^3$  ( $p=0.045$ ) and Enneking Inappropriate resection ( $p=0.032$ ). OS was significantly lower among patients who were  $>65$  years old at the time of surgery ( $p<0.001$ ), had a tumor in the mobile spine rather than fixed spine ( $p=0.046$ ), and underwent preoperative radiotherapy ( $p=0.011$ ). Multivariate analysis indicated tumors  $>100\text{cm}^3$  had a higher risk of recurrence, and patients  $>65$  years old at time of surgery had a higher risk of mortality.

### Conclusion

Surgeons must weigh the pros and cons of en bloc resection. There appears to be a higher risk for local recurrence in tumors  $>100\text{cm}^3$  and OS is worse in those  $>65$  years old at time of surgery.

<b>10:00 – 10:10    Machine Learning to Predict 5 Year Post-Op Back Pain in Patients with Grade 1 Lumbar Spondylolisthesis: A QOD Study</b>
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**Praveen Mummaneni, MD**

### Introduction

Back pain is a common symptom in patients with lumbar spondylolisthesis. Machine learning (ML) can predict improvement in back pain following surgery in patients with grade 1 lumbar spondylolisthesis.

### Objectives

We evaluated predictors of achievement of the minimum clinically important difference (MCID) in back pain and related disability after surgery in patients with grade 1 spondylolisthesis using ML models.

### Methods

This was a prospective analysis using the Quality Outcomes Database consisting of adult patients with grade 1 lumbar spondylolisthesis. 608 patients were split into an 80% training cohort/20% testing cohort. Hyperparameter tuning was performed with 5 fold cross-validation. Recursive feature selection was used to select key pre-operative variables for predicting achievement of MCID in Numerical Rating Scale Back Pain (NRS-BP) and Oswestry Disability Index (ODI). The final model was tested for accuracy on the testing cohort.

### Results

Of the algorithms, logistic regression demonstrated the best accuracy ( $0.77 \pm 0.03$ ), followed by AUROC ( $0.75 \pm 0.04$ ) at predicting MCID achievement for NRS-BP at 5 years post-operatively. Similarly, logistic regression demonstrated the best accuracy ( $0.71 \pm 0.04$ ), followed by AUROC ( $0.73 \pm 0.04$ ) at predicting MCID achievement for ODI at 5 years post-operatively. Top variables for predicting MCID for NRS-BP include baseline NRS-BP, baseline NRS-Leg Pain, baseline ODI, ASA grade, and age at time of surgery. Top variables for predicting MCID for ODI included baseline ODI, NRS-Leg Pain, educational level, baseline NRS-BP, and smoking status.

### Conclusion

Top variables for predicting MCID for NRS-BP and ODI include baseline patient reported outcomes, educational level, smoking status, ASA grade, and age at time of surgery.

**10:10 – 10:30 Break**

**10:30 – 10:50 Special Debate Session I: Regulation of Innovation: How much is enough?**  
Moderator: Doug Kondziolka

**10:30 – 10:32 Introduction**

Doug Kondziolka, MD

**10:32 – 10:40 New Products and Procedures Need to be Tightly Regulated to Prevent Disaster**

Nicholas Boulis, MD

**10:40 – 10:48 Over-Regulation is Stifling Innovation**

Adnan Siddiqui, MD

**10:48 – 10:55 Wrap Up and Transition**

**10:55 – 11:55 Peer Reviewed Abstract Session III: Pediatrics, Trauma, and Other.**

Moderators: Karin Muraszko, Bermans Iskandar, Michael McDermott

**10:55 – 11:05 Poor Surgical Outcomes Following Paenibacillus Infant Infectious Hydrocephalus**

Steven Schiff, MD

### Introduction

We previously identified Paenibacillus species in the cerebrospinal fluid of 44% of infants under 90 days of age presenting for neurosurgical evaluation with findings consistent with postinfectious hydrocephalus in Eastern Uganda (Morton et al, The Lancet Microbe 2023; Ericson et al, Clinical Infectious Diseases 2023).

### Objectives

To compare the outcomes among hydrocephalic infants with and without Paenibacillus detection at the time of hydrocephalus surgery.

### Methods

In a prospective observational trial, 78 infants with apparent postinfectious hydrocephalus who underwent a cerebrospinal fluid (CSF) diversion prior to 90 days of age had a positive CSF polymerase chain reaction result for Paenibacillus species (PP), and 111 had a negative result (PN). The primary outcome was diversion failure-free survival defined as being alive without diversion failure at the end of the observation period. Secondary outcomes included overall survival and diversion success.

### Results

After median follow-up 35.7 months, the primary outcome occurred in 42 PP (54%) and 76 PN patients (68%) (adjusted hazard ratio [aHR], 2.45; 95% confidence interval [CI], 1.42 to 4.22; P=0.001). PP patients

who underwent endoscopic diversion had the worst primary event rate (aHR, 6.47; 95% CI, 2.40 to 17.42; P<0.001). Death occurred in 16 PP (20%) and 9 PN patients (8%) (aHR, 3.47; 95% CI, 1.44 to 8.37; P=0.006). Diversion failure occurred in 28 PP (36%) and 29 PN patients (26%) (aHR, 2.24; 95% CI, 1.31 to 3.85; P=0.003).

### Conclusion

Paenibacillus PCR detection in the CSF at the time of hydrocephalus surgery was associated with a significantly increased rate of diversion failure or death, particularly for patients with endoscopic diversion. Our findings are consistent with active brain infection persisting from previous neonatal sepsis. It appears important to test such patients for evidence of active infection at the time of surgery, and to investigate the role of pre- and peri-surgical antibiotic therapy to improve outcomes.

## 11:05 – 11:15 Artificial Intelligence Assessment of Endoscopic Third Ventriculostomy Success from Intraoperative Video

Samuel Browd, MD, PhD, FAANS

### Introduction

AI and associated technologies like computer vision stand to transform our understanding of neurosurgery and impact the performance of surgery.

### Objectives

The probability of successful endoscopic third ventriculostomy (ETV) is modified by intraoperative factors such as cisternal scarring and aqueduct patency. Intraoperative assessment of these factors varied between surgeons - with good inter-rater reliability on aqueduct patency and moderate agreement on cisternal scarring. We evaluated whether an artificial intelligence (AI) system to accurately identify cisternal scarring and aqueduct patency using computer vision could replicate expert surgeon judgement using very few examples.

### Methods

We used a previously published and expert-adjudicated dataset of 10-second video clips with 840 expert ratings. 30 clips showing cistern status and 26 operative videos for aqueduct patency were scored by 14 experts and 1 super-expert providing ultimate ground truth. Data split into three datasets: training, validation, and testing, for AI training and independent verification. A pre-trained computer vision model, ResNET101, and PyTorch were used in a transfer learning paradigm to provide clip-level predictions of aqueduct patency and of cisternal scarring.

### Results

For predicting open vs closed aqueducts, the algorithm reached a promising 80% accuracy in validation and 77% accuracy in independent verification, approaching human raters (87% accuracy). In differentiating between scarred and not-scarred cisterns, the algorithm achieved 52% accuracy in validation and 56% accuracy in independent testing, which is not significantly better than chance and worse than humans (67% accuracy).

### Conclusion

Computer vision assessment of surgical video is a feasible method of identifying some clinically relevant features of endoscopic neurosurgical anatomy despite minimal input data, lack of prior knowledge, and low video resolution. When expert raters disagree frequently, computer vision models may require significantly greater quantities of training data to achieve or surpass human performance.

**11:25 – 11:35 The CSF-Brain Axis and Its Contribution to Functional Neural Networks in Preterm Intraventricular hemorrhage**

**Jennifer Strahle, MD**

Introduction

CSF plays a critical role in the growth and functioning of the central nervous system however the nature of CSF-brain interactions during development is not known. Furthermore the contribution of altered CSF circulation to neural progenitors in the pathophysiology of poor neurocognitive outcomes in preterm germinal matrix hemorrhage-intraventricular hemorrhage (GMH-IVH) is unknown.

Objectives

To determine cell type specific CSF-brain interactions during development and how alterations in CSF circulation in GMH-IVH contribute to impaired neuronal maturation and functional neural networks.

Methods

After CSF infusion of fluorescent CellTracker tracer in naïve P8 and P21 mice, intracellular FACS sorting was used to isolate CSF contacting brain cells for ScRNA-seq. IVH was induced with 20  $\mu$ L of hemoglobin into the right lateral ventricle of P4 rodents, with aCSF shams as controls. IVH rodents underwent CSF tracking at P7 or optical functional imaging of resting state networks and behavior analysis at P28.

Results

CSF interacts with spatially-distinct brain cell populations at P8 and P21, with those at P8 identified as primarily neural progenitor and oligodendrocyte precursor cells including cerebellar and pontine interneurons (Skor1, Tfap2b, Tfap2a), migratory neuroblasts (Dcx, psa-ncam, Tubb3, Dlx1); and transient amplifying progenitors in the cerebellum (Atoh1, Top2a, Mki67, Mcm) (Fig 1). IVH resulted in decreased CSF-neuron interactions, and downregulation of neurogenesis markers including Dlx1 (Fig 2). GMH-IVH resulted in negative impacts on global and network-specific resting state functional networks as well as behavior outcomes (fear conditioning) (Fig 3).

Conclusion

These findings provide novel insight into how CSF circulation within the developing brain is biologically regulated. Furthermore, disruption of CSF delivery to neurogenic populations may underly poor cognitive function in preterm IVH through altered neuronal maturation and impaired neural network formation. Therapies aimed at restoring CSF circulation to CSF contacting cells may improve cognitive outcome in GMH-IVH.

**11:25 – 11:35 Impact of Sex Differences in TBI Outcomes: Path to Precision**

**Odette Harris, MD**

Introduction

Upending traditional perspectives are the hallmark of innovation and change management.

Objectives

We explore sex difference in TBI using biomarkers and measures of gray/white matter integrity and examined the underpinnings of differential recovery; gender and adverse childhood exposures (ACES)

## Methods

#1. Conducted sequential studies of TBI versus neurologically healthy controls - cohort analysis, matched analysis and focus on brain behavior interface. Measures of gray and white matter integrity - Cortical thickness, diffusion properties and cognitive performance were examined.

#2. ACES:101 veterans completed TBI Model Systems 2010-2024 during hospitalization. Follow-up assessed Pediatric ACEs and Related Life Events Screener (PEARLS), Childhood Trauma Questionnaire-Short Form (CTQ), PTSD Checklist for Civilians (PCL-C) and Neurobehavioral Symptom Inventory (NSI) and Glasgow Coma Scale (GCS).

## Results

#1. Numerous sex differences in recovery and re-entry are identified in cohort and matched analyses. Biomarker analyses noted - In female TBI patients, cortical thinning is related to poorer neuropsychological performance (higher z-score on Trail Making Test B [TMTB]). In males, all correlations related to improved performance.

#2. ACEs were positively correlated with NSI scores ( $r = 0.66$ ,  $p < 0.05$ ) for the mild-to-moderate TBI ( $GCS > 8$ ). Females were 1.24 ( $p = .05$ ) times more likely to report more ACEs. Sex marginally moderated the association between social determinants of health (SDOH) and PTSD symptoms ( $B = 5.88$ ,  $p = .09$ ).

## Conclusion

Sexual dimorphism necessitates different clinical profiles, targets, and precise rehabilitation strategies in TBI. Higher ACEs correlated with greater neuropsychiatric sequelae and SDOH correlated with more severe PTSD symptoms following mild-to-moderate TBI. Sex moderated the association between SDOH and PTSD symptoms.

## **11:35 - 11:45 Building AI from the Neurosurgical Literature**

**Eric Oermann, MD**

### Introduction

Modern AI technologies including large language models (ChatGPT, Bard), image generators (DALI, Stable Diffusion), and more specialized models utilize web-scale datasets to learn generative models of human language and images. Early investigations show that state-of-the-art language models work well on neurosurgical challenge problems, and USMLE questions at the expense of being large, black-box, commercial products.

### Objectives

Can smaller multi-modal models trained on the neurosurgical literature meet or exceed the performance of large, generalist commercial systems?

### Methods

We converted the entirety of Neurosurgery Publications to a vision-language dataset. We used this new CNS dataset to build a suite of models at the 1-7B parameter range with similar architectures to commercial systems including a neurosurgery language model (CNSGPT), an image embedding model (CNSCLIP), and a multi-modal vision-language model (CNSAVA). We developed our own training pipeline based on open source standards, and utilized a cluster of 24x A100s to train these models in a massively distributed setting for four weeks. Models were evaluated on CNS SANS questions, and in a prospective deployment within a neurosurgical department.

## Results

We demonstrate that these lightweight CNS models are highly performant on neurosurgical language and vision-language tasks with the added benefit of being easily deployable on local hardware, providing interpretable outputs, and are transparent with regards to their datasets and legal considerations.

## Conclusion

Specialist models built by the community and for the community offer a cost-effective, transparent, and performant alternative to current commercial models.

<b>11:45 – 11:55 Early Electroencephalography Biomarkers of Cortical Dysfunction to Predict Long-term Risk of Post-traumatic Epilepsy</b>
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**Matthew Pease, MD**

## Introduction

Up to one-third of severe traumatic brain injury (TBI) patients develop post-traumatic epilepsy (PTE), often years after their injury. Early electroencephalography (EEG) biomarkers may allow for early identification of PTE risk and guidance of clinical trials of anti-epileptogenic therapies.

## Objectives

We explored if the mean absolute deviation (MAD) of the power spectral densities (PSD), a measure of focal cortical dysfunction, predicted long-term risk of PTE in the immediate post-trauma setting.

## Methods

We retrospectively analyzed a prospective database of severe TBI patients treated at a single level one trauma center from 2012 through 2018. We identified a cohort of patients who survived to two years and were outcome matched using age and the Glasgow Outcomes Scale Extended (GOSE). We used continuous EEG collected within the first five days post-trauma. We developed a novel set of EEG features to quantifying focal dysfunction through computing MAD of the PSD of the canonical EEG frequency bands (delta, theta, alpha, beta). The MAD quantifies how variable each band is across the electrodes divided by the variability in all bands. In focal dysfunction, bursts of delta or theta in a group of electrodes increase the variability of delta or theta, which is captured by an increase in the delta MAD. We developed a support vector machine to predict long-term PTE risk using delta and theta MAD, as well as average spectral power.

## Results

We identified 21 patients with PTE and 20 without who survived two years post-injury. The median time to onset of PTE was 7.2 months post-trauma and GOSE was similar when stratified by PTE at 6- and 12-months ( $p>0.73$ ). Validation accuracy was 84%, sensitivity 70%, specificity 86%, and area under the receiving operating curve of 0.80.

## Conclusion

We developed a novel set of early EEG biomarkers to measure focal cortical dysfunction that accurately predicted long-term PTE risk early after trauma.

11:55 - 12:00 Wrap up and Transition

**12:05 - 12:50 Presidential Address**

12:05 - 12:10 Introduction and Recognition of Dr. Korn

12:10 - 12:20 Introduction of the Academy President: Anil Nanda, MD

12:00 - 12:40 Presidential Address: Shenandoah Robinson, MD

**7:30 – 7:35 WELCOMING REMARKS**

Daniel Resnick, MD

**7:35 – 9:00 Peer Reviewed Abstract Session IV: Functional/Epilepsy I**

Moderators: Aviva Abosch, Fernando Vale, Bob Carter

**7:35 – 7:45 Electrophysiological and Metabolic Mechanisms Associated with Dentate Nucleus DBS for Post-stroke Rehabilitation**

André Machado, MD

Introduction

Deep brain stimulation (DBS) of the dentate nucleus (DN) for post-stroke rehabilitation is an emerging indication with promising results. Preclinical and early clinical data were presented at a past Academy meeting

Objectives

Here we combine the clinical outcomes of the first-in-man clinical trial of DN-DBS with its metabolic and electrophysiological mechanistic underpinnings.

Methods

Twelve subjects with moderate to severe post-stroke hemiparesis underwent three months of rehabilitation followed by 4-8 months of DN-DBS combined with rehabilitation. <sup>18</sup>F-fluorodeoxyglucose PET as well as DN local field potentials (LFPs) and cortical electroencephalographic recordings were acquired during motor execution tasks at baseline and post-intervention.

Results

At baseline, perilesional cortical electroencephalography and DN-LFPs event-related oscillations were significantly correlated in the  $\beta$  band. Cortico-cerebellar coherence (CCC) was observed during isometric 'hold' period, also in the  $\beta$  band, and correlated with task accuracy. Following combined DN-DBS and rehabilitation, participants showed statistically and clinically significant improvements in disability, indexed by the Fugl-Meyer Assessment Upper Extremity (FMA-UE), with a median improvement of 15 points among responders. Increments in perilesional desynchronization and decrements in CCC were significantly correlated with motor gains. FDG-PET showed significantly increased metabolic activity across perilesional areas, including the premotor cortex, that were directly correlated with motor improvements.

Conclusion

The Phase I results suggest overall safety and feasibility, with robust clinical improvements. Electrophysiological and metabolic mechanistic investigation supports the effects of DN-DBS on perilesional cortical function and plastic reorganization.

**7:45 – 7:55 Studying the Cellular Building Blocks of Human Language**

Ziv Williams, MD



## Introduction

Humans are capable of conveying exceptionally complex information through language. This capacity to produce and comprehend speech is unique to humans and is often prominently affected by conditions such as stroke, traumatic brain injury and neurodevelopmental disorders. The basic cellular building blocks that underlie human language, however, remain largely unknown.

## Objectives

To study and better understand human language at a basic cellular scale.

## Methods

Here, we developed novel techniques that allowed us to acutely record single neurons from participants undergoing planned intraoperative neurophysiology (Fig. 1a). By following their action potential dynamics and by using a combination of population modeling and decoding techniques (Fig. 1b,c), we characterize the cellular encoding properties of prefrontal cells during language production and comprehension.

## Results

We find neurons in the human prefrontal cortex that encoded detailed information about the phonetic arrangement and composition of planned words during speech production and that reliably predicted their phonetic, syllabic and morphological components during natural language production (Fig. 1d-f). Using comprehension-based tasks (Fig. 2a), we also identify prefrontal neurons that reflect information about complex naturalistic narratives and that reliably encode information about the events, items and the social agents involved across broadly varied linguistic materials (Fig. 2b-d).

## Conclusion

Taken together, these studies reveal a remarkably structured organization of linguistic representations by prefrontal neurons in humans and identify a cellular process that could support the ability of humans to produce and comprehend natural speech—opening the door for further understanding and treating language disorders.

7:55 – 8:05	<b>DBS Distribution of Neural Rhythms Predicts Shifts in Clinical Response in Obsessive-Compulsive Disorder</b>
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Sameer Sheth, MD

## Introduction

DBS for OCD achieves clinical benefit in 66% of treatment-resistant patients. However, there is still a lack of a fundamental understanding of the neurophysiological basis of the relationship between OCD behavior and neural activity.

## Objective

Our goal was to use the continuous neural recording capability of modern DBS devices to better understand the neurophysiological basis of clinical response after DBS for OCD. Given that abnormalities in daily (circadian) periodicity are a cardinal feature of mental health disorders, we hypothesized that changes in the periodicity of neural signals may provide insight into pathological network activity and therefore clinical status.

## Methods

In twelve individuals with treatment-resistant OCD receiving DBS targeted to the ventral striatum (VS), we recorded local field potential power in the alpha-theta (9 Hz) band in continuous 10-minute intervals for

several months before and after DBS. We used model-based and model-free statistical measures, including cosinor and autoregressive model R2 and sample entropy, to quantify neural predictability before and after DBS and its relationship to response status.

### Results

Leveraging >48,000 hours of at-home recordings, we found that 9 Hz VS neural activity is highly periodic in the symptomatic state. Predictability of this signal decreased after DBS initiation and distinguished clinical responders from non-responders. Across all patients, the distributions of each of the four output measures significantly differed between the symptom burdened state and the symptom unburdened state ( $p < 10^{-2}$ ). Linear and non-linear autoregressive R2 measures enabled accurate classification of clinical status from neural data with a balanced accuracy of 82% and 84%, respectively, corresponding to an area under the receiver operating characteristic curve of 85% and 89%, respectively.

### Conclusion

These results reveal a reliable neurophysiological biomarker corresponding to clinical response in OCD. Such a biomarker could be used to guide therapeutic decision-making or as a control signal for adaptive DBS.

8:05 – 8:15	<b>Language Experience Drives Phonological and Word Specialization in Human Temporal Lobe</b>
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**Edward Chang, MD**

### Introduction

The world's 7000 spoken languages share the important characteristic that they are all produced with the same set of vocal articulators. While there are differences in the specific inventories of speech sounds across languages, the basic acoustic properties of these sounds are largely shared. Yet without experience, listeners are unable to interpret these sounds as meaningful phonological units like words.

### Objectives

Our objective was to determine the shared and language-specific properties of speech are encoded in the human brain depending on language experience.

### Methods

We recorded direct high-density electrocorticography (ECoG) while participants passively listened to natural speech in their native language and a language that was unfamiliar to them.

### Results

We found that native and unfamiliar languages elicited significant responses to speech in the same cortical sites throughout the temporal lobe, and further, that tuning to broad acoustic-phonetic classes was consistent across native and unfamiliar speech conditions. Language-experience dependent activity emerged specifically for encoding sequences of speech sounds and for identifying where words begin and end during continuous speech.

### Conclusion

Together, this study demonstrates what is shared and different in the STG processing of speech across different languages. These results support a model of human speech processing wherein neural representations in the temporal lobe combine language-agnostic acoustic-phonetic features and language-specific sequence and word level information.

**8:15 – 8:25      Staged, Bilateral Focused Ultrasound Thalamotomy in Essential Tremor**

**Vibhor Krishna, MD**

Introduction

Unilateral focused ultrasound ablation of the ventral intermediate nucleus of the thalamus (Vim) for essential tremor reduces contralateral tremor. However, the untreated side tremor or midline symptoms limit the quality of life for some patients. Historically, bilateral lesioning caused unacceptable risks and was abandoned to be replaced by deep brain stimulation. With the increasing acceptance of unilateral focused ultrasound ablation, the interest in testing bilateral lesioning was renewed.

Objectives

To evaluate the safety and efficacy of staged, bilateral focused ultrasound thalamotomy in essential tremor patients successfully treated with unilateral Vim thalamotomy.

Methods

A prospective, open-label, multicenter trial recruited patients from July 2020 to October 2021 at seven sites in the United States. Essential tremor patients with medication-refractory tremors who had undergone unilateral focused ultrasound thalamotomy at least nine months before enrollment were eligible. The primary efficacy outcome was tremor score (clinical rating scale for tremor subscale A and B) at three months for the treated side. Secondary outcomes included postural tremor and tremor-related disability. The primary safety endpoint was the incidence and severity (mild, moderate, or severe) of device- and treatment-related adverse events. A speech and language pathologist assessed speech and swallowing function.

Results

Sixty-two subjects were enrolled, and 51 treated (mean age: 73 years, SD: 13.9; 86.3% male). The mean tremor score improved from 17.4 (SD:5.4) to 6.4 (SD:5.3) at 3 months (66% improvement, 95% CI - 59.8% to 72.2%,  $p<0.001$ ). There was significant improvement in postural tremor (2.5 [SD:0.8] to 0.6 [SD:0.9],  $p<0.001$ ) and mean disability score (10.3 [SD:4.7] to 2.2 [SD:2.8],  $p<0.001$ ). Twelve subjects developed mild ataxia, which persisted in six subjects at 12 months. Other adverse events included numbness/tingling (n=17 total, n=8 at 12 months), dysarthria (n=15 total, n=7 at 12 months), unsteadiness/imbalance (n=10 total, none at 12 months), and taste disturbance (n=7 total, n=3 at 12 months). The speech difficulties including phonation, articulation, and dysphagia, were mostly mild and transient.

Conclusion

Staged, bilateral focused ultrasound thalamotomy was safe and significantly reduced tremor severity and functional disability scores. Adverse events for speech, swallowing, and ataxia were mostly mild and transient.

**8:25 – 8:35      Focused Ultrasound Neuromodulation as a Novel Neurosurgical Treatment for Opioid and Substance Use Disorder**

**Ali Rezai, MD**

Introduction

The addiction crisis continues to be a health care challenge evidenced by >110,000 drug overdose deaths in the US in 2023. Despite advances in medication and behavioral treatments, success rates remain low. Novel

therapeutic strategies such as neuromodulation are needed to address the substance use disorder (SUD) epidemic. Focused ultrasound (FUS) neuromodulation is a novel neurosurgical innovation with great potential for the treatment of neurological and behavioral conditions. We initiated a first-in-human FDA and National Institute on Drug Abuse (NIDA) sponsored study to evaluate nucleus accumbens (NAc) FUS neuromodulation for severe and treatment-resistant SUD.

#### Objectives

To evaluate the safety and efficacy of nucleus accumbens (NAc) FUS neuromodulation for severe and treatment-resistant SUD.

#### Methods

This prospective, open-label study enrolled participants with severe, primary opioid and co-occurring SUD. Participants underwent one 20-minute FUS treatment of bilateral NAc using an MRI-guided low-intensity 220 kHz FUS system (Insightec). Safety, tolerability, feasibility, and effects of FUS were assessed by evaluating adverse events, substance craving, substance use (urine toxicology), mood/anxiety, and anatomical/functional MRI throughout 90-days post-FUS treatment.

#### Results

Eight participants with severe treatment-resistant SUD received bilateral NAc FUS. There were no serious adverse events or MRI abnormalities. Post-FUS, participants had an immediate and persistent reduction (91%) in cravings for multiple substances including opioids, amphetamine, cocaine, and alcohol. Seven participants remained completely abstinent at 30 days, and five at 90-days post-FUS. All participants had improvements in depression, anxiety, behavioral and psychosocial functioning. Functional MRI demonstrated decrease in connectivity in the reward neurocircuitry and cognitive control systems.

#### Conclusion

Bilateral NAc FUS neuromodulation is safe and well-tolerated. One FUS treatment resulted in immediate and sustained (through 90 days) reduction of craving and use of opioids and other substances. FUS is a novel therapeutic strategy for severe SUD. Future sham-controlled, randomized studies in a larger sample of participants are warranted.

<b>8:35– 8:45      Deep Brain Stimulation of the Fornix Selectively Disrupts Memory Encoding</b>
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**Wael Assad MD**

#### Introduction

Alzheimer's disease (AD) is a debilitating neurodegenerative disorder with a high burden of care and limited treatment options. Given the long history of inadequate pharmacologic therapies, the possibility of invasive brain stimulation to restore memory function was explored through stimulation of the circuit of Papez via the fornix. However, whether such stimulation might produce acute, item-specific, memory enhancement is unknown.

#### Objectives

We examined the effect of deep brain stimulation of the fornix (DBS-f) on memory in patients with mild AD enrolled locally in the prospective, multi-center ADvance II trial. We sought to assess the specific effects of DBS-f on memory encoding, apart from potential effects on attention, working memory, or other relevant functions.

## Methods

Subjects undertook a parametric, visual-spatial memory task in two settings: intra-operatively and 4 weeks post-operatively. The task required subjects to encode the location of a dot (memorandum) displayed in the periphery of a 5-second movie clip. The task included an immediate-report phase (to assess attention and working memory) and a subsequent delayed recall phase (to assess more durable memory encoding/recall). Four stimulation conditions were assessed: OFF, LOW (40 Hz), HIGH (130 Hz) and THETA (6 Hz; +/- theta burst). Stimulation was delivered during the encoding phase. Data were analyzed using both bootstrap statistics and Bayesian modeling.

## Results

Contrary to the hope that DBS-f might have acute beneficial mnemonic effects, memory encoding was impaired at a range of stimulation conditions, especially HIGH. Importantly, attention, working memory, and visual-motor function were spared, as revealed by preserved immediate report performance despite stimulation.

## Conclusion

This rigorous assessment of acute memory effects of DBS-f suggests that 1) open-loop fornix stimulation has a specific, possibly dose-dependent, disruptive effect on memory encoding, and 2) any potential benefit of DBS-f in AD would need to rely upon more chronic neuromodulatory mechanisms.

8:45- 8:40	Wrap up and Transition
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8:50 - 10:00	<b>Peer Reviewed Abstract Session V: Cerebrovascular I</b> Moderators: Peter Vajkoczy, Judy Huang, Peter Kan
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8:50 - 9:00	<b>Results of the COMMAND Trial, The First FDA Approved Study of a Novel Transvascular Brain Computer Interface</b>
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J Mocco, MD

## Introduction

We will present the results of the COMMAND trial, an FDA approved early feasibility study with the Synchron motor neuroprosthesis. The Synchron motor neuroprosthesis is intended for subjects with severe permanent motor impairment and persistent functioning motor cortex. The device transmits cerebral cortex neural signals externally, via a standard Bluetooth signal, to control digital devices.

## Objectives

To evaluate the safety and feasibility of a novel transvascular brain computer interface.

## Methods

FDA approval was obtained to enroll up to six patients, aged 21 to 75 yrs old, with severe upper limb paralysis in order to assess the safety and feasibility of the Synchron motor neuroprosthesis. The primary endpoint was serious adverse events (SAEs) resulting in death or permanent increased disability during the one-year post-implant evaluation period. The secondary endpoint was rate of sinus occlusion/stenosis or device migration. Pre-specified secondary outcomes included time to device activation and successful transmission, performance rate on motor signal transmission test, and QOL outcomes.

## Results

Six patients were enrolled. No SAEs resulting in death or permanent increased disability were encountered to date, with two patients remaining to complete follow up. At the time of presentation at the Academy all patients will have reached the one year follow up endpoint and final data will be presented. No occurrences of sinus occlusion/stenosis or device migration have occurred to date. Pre-specified secondary outcomes data will be presented.

## Conclusion

The Synchron motor neuroprosthesis demonstrates early indications of safety and potential clinical benefit for patients with severe upper limb paralysis.

<b>9:00– 9:10</b>	<b>Changes in Circulating Biomarkers Reflect Changes in Iron Content and Permeability in Cerebral Cavernous Malformations</b>
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Issam Awad, MD

## Introduction

An increase in mean lesional iron content (>6%), measurement by quantitative susceptibility mapping (QSM), and/or vascular permeability (>40%), assessed by dynamic contrast enhanced quantitative perfusion (DCEQP) on MRI, have been associated with new symptomatic hemorrhage (SH) in cerebral cavernous malformations (CCM).

## Objectives

Circulating proteins and metabolites have been associated with hemorrhagic activity of CCMs, but prospective changes in levels of these molecules have not been compared to changes in QSM and DCEQP. Plasma samples and lesional QSM and DCEQP were simultaneously acquired at the beginning and end of 60 one-year epochs of prospective follow-up in 46 CCM patients with SH in the prior year enrolled in the NIH multicenter Trial Readiness (U01 NS104157), and are correlated herein.

## Methods

Plasma levels of 16 proteins and 12 metabolites previously associated with CCM hemorrhage were assessed by ELISA and liquid-chromatography mass spectrometry, respectively. Multiomic combinations of plasma levels of proteins and metabolites reflecting QSM and/or DCEQP changes were selected based on the sum of squared error (SSE) from LOOCV, accuracy (sensitivity/specificity on receiver operating curves), and the biomarker's error rate.

## Results

A combination of the relative changes in plasma levels of 3 proteins (ROBO4, CD14, thrombomodulin) and 1 metabolite (acetyl-L-carnitine) reflected a mean increase in QSM>6% (97.2%/100% specificity/sensitivity,  $p=3.1e-13$ ). A combination of relative changes in plasma levels of endoglin and 3 metabolites (pipercolic, arachidonic acid and hypoxanthine) correlated with an increase in mean DCEQP >40% (99.6%/100% specificity/sensitivity,  $p=4.1e-17$ ).

## Conclusion

Changes of plasma levels of proteins and metabolites reflect with great accuracy the changes in lesional iron content and permeability during prospective follow-up of CCMs with recent SH. Results have mechanistic implications, and provide a proof of concept that blood tests could replace more complex and costly imaging biomarkers in monitoring of CCM hemorrhage, and as secondary outcomes in clinical trials.

**9:10 – 9:20      mTORC1 Inhibitor Rapamycin Inhibits Growth of Cerebral Cavernous Malformations in Adult Mice**

**Jan-Karl Burkhardt, MD**

Introduction

Cerebral cavernous malformations (CCMs) are vascular malformations that frequently cause stroke. CCMs arise due to loss of function in one of the genes that encode the CCM complex, a negative regulator of MEKK3-KLF2/4 signaling in vascular endothelial cells. Gain-of-function mutations in PIK3CA (encoding the enzymatic subunit of the PI3K (phosphoinositide 3-kinase) pathway associated with cell growth) synergize with CCM gene loss-of-function to generate rapidly growing lesions.

Objectives

To establish and test a faithful adult CCM model replicating progressive human CCMs and to test treatment effects of medications preclinically in preparation for clinical trials.

Methods

We recently developed a model of CCM formation that closely reproduces key events in human CCM formation through inducible CCM loss-of-function and PIK3CA gain-of-function in mature mice. In the present study, we use this model to test the ability of rapamycin, a clinically approved inhibitor of the PI3K effector mTORC1, to treat rapidly growing CCMs.

Results

We show that both intraperitoneal and oral administration of rapamycin arrests CCM growth in mice (Figure 1-3), reduces perilesional iron deposition, and improves vascular perfusion within CCMs.

Conclusion

Our findings further establish this adult CCM mouse model as a valuable preclinical model and support clinical testing of rapamycin to treat rapidly growing human CCMs.

**9:20 – 9:30      Racial & Ethnic Disparities in Treatment & Outcomes of Unruptured Intracranial Aneurysms: An NVQI-QOD Analysis**

**Kevin Cockroft, MD**

Introduction

Racial disparities are commonplace in modern medicine. We suspected that such disparities are likely in the care of patients with unruptured intracranial aneurysms (UIAs).

Objectives

Our objective is to evaluate racial differences in aneurysm characteristics, treatments, and outcomes in patients undergoing treatment of UIAs.

Methods

NVQI-QOD registry was queried for patients who underwent treatment of UIA. Comparisons of demographics, aneurysm characteristics, treatments, and outcomes were made across races. Due to low sample sizes in several race groups, non-Hispanic Whites (NHW) were compared with all other races combined, defined as Black, Hispanic, and other non-Whites (BHNW). Multivariate logistic regression was performed to control for known confounders.

## Results

3042 UIA treatments were analyzed, consisting of 74.4% NHW, 12.1% Black, 7.6% Hispanic, 3.6% Asian, 1.7% American Indian, and 0.4% Pacific Islander patients. NHW patients were less frequently symptomatic (23.9% versus 33.2%,  $p < 0.0001$ ), and less likely to have open surgery (14.4% versus 20.4%,  $p < 0.0001$ ). There was no significant difference in intra-operative complication rates. NHW patients were less likely to have post-operative complications (4.3% versus 7%,  $p = 0.005$ ). Patients had similar rates of aneurysm occlusion. NHW patients were less likely to have a modified Rankin score (mRS) of  $>3$  at discharge (7.8% versus 11.2%,  $p = 0.02$ ), length of stay (LOS)  $>3$  days (24.4% versus 35.5%,  $p < 0.0001$ ) and nursing home discharge or death (2.8% versus 1.5%,  $p = 0.015$ ). After controlling for known confounders, BHNW patients had a higher rate of post-operative complications (OR 1.65, 95% CI 1.09-2.46,  $p = 0.016$ ), modified Rankin score (mRS) of  $>3$  at discharge (OR 1.72, 95% CI 1.14-2.57,  $p = 0.009$ ), LOS  $>3$  days (OR 1.5, 95% CI 1.19-1.87,  $p < 0.001$ ) and poor discharge status (OR 2.29, 95% CI 1.21-4.29,  $p = 0.01$ ).

## Conclusion

Analysis of the NVQI-QOD registry indicates significant racial disparities in aneurysm characteristics, treatment modalities and outcomes in patients undergoing treatment of UIAs in the United States.

<b>9:30– 9:40</b>	<b>Auricular Vagus Nerve Stimulation Reduces Inflammation &amp; Vasospasm In Subarachnoid Hemorrhage: A Single-Center RCT</b>
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**Eric Leuthardt, MD**

## Introduction

Inflammation contributes to morbidity following subarachnoid hemorrhage (SAH). Transauricular vagus nerve stimulation (taVNS) offers a noninvasive approach to target the inflammatory response following SAH.

## Objectives

The primary aims of this trial were to determine if taVNS following SAH reduces TNF- $\alpha$  in the plasma and CSF, and reduces the rate of radiographic vasospasm.

## Methods

In this prospective, triple-blinded, randomized, controlled trial, twenty-seven patients were randomized to taVNS or sham stimulation. Blood and cerebrospinal fluid (CSF) were collected to quantify inflammatory markers. Cerebral vasospasm severity and functional outcomes (modified Rankin Scale, mRS) were analyzed.

## Results

No adverse events occurred. Radiographic vasospasm was significantly reduced ( $p = 0.018$ ), with serial vessel caliber measurements demonstrating a more rapid return to normal than sham ( $p < 0.001$ ). In the taVNS group, TNF- $\alpha$  was significantly reduced in both plasma (days 7 and 10) and CSF (day 13); IL-6 was also significantly reduced in plasma (day 4) and CSF (day 13) ( $p < 0.05$ ). Patients receiving taVNS had higher rates of favorable outcomes at discharge (38.4% vs 21.4%) and first follow-up (76.9% vs 57.1%), with significant improvement from admission to first follow-up ( $p = 0.014$ ), unlike the sham group ( $p = 0.18$ ). The taVNS group had a significantly lower rate of discharge to skilled nursing facility or hospice ( $p = 0.04$ ).



### Conclusion

Transauricular VNS is a non-invasive method of neuro- and systemic immunomodulation. This trial supports that taVNS following SAH can mitigate the inflammatory response, reduce radiographic vasospasm, and potentially improve functional and neurological outcomes.

<b>9:40 – 9:50</b>	<b>Endovascular Aneurysm Treatment: Computational Modeling using Lagrangian Platelet Tracking Techniques</b>
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**Michael Levitt, MD**

### Introduction

Predicting the outcome of endovascular treatment of cerebral aneurysms using computational fluid dynamics (CFD) simulations has shown promise, but not yet reached clinical practice due to methodological uncertainties. Traditional CFD focuses only on the effect of blood flow on the vessel (or aneurysm) wall, but does not account for blood components such as platelets, which may mediate aneurysm thrombosis after endovascular treatment.

### Objectives

The goal of this work is to simulate the behavior of platelets in the setting of endovascular aneurysm treatment.

### Methods

Patient-specific anatomy was derived from segmentation of rotational angiography, and boundary conditions (blood flow velocity and blood pressure) were recorded from each patient using a dual-sensor endovascular microwire. We applied particle-tracking methods (Lagrangian reference frame CFD) to the simulation of cerebral aneurysms before and after endovascular treatment with either embolic coils or flow-diverting stents. Thousands of massless particles acting as platelet surrogates were placed into the simulations, with additional particles added from the inlet for each cardiac cycle. Each particle's trajectory, residence time (RT) within the aneurysm domain, and shear history (SH; defined as accumulated shear stress over time) was recorded.

### Results

A total of 22 patients with unruptured aneurysms (17 treated with embolic coils, 5 treated with flow-diverting stents) were studied. Lagrangian particle tracking was successfully simulated in all cases before and after treatment. In both treatment groups, the post-treatment simulations resulted in reductions in platelet entry into the aneurysm, qualitative changes in platelet trajectories, and significantly increased RT and decreased SH within the aneurysm dome, suggesting stagnant flow.

### Conclusion

The application of Lagrangian particle tracking techniques in CFD simulations of cerebral aneurysms before and after treatment offers novel insights into the behavior of blood flow not captured in traditional CFD metrics. These insights may be useful in predicting endovascular treatment outcome.

<b>9:50 – 10:00</b>	<b>Performance of a Transcranial Bioadhesive Ultrasound Patch in Human Volunteers</b>
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**Daniel Newell, MD**

### Introduction

Accurate and continuous monitoring of cerebral blood flow is valuable for clinical and neurocritical care and research. Transcranial Doppler (TCD) ultrasonography is a widely used non-invasive method for

evaluating the cerebral vasculature and blood flow, but the single beam and duplex probe design limits the measurement accuracy of the complex three-dimensional (3D) vascular networks and the practicality for prolonged recording.

### Objectives

The objective was to test a conformal ultrasound patch prototype for hands-free volumetric imaging of the circle of Willis and for examination and continuous monitoring of cerebral blood flow in normal human volunteers.

### Methods

Ultrafast ultrasound imaging using a bioadhesive patch with 240 ultrasound elements was used to accurately render the circle of Willis in 3D and minimize human errors during vessel examinations through the established transcranial windows.

### Results

The accuracy of the conformal ultrasound patch was compared with a conventional TCD probe on 36 participants, showing a mean difference and standard deviation of difference as minus;  $1.51 \pm 4.34$  cm/s<sup>-1</sup>,  $-0.84 \pm 3.06$  cm/s<sup>-1</sup>, and  $-0.50 \pm 2.55$  cm/s<sup>-1</sup> for peak systolic velocity, mean flow velocity and end diastolic velocity, respectively. The ultrasound patch was used to measure (using the temporal window) MCA and PCA flows, which dominate the supply of blood to the brain. The motion tolerance of the device was determined to be within about  $\pm 20$  degrees with head roll, yaw and pitch.

### Conclusion

Improved accuracy and stability of recording of blood flow spectra at selected locations continuously can offer many advantages for neurovascular diagnostics and care.

**10:00 – 10:20 Break**

**10:20 – 11:00 Special Session**

**10:20 – 10:25 Introduction of Dr. Levenson: Shenandoah Robinson, MD**

**10:25 – 10:55 Dr. Levenson**

**10:55 – 11:00 Wrap up and Transition**

**11:10 – 12:45 Peer Reviewed Abstract Session VI: Tumor II**

**Moderators: Ganesh Rao and Linda Liau**

**11:10 – 11:20 Long-Term Prospective Quality-of-life Outcomes In 445 Patients with Sporadic Vestibular Schwannoma**

**Michael Link, MD**

### Introduction

Quality-of-life data offer insights into nuanced, and often less tangible aspects of the patient care experience that are frequently overlooked by physical examination and other traditional ‘objective’ diagnostic tests.

Quality-of-life data also contextualize these traditional outcome measures and their true impact on daily life in a way that challenges traditional medical viewpoints.

### Objectives

To evaluate the long-term changes in sporadic vestibular schwannoma (VS) disease-specific quality-of-life (QOL) outcomes.

### Methods

Prospective longitudinal study using the Penn Acoustic Neuroma Quality of Life (PANQOL) Scale. The current report summarizes QOL outcomes for all subjects diagnosed with sporadic VS who completed a baseline survey before treatment and at least one follow-up survey after treatment. Eligible subjects were recruited through the Mayo Clinic Rochester clinical practice and the Acoustic Neuroma Association.

### Results

A total of 445 patients were eligible for study with a mean duration of follow-up of 4.4 (SD 2.3) years, including 122, 218, and 105 in the observation, microsurgery, and radiosurgery groups, respectively. Patients managed with observation ( $p=0.03$ ) or microsurgery ( $p<0.001$ ) demonstrated improvement in anxiety scores. Changes in facial function scores differed significantly by management group ( $p=0.01$ ), with patients undergoing microsurgery demonstrating a mean decline of 10 points in facial function scores compared with mean declines of 3 for those managed with observation or radiosurgery. Hearing loss scores decreased similarly over time for all three groups ( $p=0.3$ ). There were minimal changes in total PANQOL scores over time across all management groups ( $p=0.5$ ).

### Conclusion

Long-term changes in total QOL among VS management groups are not significantly different. Microsurgery may continue to confer an advantage regarding anxiety, presumably due to the benefit of a 'cure' but with a greater decline in facial function when compared to observation or radiosurgery. Long-term declines in hearing loss scores were not statistically significant among groups.

<b>11:20 – 11:30 Novel Oncomagnetic Treatment of GBM and DIPG-Bench to Beside Studies with Update Rx of 11 Patients</b>
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David Baskin, MD

### Introduction

We have developed a novel technology to treat GBM and DIPG utilizing oscillating magnetic fields (OMF) to kill tumor cells. The mechanism of action is to increase mitochondrial reactive oxygen species (ROS) to toxic levels. Normal brain cells have low ROS levels and high antioxidant defenses and are not affected by this therapy. This presentation provides updates regarding our work.

### Objectives

We have expanded our bench to bedside studies to demonstrate precise mechanisms of action utilizing a number of cell survival assays, flow cytometry, antioxidant blockage, and RNA sequencing. Studies have included syngeneic and immune PDX mouse models, and treatment in 11 patients.

### Methods

We delineated mechanisms of actions in cell culture studies assessing for reactive oxygen species and caspase 3 expression. Clonogenic assays, cell survival studies, flow cytometry, and RNA sequencing were utilized. Efficacy was assessed using syngeneic and PDX mouse models and expanded access treatment in 9 GBM and two DIPG patients.

## Results

Cell culture studies documented elevation of ROS leading to apoptotic cell death. Optimal oscillating frequencies and on/off parameters were determined. Six hours per day of treatment were sufficient for optimal results. Effects were demonstrated in both syngeneic and PDX mouse glioma using 7T MRI scans. 9 endstage GBM patients and two DIPG patients were treated using a removable helmet, six hours a day. No head shave or electrodes were required. Patients received treatment in two hour blocks, thrice daily. Dramatic reduction in contrast enhancement and improvement in OS were seen in 7/9 GBM patient and 1 DIPG patient. No adverse events occurred.

## Conclusion

OMF is a powerful and disruptive technology for treatment of GBM. IDE studies in the US and clinical trials in Europe are underway to further study this potentially powerful technique, which may reduce the need for chemoradiation and resulting toxicity.

## **11:30 – 11:40 Result of a Phase 1 Trial Evaluating the Use of Vascularized Pericranial Flap on the Resection Cavity of Glioblastoma**

**John Boockvar, MD**

### Introduction

Transposition of vascularized temporoparietal fascial flaps (TPFF) or peri-cranial flaps (PCF) along a GBM resection cavity offers a mechanism of bypassing the blood-brain-barrier (BBB).

### Objectives

We conducted a first-in-human Phase I trial assessing the safety of lining the resection cavity with autologous TPFF/PCF of newly diagnosed patients with GBM.

### Methods

12 patients underwent safe, maximal surgical resection of newly diagnosed GBMs, followed by lining of the resection cavity with a pedicled, autologous TPFF or PCF. Safety was assessed by monitoring adverse events. Secondary analysis of efficacy was examined as the proportion of patients experiencing progression-free survival (PFS) as indicated by response assessment in neuro-oncology (RANO) criteria and overall survival (OS).

### Results

A total of 12 patients undergoing the above-mentioned procedure between November 2018 and November 2022 were included in the study with a median age of 57 years, mean tumor volume and mean follow-up of 56.6 cm<sup>3</sup> and 23.2 months, respectively. All the patients had undergone gross total resection. Grade I to III adverse events were encountered in 3 patients without any Grade IV or V serious adverse events. Disease progression at the site of the original tumor was identified in only 4 (33%) patients (median 23 months), 3 of whom underwent re-resection which showed robust immune infiltrates within the transplanted flap without any evidence of tumor infiltration into the implanted flap. At the time of this manuscript preparation, only 4/12 (33%) of patients have died. A total of 10 patients (83.3%) had 6-month PFS. The median PFS and OS were 9.10 and 17.6 months respectively. 33% of patients have lived for more than two years and the longest survivor currently is alive at 60 months.

### Conclusion

This pilot study suggests that insertion of pedicled autologous TPFF/PCF along a GBM resection cavity is safe and feasible.

**11:40 – 11:50 Multiplicative Impact of Specific Somatic Copy Number Alterations on Meningioma Recurrence Risk**

**Jennifer Moliterno, MD**

Introduction

Somatic copy number variations (SCNAs), involving significant chromosomal aberrations, are prevalent in meningiomas, and contribute to aggressive behavior and recurrence.

Objectives

We aimed to investigate the impact of SCNAs on meningioma recurrence, examining their patterns of mutual exclusivity and co-occurrence.

Methods

After classifying meningiomas into molecular subtypes, we performed univariate and multivariate Cox proportional hazards regression analysis to identify SCNAs associated with recurrence, followed by fitting the regression model with the number of SCNAs as factor covariates to identify events that further increase the risk of recurrence (multiplicative interaction).

Results

After quality control, we included data from 334 meningiomas (Fig 1). The univariate analysis revealed that there were five SCNAs that were associated with recurrence risk (adjusted  $P < 0.05$ ): chromosome 10qLOSS, 11pLOSS, 2pLOSS, 14qLOSS and 18qLOSS. All five SCNAs increased the risk of recurrence, and the accumulation of multiple events further increased this risk, such that a single SCNA tripled the risk, whereas two out of five co-occurring risk SCNAs increased the chance of a recurrence to more than ninefold ( $P = 3.6e-05$ ) (Fig. 2). No tumor harbored all five risk SCNAs but meningiomas with four co-occurring events had a recurrence risk of more than 22-fold (Table 1).

Conclusion

Accumulation and specific combinations of five risk SCNAs significantly raised the likelihood of meningioma recurrence. While chr1pLOSS was not sufficient for recurrence, it was necessary, suggesting that it triggers chromosomal instability leading to accumulation of these newly identified five risk SCNAs. Our findings have significant implications to predict recurrence in meningiomas, regardless of their grade.

**11:50 – 12:00 Multiomic and Clinical Analysis of Multiply Recurrent Meningioma Reveals Risk Factors and Insights into Evolution**

**Albert Kim, MD**

Introduction

Although meningiomas, the most common primary brain tumor, are often effectively treated with surgery and radiation, an important subset of meningiomas behave aggressively and are characterized by treatment resistance and multiple recurrences. Whether multiply recurrent meningiomas (MRMs) are molecularly distinct from non-recurrent meningiomas at initial diagnosis and whether the molecular features of MRMs evolve with subsequent recurrences are fundamental questions that have not yet been addressed.

Objectives

To identify clinical and molecular features associated with MRMs and determine if longitudinal molecular changes occur in paired MRM samples.

## Methods

In this dual institution study, clinical parameters from the medical record were collected for 1315 meningioma patients. After propensity score matching, 31 were identified as multiply recurrent and 84 as nonrecurrent. Whole exome sequencing was performed on 44 meningiomas, EPIC bead chip methylation array on 43, and RNA-sequencing on 66.

## Results

On multivariable binomial logistic regression, MRMs were significantly associated with male sex ( $P=0.012$ ), subtotal resection ( $P=0.001$ ), higher number of meningiomas on presentation ( $P=0.017$ ), and histopathological sheeting ( $P=0.002$ ). Multiomic analysis of primary meningiomas revealed MRMs exhibit greater global copy number alternations (CNA) ( $P=0.0113$ ) and increased DNA methylation ( $P=0.0236$ ). Integrated methylation profiling and RNA-sequencing identified candidate driver genes of MRMs. Among these genes, we demonstrate in meningioma cells that knockdown of EDNRB, a locus with higher promoter methylation and decreased gene expression in MRMs, leads to increased cell proliferation. CNA, subclonal evolution, and methylation profiles of MRMs did not significantly change from primary tumor to recurrence, even after radiation treatment, suggesting MRMs are molecularly aggressive from initial diagnosis.

## Conclusion

We identify several novel clinical and molecular risk factors associated with MRMs. MRMs harbor unique molecular features on presentation, which do not appear to change during evolution and after treatments. Findings from this study hold implications for the development of biomarkers and therapeutic agents for these challenging tumors.

<b>12:00 – 12:10 Intraoperative Navigation with Virtual Cutting Guides Facilitates En Bloc Resection of Primary Bone Tumors of the Spine</b>
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**Laurence Rhines, MD**

## Introduction

En bloc resection with negative margins may prevent local recurrence and improve survival in patients with primary spinal malignancies. These surgeries are challenging due to the complex anatomy and nearby vital structures. Using pre-planned virtual cutting guides to perform navigated osteotomies may be a reliable method for safely obtaining tumor-free surgical margins.

## Objectives

Detail the technique and present short-term outcomes.

## Methods

Patients who underwent en bloc resection of the spine using virtual cutting guides were retrospectively analyzed. Segmentation, delineating tumor from normal tissue, was performed from pre-operative CT and MRI scans and used to generate a computer-assisted design (CAD) model of the tumor and local anatomy. Virtual surgical planning was performed, creating osteotomy planes on the 3-D CAD model. During surgery, this model was loaded onto the navigation system and fused with the intraoperative CT. The pre-planned osteotomy planes were then visualized as “virtual cutting guides” during the real-time stereotactic navigation. An ultrasound-powered cutting tool was then integrated into the navigation system and used to perform the osteotomies.

## Results

Thirteen patients were included with six chondrosarcomas, four chordomas, two osteosarcomas, and one high-grade sarcoma. Negative margins were achieved in all patients. There was one intraoperative complication involving nerve injury during dissection, unrelated to the osteotomies. Mean follow-up was  $19.4 \pm 17.3$  months. Six patients had postoperative complications including infection (n=2), seroma (n=1), wound dehiscence (n=1), pulmonary embolism (n=1), and S1 stress fracture (n=1).

## Conclusion

Using virtual cutting guides to perform navigated osteotomies is a safe technique that can facilitate complex spine tumor resections.

## 12:10 – 12:20 CAR T- Cell Therapy: Targeting Glioblastoma and Immunosuppressive Cells in the Tumor Microenvironment Simultaneously

Alfredo Quinones-Hinojosa, MD

## Introduction

Glioblastoma Multiforme (GBM), the most common and devastating primary brain cancer, resists standard of care. Gene therapy, anti-angiogenics, and immune checkpoint inhibitors (ICI) struggle to gain FDA approval and failed for GBM. Adoptive T-cell immunotherapy; chimeric antigen receptor (CAR) T-cell therapy has shown promise. Success is hampered by antigen diversity, and immunosuppressive tumor microenvironment (TME). While these can be mitigated through tumor-infiltrating lymphocytes (TILs), the GBM TME impairs their functionality.

## Objectives

We aimed to develop and evaluate the efficacy of MC9999 (CAR) T cells targeting PD-L1, sourced from healthy donors and GBM patients.

## Methods

We developed novel MC9999 CAR-T cells targeting PD-L1 from healthy donors and GBM patients. These were tested in vitro and in vivo against patient-derived primary lines from our Biobank and TME immunosuppressive cells from patients. We have performed xCELLigence impedance assays, degranulation assays and ELISA to measure granule components (e.g., granzyme B). We have assayed MC9999 CAR-T cells in vivo in mice bearing tumors via intravenous and intratumoral delivery and compared them to non-armored T-cells and controls (n=15/group). For antigen analysis after treatment, we used Akoya 6-plex staining. To elucidate molecular mechanisms, we performed Sc-RNA sequencing in mice infused with CAR-T cells and non-CAR controls.

## Results

Our novel MC9999 CAR is highly specific and can effectively target primary GBM lines and tumor associated macrophages in vitro. Achieving complete tumor remission in vivo after intravenous and locoregional infusion of MC9999 CAR-T cells without recurrence in over 150 days in patient derived GBM cell lines and commercial cell lines in repeated experiments. These results are statistically significant compared with non-CAR-T cells and controls (PBS vs MC9999 CAR T:  $p=0.0043$ , non-CAR vs MC9999 CAR-T:  $p=0.0031$ ).

## Conclusion

Our novel MC9999 CAR-T is specific against PD-L1 and can efficiently target and eradicate GBM and its microenvironment making it a strong candidate for clinical trial studies.

**12:20 – 12:30 Longitudinal Molecular Evolution of IDH-wildtype Glioblastoma**

**Mitchel Berger, MD**

Introduction

Glioblastoma remains a molecularly heterogeneous disease in need of better predictive biomarkers and more efficacious therapies.

Objectives

To investigate how longitudinal molecular evolution of glioblastoma drives tumor progression and treatment resistance.

Methods

Comprehensive histopathologic, genomic, and epigenomic profiling of paired initial and recurrent IDH-wildtype glioblastoma samples from 106 patients was correlated with clinical outcome data.

Results

Most glioblastomas (91%) demonstrated genetic evolution and/or epigenetic subclass shifting between primary and recurrent tumors. TERT promoter mutation and CDKN2A homozygous deletion were uniformly shared between initial and recurrent tumors, indicating these are fundamental early events in gliomagenesis, whereas alterations involving EGFR, PDGFRA, PTEN, NF1, and TP53 were commonly private to initial or recurrent tumors, indicating acquisition later during clonal evolution. 11% of glioblastomas developed temozolomide-induced hypermutation at recurrence, and these patients had longer overall survival. Higher DNA methylation levels at 4 specific CpG sites in the MGMT promoter was predictive for developing temozolomide-induced hypermutation. Moreover, 17% of glioblastomas underwent sarcomatous transformation at recurrence, which were highly enriched for NF1 inactivation and mesenchymal epigenetic subclass. Unlike IDH-mutant astrocytomas which uniformly become more globally hypomethylated at recurrence, IDH-wildtype glioblastomas were heterogeneous with subsets becoming more globally hypermethylated, hypomethylated, or relatively stable. Finally, we developed a DNA methylation evolution signature that significantly correlated with clinical outcomes for patients with IDH-wildtype glioblastoma.

Conclusion

Nearly all glioblastomas undergo genomic and epigenomic evolution. Genomic analysis at time of recurrence can reveal acquired treatment resistance mechanisms (EGFR variant switching, novel MGMT gene amplification) that may impact therapeutic decision making.

**12:30 – 12:40 CARv3-TEAM-E T Cells for Recurrent Glioblastoma**

**Bryan Choi, MD, PhD**

Introduction

Chimeric antigen receptor (CAR) T cells represent a promising approach to cancer and have proven efficacy against hematological malignancies, for which they have become the standard of care. However, the use of CAR T cells in solid tumors has been limited.

Objectives

We developed an engineered T cell (CARv3-TEAM-E) that targets EGFRvIII through a CAR while also locally secreting a T-cell-engaging antibody molecule (TEAM) against wild-type EGFR, which is not



expressed in the normal brain but is nearly always expressed in glioblastoma. We sought to determine the safety and bioactivity of intrathecal CARv3-TEAM-E T cells in patients with recurrent glioblastoma.

### Methods

This is a nonrandomized, open-label, single-site Phase I clinical trial. Three patients with EGFRvIII-positive recurrent glioblastoma were enrolled in a safety run-in cohort. Patients were treated with 10 million CARv3-TEAM-E T cells and monitored for toxic effects. Cerebrospinal fluid (CSF) and blood were sampled longitudinally and subjected to correlative analyses.

### Results

No dose-limiting toxic effects were noted. Radiographic tumor regression occurred in all three patients within days after treatment, but this response was transient in two of the patients. Tumor regression correlated with decreased detection of antigen-specific RNA derived from extracellular vesicles (EVs) in both CSF and peripheral blood.

### Conclusion

Early data suggest safety and anti-tumor activity of CARv3-TEAM-E T cells in recurrent glioblastoma. EV-based liquid biopsy may assist in monitoring response to cell therapy. Ongoing enrollment has been modified to enhance durability using lymphodepletive chemotherapy. Additional arms will evaluate this approach in the setting of EGFRvIII-negative tumors and newly-diagnosed disease.

12:40 – 12:45	Wrap up and Adjourn
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1:30 – 4:30	<b>Academy Emerging Investigators' Program</b> Program Director: Gregory Zipfel
1:30 – 2:30	Introduction & Lectures
2:30 – 4:30	Meetings with Established Investigator Faculty

7:30 – 8:20 **The Oldfield Session of Excellence**  
Moderator: Nino Chiocca

7:35 – 7:45 **Inspiration and Innovation in Functional Neurosurgery**  
Russell Lonser, MD

7:45 – 7:55 **Inspiration and Innovation in Cerebrovascular Disease**  
Howard Riina, MD

7:55 – 8:05 **Inspiration and Innovation in Spinal Neurosurgery**  
Vincent Traynelis, MD

8:05 – 8:15 **Inspiration and Innovation in CNS Tumors**  
Melanie Hayden Gephart, MD

8:15 – 8:20 **Wrap-up and Transition**

8:20 – 9:50 **Peer Reviewed Abstract Session VII: Spine and Other**  
Moderators: Gerald Grant and Andrew Dailey

8:20 – 8:30 **Risk factors for Pathologic Fracture Following Stereotactic Body Radiotherapy for Spinal Metastases**  
Benjamin Elder, MD

Introduction

Stereotactic body radiation therapy (SBRT) is an effective treatment option for patients with oligometastatic spine disease with high local control rates. However, a potential complication after high-dose radiotherapy is a pathological vertebral compression fracture (VCF), affecting up to 20% of treated patients.

Objectives

To determine risk factors for VCF following SBRT for metastatic spine disease.

Methods

Patients treated with SBRT for spine metastases at a single institution between 2008 and 2019 were retrospectively reviewed, and patients with a CT scan of the spine within one year prior to SBRT were included. Univariate analysis and multivariable logistic regression was used to identify predictors of post-SBRT VCF.

Results

292 patients with 392 unique lesions were included. The most common pathologies were prostate (n=193), kidney/renal cell (n=46), and lung (n=33). SBRT was generally delivered in 1 to 3 fractions with doses ranging from 16-24 Gy in 1 fraction and 24-36 Gy in 3 fractions. Of the 392 lesions, 73 suffered VCF: 21 with <25% height loss, 15 with 25-40% height loss, and 37 with >40% height loss. On univariate analysis, patients with VCF had lower average Hounsfield units (HU), more WBB sectors involved, higher SINS,

more commonly female, and more commonly had non-prostate pathology. VCF rates were similar between moderate and high-dose radiation schema. On multivariable analysis, predictors of VCF were  $HU \leq 229$  (OR=6.4;  $p < 0.001$ ),  $\geq 3$  WBB segments (OR=2.7;  $p < 0.001$ ), and  $SINS \geq 8$  (OR=2.1;  $p = 0.02$ ).

### Conclusion

Low preradiation HU, involvement of more WBB sectors, and higher SINS score were independent predictors of VCF following SBRT for metastatic spine disease.

## **8:30 – 8:40      Genotype-Guided Opioid Therapy in Patients Undergoing Lumbar Spine Surgery**

**Joe Cheng, MD**

### Introduction

Despite multimodal therapy and ERAS Protocols in lumbar spine surgery, 33%-50% of patients experience inadequate post-operative pain control. The CYP2D6 enzyme metabolizes opioids routinely used with polymorphisms contributing to variability in opioid responsiveness and therapeutic results, with a subset having additional side effects.

### Objectives

Determine feasibility of clinical pharmacogenomic testing is feasible and outcomes of a genotype-guided opioid prescribing strategy on pain control after lumbar spine surgery.

### Methods

Prospectively randomization to CYP2D6-genotype-guided opioid selection (GG) arm (normal metabolizers prescribed tramadol; intermediate, poor, and ultra-rapid metabolizers prescribed non-CYP2D6 opioids (e.g., morphine or hydromorphone), or standard care (SC) arm. Implementation metrics, provider response, medication changes, and patient-reported outcomes including pain and functional status collected at baseline, post-operative days 2-5, 2 weeks, and 3 months.

### Results

96% (69/75) of patients approached agreed to participate. 69 patients randomized (55% female) with 34 in GG arm, 35 in SC arm. For all patients, 55% were normal metabolizers (GG: 59%, SC:51%), 27% intermediate metabolizers (GG: 28%, SC:27%), 5% ultra rapid metabolizers (GG: 3.1%, SC: 6%) and 13% poor metabolizers (GG: 12%, SC:14%). At baseline, no differences in VAS back pain ( $p=0.76$ ), VAS leg pain (0.51) and ODI ( $p=0.60$ ) and EQ-5D ( $p=0.40$ ) between cohorts. Post-operatively, GG arm reported lower ODI scores ( $p=0.02$ ) and higher EQ-5D scores (0.04), with similar VAS back ( $p=0.45$ ) and leg pain scores ( $p=0.48$ ). Hospital length of stay and discharge home (48%) similar between groups ( $p=0.77$ ).

### Conclusion

CYP2D6-guided opioid therapy is feasible and indicates improvement of postoperative functional disability and quality of life after lumbar spine surgery, with a high acceptance of pharmacogenetic testing as part of a clinical trial among patients with spine-related pain.

## **8:40 – 8:50      Bench to Bedside and Back Again: Translational Opportunities in Spinal Cord Injury**

**Ann Parr, MD**

### Introduction

Spinal cord injury (SCI) is devastating. There is likely no single cure and a toolbox of treatments should be explored including combination therapies.

### Objectives

Our NIH funded laboratory has focused on 3 different therapies: new and replicable culture techniques for induced pluripotent stem cell (iPSC) derived regionally specific neuronal progenitor cells (NPCs) to create a relay system in chronic SCI, a 3D printed matrix to create spinal cord organoids/assembloids, and epidural stimulation to encourage appropriate connectivity.

### Methods

Our translational laboratory has utilized standard cell culture techniques to develop our cell protocols. We collaborated with Mechanical Engineering to produce our 3D scaffolds and compared them to 2D cell culture, utilizing both imaging and electrophysiological methods. We utilize standard behavioral testing including locomotive and electrophysiological measures in conjunction with tail nerve electrical stimulation to explore these effects.

### Results

We have created a fast, clinically relevant method of producing regionally specific spinal ventral and dorsal iPSC derived NPCs (Fig 1). We have developed a new method of 3D printing these cells (Fig 2). We have tested the effects of epidural stimulation on these cells after transplantation in a rat model. We also have a human clinical trial of epidural stimulation in concert with these studies.

### Conclusion

Spinal cord injury is complex and a combinatorial therapy is likely needed. We have discovered that while epidural stimulation is beneficial to many patients in a clinical setting, a lack of sensory/proprioceptive function remains a problem, and cell transplantation therapy should be further explored. Further, some of our patients have demonstrated neuroplasticity in that they retain function after the stimulation is off (Fig 3). Thus, we have further studied this in our rat model to elucidate mechanism. Our takeaway message is that there is interplay between basic science and clinical research that is crucial to advancement in the field.

<b>8:50 – 9:00</b>	<b>Machine Learning-based Cluster Analysis Identifies Four Unique Phenotypes of Degenerative Cervical Myelopathy Patients</b>
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**Michael Fehlings, MD**

### Introduction

Degenerative cervical myelopathy (DCM), the predominant cause of spinal cord dysfunction among adults, exhibits a diverse range of symptoms. Traditional classification using the mJOA has not been able to address its complex heterogeneity.

### Objectives

This study employs machine learning-based clustering algorithms to identify distinct patient clinical profiles and functional trajectories following surgery.

### Methods

We implemented both latent profile analysis and k-means clustering on aggregated data from three large DCM trials. Key covariates, including the Nurick score, NDI (neck disability index), neck pain, and motor and sensory scores, were employed for clustering. Outcome differences among identified phenotypes were assessed using ANOVA, followed by posthoc Tukey test.

## Results

A total of 1,047 DCM patients (mean [SD] age: 56.80 [11.39] years) had complete one-year outcome assessment. Both LPA and k-means clustering identified four DCM patient phenotypes: 'severe multimodal impairment'; (n=286), 'minimal impairment'; (n=116), 'motor-dominant'; (n=88) and 'pain-dominant'; (n=557) groups. The 'severe multimodal impairment' group, comprised of frail elderly patients, demonstrated the worst overall one-year outcomes (SF-36 PCS mean [SD]: 40.01 [9.75]; SF-36 MCS mean [SD]: 46.08 [11.50]), but exhibited the most substantial neurological recovery after surgery (mJOA mean [SD]: 3.83 [2.98]). A higher frailty score and a positive smoking status predicted membership in phenotype 1 ('severe multimodal impairment' group).

## Conclusion

Unsupervised learning algorithms applied to baseline DCM symptoms enabled the prediction of distinct patient phenotypes. The concept of symptom clustering provides a valuable framework for uncovering novel DCM subpopulations, enhancing patient identification beyond the use of a single patient-reported outcome measure such as the mJOA.

<b>9:00 – 9:10      Predictors of Oswestry Disability Index Deterioration at 5 Years After Surgery for Grade 1 Spondylolisthesis: QOD Study</b>
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**Oren Gottfried, MD**

## Introduction

There is limited data on patient characteristics that contribute to long-term functional decline in patients with grade 1 spondylolisthesis who undergo surgery. The aim of this study is explore the factors that contribute to functional deterioration at 5 years postoperatively.

## Objectives

Worse pain and functional status at baseline are expected to be correlated with functional deterioration at 5 years after surgery.

## Methods

This was an analysis of the prospective Quality Outcomes Database Grade 1 Spondylolisthesis cohort which included adult patients who were diagnosed with primary grade 1 spondylolisthesis undergoing elective surgery at 14 highest enrolling sites. Function was measured with Oswestry Disability Index (ODI). Patients were dichotomized based on whether their ODI improved or worsened at 5-year follow-up compared to baseline. Those who maintained the same ODI were excluded. A multivariable logistic model using the stepwise selection method was used to find the most contributive predictors of ODI deterioration.

## Results

Of the 608 patients with grade 1 spondylolisthesis who underwent surgery, 483 had 5-year follow-up ODI. Of these, 36 (7.5%) had worse ODI, 110 (22.8%) had no change in ODI, and 337 (69.8%) had improved ODI at 5-year follow-up. The 5-year follow-up rate was 81%. Patients with worse and improved ODI had similar age (65.4±12.6 vs 61.7±11.6), BMI (31.9±5.9 vs 30.2±6.4), and ASA grade (2.4±0.6 vs 2.3±0.6). Surgical characteristics were also similar between the two groups with similar length of surgery (175±79.3min vs 174±86.8min), and length of stay (2.6±1.5d vs 2.7±1.8d) (all p>0.05). The two groups had similar baseline back pain (6.9±2.4 vs 6.8±2.6) and leg pain (5.9±2.6 vs 6.6±2.8) (all p>0.05). Using multivariable logistic modeling, worse baseline back pain (OR=1.02, p<0.01) was predictive of worse ODI at 5 years.

### Conclusion

Patients with worsened and improved function at 5-year follow-up after surgery for grade 1 spondylolisthesis did not differ in demographics, comorbidity, or surgical characteristics. Worse back pain at baseline was a significant predictor for ODI deterioration at 5 years.

<b>9:10 – 9:20      The Integration of Regional Analgesia into ERAS Improves Perioperative Outcomes in MIS and Anterior Lumbar Spine Surgery</b>
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**John O’Toole, MD**

### Introduction

Enhanced recovery after surgery (ERAS) pathways and regional analgesia, such as erector spinae plane blocks (ESPB), have both individually shown promise in improving perioperative outcomes in spine surgery. However, limited research exists on their combined effects. Additionally, the role of ESPB in anterior lumbar operations remains unclear.

### Objectives

This retrospective study aimed to investigate the impact of combining ESPB with an established ERAS pathway on perioperative outcomes in elective minimally invasive spine surgery, specifically single-level transforaminal interbody fusion (TLIF) as well as standalone anterior lumbar surgeries, including anterior lumbar interbody fusion (ALIF) and lumbar total disc replacement (TDR).

### Methods

Consecutive patients undergoing TLIF were divided into PreERAS, ERAS, and ERAS+ESPB groups. Similarly, patients undergoing standalone ALIF or TDR were categorized based on ESPB administration. Primary outcomes were in-hospital self-reported pain scores and postoperative opioid requirements, while secondary outcomes included length of stay (LOS) and complications.

### Results

Both ERAS and ESPB resulted in significantly reduced opioid requirements following MIS TLIF, with ERAS+ESPB demonstrating the greatest reduction. In anterior lumbar surgery patients, ESPB was associated with shortened LOS as well as lower pain scores, particularly on postoperative day zero and a trend toward lower total admission opioid utilization. Patients with reduced opioid intake following ESPB had shorter LOS, while previous lumbar surgeries did not significantly impact perioperative outcomes in the ESPB group. No adverse events related to ESPB were observed.

### Conclusion

The addition of ESPB regional analgesia into established ERAS pathways in minimally invasive and standalone anterior lumbar spine surgery led to improved perioperative outcomes. Prospective studies are underway to more precisely define the magnitude of the effect of ESPB on postoperative pain scores, opioid utilization and length of stay.

<b>9:20 – 9:30      Neurological Surgery Residency Programs in the United States: A National Cross-Sectional Survey</b>
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**Brian Nahed, MD**

### Introduction

Neurosurgical residency training provides the surgical training, knowledge, and psycho-social skills to develop into a competent neurosurgeon. Given the breadth and depth of opportunities across programs, we

investigated the impact of program structure, resources, and opportunities on resident training and academic productivity.

### Objectives

Characterize trends, opportunities, and impact in a comprehensive analysis of US neurosurgical training programs

### Methods

A 34-question survey was circulated to 117 programs to assess neurosurgical residency programs, including curricular structure, fellowship availability, recent program changes, graduation requirements, and resources supporting career development. Mean resident productivity by program was collected from the literature. Demographic data was also collected from publicly available websites and reports from the National Resident Match Program.

### Results

Seventy five programs (64.1%) responded. There was a median of 2.0 (range 1.0-4.0) resident positions per year and 1.0 (range 0.0-2.0) research/elective years. Programs offered a median of 1.0 (range 0.0-7.0) CAST-accredited fellowships, with endovascular being most frequently offered (53.8%). There was a median number of 3.0 clinical sites (range 1.0-6.0). Residents received funding in 46.7% of programs, and a median academic stipend of \$1000 (range \$0-\$10 000) per year. Wellness activities occurred in 93.3% of programs. Annual academic stipend size was the only significant predictor of resident academic productivity ( $R^2 = 0.17, P = .002$ ).

### Conclusion

Neurological surgery residency programs successfully train the next generation of neurosurgeons focusing on education, clinical training, case numbers, and milestones. These programs offer trainees the chance to tailor their career trajectories within residency, creating a rewarding and personalized experience that aligns with their career aspirations.

9:30 – 9:50	BREAK
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9:50 – 11:05	<b>Peer Reviewed Abstract Session VIII: Cerebrovascular II</b> Moderators: Mike Lawton, Christopher Ogilvy, and Felipe Albuquerque
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9:50 – 10:00	<b>Experimental and Clinical Evidence for Volitional Anesthetic Conditioning as a Novel Treatment Strategy for SAH</b>
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Gregory Zipfel, MD

### Introduction

The two most treatable causes of poor patient outcome after SAH are Early Brain Injury (EBI) and Delayed Cerebral Ischemia (DCI). Currently, treatment strategies to prevent or reduce EBI and DCI are limited.

### Objectives

Our objective was to apply a therapeutic strategy - conditioning - that is not only powerful but also remarkably pleiotropic, with proven protective effects on all major cell types of the CNS.

## Methods

Apply volatile anesthetic conditioning to rodent models of SAH with genetic or pharmacologic interventions examining specific molecular pathways; and examine cohorts of SAH patients who underwent general anesthesia for aneurysm repair via inhalational anesthetic alone vs. combined anesthetics (propofol infusion plus lower dose volatile anesthetics) or total intravenous anesthesia (propofol infusion without any volatile anesthetics).

## Results

First, we showed that brief exposure to isoflurane provided strong protection against DCI and neurologic deficits in mouse SAH. Second, we explored dosing and underlying mechanism of isoflurane conditioning-induced DCI protection including pharmacologic and genetic data implicating HIF-1, eNOS, iNOS, and NF-kB. Third, we showed that clinically relevant doses of other commonly used volatile anesthetics such as sevoflurane and desflurane provide similarly strong protection against DCI and neurological deficits in mouse SAH but that anesthetic doses of the intravenous anesthetic, propofol, did not. Fourth, we cross-validated these experimental observations with three retrospective clinical studies examining two large cohorts of SAH patients. We found that SAH patients who received volatile anesthetics alone during aneurysm repair (coiling or clipping) were associated with lower incidence of angiographic vasospasm and less DCI compared to those who received combined anesthetics (propofol infusion plus lower dose volatile anesthetics) or those who received total intravenous anesthesia (propofol infusion without any volatile anesthetics).

## Conclusion

Our preclinical and clinical evidence suggest volatile anesthetics may have a role in attenuating secondary brain injury after SAH and improving functional/cognitive outcomes in SAH patients.

## **10:00 – 10:10 Spatial Gene Profiling in 3D-Printed Aneurysm Model with Complex Flow Patterns**

**Alexander Khalessi, MD**

### Introduction

Intracranial aneurysms pose a significant health challenge, with rupture risk prediction and prevention hindered by limited understanding of their cellular and molecular mechanisms within complex fluid dynamics environments.

### Objectives

In this study, unique spatial gene profiling was applied to investigate the responses of individual endothelial cells to the complex flow conditions present in aneurysms created by using a 3D-printed endothelialized in vitro model.

### Methods

The aneurysm model was constructed by attaching the 3D-printed half aneurysm onto a glass slide on which the Human umbilical vein endothelial cells (HUVECs) were seeded. The disturbed and laminar flow regions were characterized with time-averaged wall shear stress using Computational Fluidic Dynamics. The endothelial alignment and cell-covered area in both flow regions were characterized using fluorescent microscopy. The expressions of 43 genes in each endothelial cell at the two regions were quantified by using a spatial gene profiling technology.



## Results

From Day 0 to Day 3, HUVECs displayed heightened alignment and cell density in the laminar flow region relative to the disturbed flow region. Furthermore, ECs in the disturbed flow region showed reduced expression of eight athero-protective genes and elevated expression of twelve atherogenic and inflammatory genes compared to those in the laminar flow region.

## Conclusion

This study introduces a novel use of spatial gene profiling technology to examine the cellular and molecular responses of ECs at the single-cell level in complex fluid dynamics within a 3D-printed live-cell aneurysm model in vitro. This platform offers an effective means to investigate mechanisms underlying aneurysm development and progression.

<b>10:10 – 10:20    Establishing a Bench at the Bedside in the Angio Suite: Using Prospective Tissue Banking to Understand Ischemic Stroke</b>
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**Justine Fraser, MD**

## Introduction

Advancements in therapeutics for ischemic stroke have been impeded by translational barriers between often-used animal models and the human condition. Understanding stroke in real-time in clinical patients is crucial to moving therapeutic development forward. We established a prospectively enrolling tissue bank for patients undergoing mechanical thrombectomy, and then used that as a platform for comparative studies. Our central aim was to create, validate, and utilize our tissue bank to make novel discoveries in human ischemic stroke.

## Objectives

NA

## Methods

In 2017, we established the “Blood And Clot Thrombectomy Registry And Collaboration” (BACTRAC; NCT03153683) to prospectively collect, bank, and evaluate tissues from ischemic stroke patients. Specifically, we collected systemic arterial blood, the removed intracranial thrombus, and static blood from the intracranial circulation just distal to the thrombus prior to thrombectomy. In addition, we established controls from arterial blood collected from non-stroke patients during routine cerebral angiograms. Tissues were processed and banked in a lab space adjacent to the angio suite. We performed proteomic and RNA integrity analyses to validate our methods. Thereafter, we used these specimens and performed proteomic, transcriptomic, acid/base, and immune cell analyses. There were linked clinically through collection and analysis of clinical and radiographic data.

## Results

To date, we have enrolled 213 stroke subjects, and 96 controls. RNA integrity analyses and proteomics demonstrated satisfactory quality of RNA and protein from both systemic and intracranial blood samples. Initial analysis of acid/base balances demonstrated significant differences in systemic changes between men and women undergoing thrombectomy. Intracranial transcriptomics demonstrated a notable “storm” of cytokine activation in response to stroke. Proteomic evaluations demonstrated notable and significant differences in local protein changes intracranially compared to systemic circulation. Linking such changes clinical data have yielded insights into stroke risk factors. For example, we have noted significant differences in proteomic changes in stroke patients from Appalachian vs non-Appalachian regions of our state.

### Conclusion

The BACTRAC registry has provided a major platform for deep analyses of ischemic stroke in the human population. As the tissue bank has expanded, multifactorial analyses have yielded novel findings about how clinical aspects of patients correlate to significant pathophysiologic changes during ischemic stroke. This platform may provide for a more patient specific understanding of the disease, leading to more relevant future therapeutics.

## **10:20 – 10:30 Machine Learning Based Rupture Risk Prediction for Intracranial Aneurysms in Comparison to the PHASES Score**

**Bernard Bendok, MD**

### Introduction

Aneurysm risk prediction remains an imprecise science which places patients at risk for either over or undertreatment. Machine learning (ML) models may improve clinical practice by adding precision to risk assessment, however ML models must be validated against current standard approaches.

### Objectives

This systematic review and meta-analysis aim to comprehensively assess the current landscape of machine learning (ML) applications in predicting the risk of aneurysm rupture and compare the performance with the widely used PHASES score.

### Methods

A systematic review of PubMed, Scopus, Web of Science, and Cochrane Library was conducted. All studies utilizing ML tools to predict the rupture risk of intracranial aneurysms were included. Risk of bias was assessed using the Quality Assessment of Diagnostic Accuracy Studies-2 tool. Meta-analysis was conducted with consideration to the ML algorithms and comparison was made with PHASES score.

### Results

36 studies including 22,462 patients were analyzed. ML techniques, including 124 models using 25 algorithms, were utilized in these studies. Based on our analysis ML provides comparable sensitivity (0.743 vs 0.771) and higher specificity (0.763 vs 0.507) compared to the PHASES score for aneurysm rupture risk prediction. Pooled analysis of the 36 included studies using 60 models for ML and 5 models for the PHASES score, showed higher performance metrics for ML models than PHASES score (AUC 0.84 vs 0.64). Among various ML models, deep learning (DL) exhibited the highest sensitivity (0.803) and specificity (0.788).

### Conclusion

ML techniques may enhance prediction of intracranial aneurysm rupture compared to traditional approaches such as the PHASES score. Our analysis demonstrates a comparable sensitivity between ML models and the PHASES score; however, specificity was higher among the ML models, particularly DL. Incorporating hemodynamic parameters may further enhance the accuracy of ML models, however further external validation is to be pursued.

## **10:30 – 10:40 A Mast Cell-Specific Receptor Mediates Post-Stroke Brain Inflammation via a Dural Brain Axis**

**Risheng Xu, MD**

## Introduction

The immune environment surrounding the central nervous system plays a fundamental role in monitoring the brain for signs of injury. Pathologies such as ischemic stroke can trigger an inflammatory response that further exacerbates neuronal injury and prevents long-term recovery. The underlying mechanisms that drive this hyperactive immune cell response after ischemic brain injury remains unclear.

## Objectives

To investigate the role of a Mrgprb2/MrgprX2, a mast cell specific receptor, in the neuroinflammatory cascade after ischemic stroke.

## Methods

Utilizing genetic, pharmacological, and skull-bone marrow transplant techniques, we demonstrate that Mrgprb2-positive mast cells are critical for downstream neuroinflammation after ischemic stroke.

## Results

We show that Mrgprb2<sup>-/-</sup> mice are protected from ischemic stroke injury, and localize Mrgprb2 expression to meningeal mast cells only. Activation of Mrgprb2 after stroke causes mast cell degranulation and release of cytokines and chemokines that attract downstream immune cells. Meningeal mast cells via Mrgprb2 specifically regulate recruitment of skull bone marrow neutrophils into the brain. We demonstrate that the human ortholog of this receptor, MRGPRX2, is expressed in human meningeal mast cells. These cells are activated in stroke patients, due in part to upregulation of the neuropeptide substance P, a known ligand of MRGPRX2. Further, pharmacologic inhibition of Mrgprb2 reduces post-stroke brain inflammation and improves motor outcomes in mice.

## Conclusion

Collectively, our study identifies Mrgprb2 as a critical mediator of mast cell activation after ischemic stroke, deciphering an important regulatory component of the brain-dural-immune interface. This meningeal mast cell receptor provides a specific and druggable target to attenuate post-stroke inflammation and holds therapeutic potential.

<b>10:40 – 10:50 Intraoperative High Resolution MicroDyna CT Angiography for Perforator Vessel Mapping during Microsurgical Clipping</b>
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**Omar Choudhri, MD**

## Introduction

Micro Dynamic computed tomography (micro DynaCT) is an advanced intraoperative flat panel angiography imaging technique that provides real-time high-resolution microvascular imaging of submillimeter intracranial perforator vessels.

## Objectives

In this study, we aimed to evaluate the clinical feasibility, accuracy, and utility of employing an intraoperative micro DynaCT imaging protocol in a hybrid operating room during microsurgical treatment of various neurovascular pathologies. Perforator occlusion with subcortical ischemia is often missed during intraoperative neurophysiologic monitoring and microDyna CT may provide a useful adjunct in this assessment.

## Methods

We retrospectively reviewed 20 patients who underwent micro DynaCT during arteriovenous malformation resection, aneurysm clipping, and dural arteriovenous fistula treatment at the Hospital of the University of Pennsylvania between July 2022 and April 2024. Angiography was performed using the biplane angiographic suite (Artis Icono; Siemens) and reconstructions were performed using DynaCT software. A 50% contrast dilution mixture (isovue-300) was employed for 34cc volume injected at 2cc/sec (Nemoto Press DuoElite). Ability to identify and measure recurrent artery of Heubner, anterior choroidal artery, lateral lenticulostriate perforators was assessed by 3 separate neuroradiologists on 0.5mm multiplanar reconstructed datasets.

## Results

Among the 20 patients reviewed, the average age was 51.5 years, with 45% (9/20) of the patients being male. 75% (15/20) of the procedures were aneurysm clippings, 11.1% (3/20) were arteriovenous malformation resections, and 10% (2/20) were dural arteriovenous fistula treatments. The average contrast volume used was 52.14 mL. Each of the evaluating neuroradiologists were able to consistently spatially resolve the 3 perforator vessels in 90% (18/20) of the cases.

## Conclusion

Intraoperative microDynaCT allows reproducible perforator vessel imaging during microsurgical clipping with superior spatial resolution despite presence of intraoperative surgical equipment and absence of bone flap, with minimal artifact. This intraoperative imaging technique has the potential to reduce morbidity from perforator compromise during microsurgery.

## **10:50-11:00 First In Human Isotope Tracing Reveals Metabolic Vulnerabilities in Glioblastoma**

**Wajad Al-Holou, MD**

## Introduction

The brain avidly consumes glucose to fuel neurophysiology. However, cancers of the brain, including glioblastoma, lose aspects of normal biology and gain the ability to proliferate and invade healthy tissue by rewiring glucose utilization to drive tumor growth and treatment resistance. How brain cancers utilize glucose utilization to fuel these processes is poorly understood.

## Objectives

NA

## Methods

We developed a clinical trial to perform intraoperative radiolabeled stable isotope tracing studies utilizing <sup>13</sup>C-labeled glucose in patients undergoing resection of a brain tumor. Isotope tracing allows for direct interrogation of metabolic pathway activity in cancer. To perform these studies, metabolic substrates containing heavy (but not radioactive) isotopes such as <sup>13</sup>C are administered in living systems. The isotopes are then tracked into their downstream fates by mass spectrometry. In this study, we combined this analysis of the intraoperatively obtained tissue with newly developed quantitative metabolic flux analysis and gene expression analyses.

## Results

We have identified that normal human cortex funnels glucose-derived carbons towards physiologic processes such neurotransmitter synthesis and the TCA cycle. In contrast, brain cancers downregulate these physiologic processes, and instead use glucose-derived carbons to produce molecules needed for

proliferation and invasion, such as NAD/NADH. Furthermore, we determined that molecules normally synthesized by the brain de novo, such as serine, are not produced by cancers, but rather scavenged from the environment. To assess this potential metabolic vulnerability, we performed in vivo experiments using a serine-restricted diet, which showed that serine restriction significantly decreased tumor size with a significant decrease in Ki-67 index.

### Conclusion

This study is the first to directly measure biosynthetic flux in both glioma and cortical tissue in human brain cancer patients. We show that brain tumors rewire glucose carbon utilization away from oxidation and neurotransmitter production towards biosynthesis to fuel growth. Blocking these metabolic adaptations with dietary interventions presents promising translational opportunities.

11:00 – 11:05 **Wrap-up and Transition**

11:05 – 11:25 **Academy Award Presentation and Lecture**

11:05 – 11:10 **Announcement of NREF Winners**

Gregory Zipfel, MD

11:10 – 11:15 **Introduction of Academy Award Winner**

Michael Vogelbaum, MD

11:15 – 11:25 **Academy Award Presentation Abstract: A Proteogenomic Blood Test for Acute Spinal Cord Injury**

Tej Azad, MD

### Introduction

Physical examination and neuroimaging are integral for acute spinal cord injury (SCI) management, yet have limited spatiotemporal resolution and do not capture molecular features of SCI.

### Objectives

To develop an acute SCI blood test that enables rapid diagnosis, correlates with injury severity, and predicts long-term outcomes.

### Methods

We performed methylome profiling of human spinal cords and integrated these profiles with methylomes from 25 tissue/cell types, including cortical neurons, deriving a signature of spinal cord identity. We designed a bespoke droplet digital PCR (ddPCR) assay to target promising CpG sites, applied this ddPCR assay to cell free DNA (cfDNA) extracted from preoperative blood samples, and performed targeted proteomics in the same samples. Finally, we developed an algorithm to integrate cfDNA and protein into a unified Spinal Cord Injury Index (SCII).

### Results

The ddPCR assay was applied to preoperative blood draws from 50 prospectively enrolled surgical patients with acute SCI and 20 controls (AUC:0.89, P<0.0001), detecting spinal cord-specific cfDNA at levels down to 4.4 haploid genome equivalents/mL plasma. A dimensionality-reduction algorithm selected a parsimonious set of four proteins, which were integrated with spinal cord-specific cfDNA to derive the

SCII. The SCII discriminated SCI patients from controls (AUC:0.91,  $P < 0.0001$ , A), correlated with presentation ASIA scores ( $P < 0.0001$ , B), and predicted six-month ASIA conversion (AUC:0.77,  $P = 0.006$ , C).

### Conclusion

A proteogenomic assay detects neuroglial cell death in the blood of SCI patients and predicts clinically meaningful endpoints. Future work will focus on prospective validation with serial blood draws and translation to other acute neurosurgical pathology.

11:25            **Wrap-up and Transition**

11:25 – 12:45 **Peer Reviewed Abstract Session IX: Functional and Epilepsy**  
Moderators: Costas Hadjipanayas and Daniel Resnick

11:25 – 11:35 **NeuroVision: Advancing Brain Magnetic Resonance Imaging with Vision-Language Models**

Todd Hollon, MD

### Introduction

Brain magnetic resonance imaging (MRI) is central in the diagnosis and treatment of neurological diseases. Despite recent advances in artificial intelligence (AI), the complexity of neurological diseases and brain MRIs have prevented the development of decision support tools and computer-aided diagnosis. Such systems have the potential to automated triage, recommend clinical referrals, and diagnose neurological diseases.

### Objectives

To develop, train, and validate NeuroVision, the first general-purpose MRI AI model for automated brain MRI diagnosis, acuity assessment, triage.

### Methods

A diverse, multicenter dataset of over 500K brain MRIs were collected and curated for the study, the largest MRI dataset to date. Brain MRIs and their associated radiology reports were used to train NeuroVision, a vision-language model with 10 billion parameters, using a contrastive language-image pair objective function.

### Results

NeuroVision was tested on a prospective, health system-scale patient cohort using the following metrics: acuity/severity assessment, clinical referral recommendation, and radiologic diagnosis. The testing cohort include 25K consecutive patients. NeuroVision achieve a acuity assessment and clinical referral recommendation accuracy of over 95%. NeuroVision achieved a mean diagnostic accuracy of over 90% across the major neurologic and neurosurgical disorders, including traumatic, infectious, inflammatory, vascular, developmental, and neoplastic lesions (52 diagnoses total).

### Conclusion

We present the first general-purpose vision-language model for brain MRI interpretation and diagnosis. Neurovision was trained and validated using health system-scale datasets, achieving high performance on clinically actionable diagnostic tasks. NeuroVision provides a bridge from scan-to-neurosurgeon using state-of-the-art AI models that will continue to improve in the 21st century.

**11:35 – 11:45 Cortico-Thalamic Synchronization: A Blueprint for Precision Neuromodulation in Epilepsy Surgery**

**Jorge Gonzalez-Martinez, MD**

Introduction

Since Penfield and Jasper's centrencephalic epilepsy theory, thalamic rhythmicity and cortical excitability's role in spike-wave seizures has been extensively explored. The thalamocortical neuronal network, governing physiological oscillations like sleep spindles, serves as the substrate for spike-wave seizure patterns. While altered thalamus-cortex connectivity is noted in generalized epilepsy, thalamocortical synchronization in focal seizures is less studied.

Objectives

Our objective is to scrutinize personalized cortical-thalamic connectivity and ictal synchronization, thereby refining thalamic targets for precise neuromodulation.

Methods

We prospectively analyzed intracerebral recordings from drug-resistant epilepsy patients with cortico-thalamic SEEG electrodes. Structural and functional thalamus-cortex connectivity was assessed using high-definition DTI and thalamic stimulation-evoked responses. Ictal patterns from distinct thalamic nuclei were qualitatively and quantitatively analyzed for synchronization with epileptogenic cortical areas, with emphasis on the posterior quadrant.

Results

In total, 45 patients with 300 seizures were studied. Thalamic regions were implanted with 3-4 electrode contacts per patient. Connectivity studies revealed specific pulvinar-posterior quadrant cortex correlations, anterior nucleus-basal and rostral fronto-temporal connections, and motor thalamus-Rolandic cortex correlations. During seizures, maximal involvement and synchronization were identified between the pulvinar and seizures organized in the posterior quadrant cortex, while the motor thalamus correlated with Rolandic seizures. The anterior nucleus had minimal involvement in Rolandic and posterior quadrant seizures but modest correlation with anterior frontal and temporal seizures. Subsequently, 5 patients with posterior quadrant epilepsies underwent chronic neuromodulation device implantation in the pulvinar, achieving an 85% reduction in seizure severity and frequency.

Conclusion

Thalamic projection specificity to cortical regions and seizure activity organization highlight the importance of individualized thalamic target selection for optimal seizure outcomes in non-resective surgery candidates.

**11:45 – 11:55 Brain-State Modeling for Adaptive Closed-Loop Neuromodulation of Drug-Resistant Epilepsy**

**Dario Englot, MD**

Introduction

The future of closed-loop adaptive neuromodulation for drug-resistant epilepsy relies on a biomarker that can effectively quantify seizure propensity in a smooth and continuous distribution for effective device feedback. Akin to the concept of a 'tornado watch'; describing proper conditions for tornadic activity versus an actual funnel cloud forming: An electrographic epileptic biomarker must allow for the mapping of high-risk brain states that are presumed to exist (tornado-watch) without immediate transformation to seizure

activity. The main challenge is that presumed high seizure propensity states cannot be labeled as ‘pre-ictal’ if they do not immediately form a seizure.

### Objectives

Thus, we sought to create an artificial intelligence model that can self-organize high-dimensional brain states based on raw stereotactic electroencephalography (SEEG) data without labels.

### Methods

To develop and validate the patient-specific brain-state model architecture, we utilized our cohort of approximately 17,000 hours of continuous SEEG data from 118 patients with drug-resistant epilepsy undergoing SEEG presurgical evaluation (Figure 1A-H). Next, we evaluated if the trained 512-dimensional brain-state model effectively organized based on post-hoc inclusion of known peri-ictal labels by reducing the dimensionality to two and clustering the data (examples in Figure 2A-F). Finally, we tested the hypothesis that the brain-states could be selectively neuromodulated by single-pulse electrical stimulation (SPES).

### Results

The projection of all SEEG data into a two-dimensional representation of the 512-dimensional brain-state space allowed for clear self-organization of pre-ictal, ictal, and post-ictal epochs based on post-hoc inclusion of known peri-ictal labels (Figure 2G-J), including on withheld data. Neuromodulation of the state-space through SPES reveals increased brain-state transitions (t-test p-value range: 0.0367 to 6.25e-5, Figure 3A-C) and increase in unique brain-states (p-values 8.81e-3 to 6.74e-8, Figure 3D-F) during low-energy stimulation.

### Conclusion

We have developed a self-organized patient-specific electrographic biomarker of seizure propensity with evidence of neuromodulation during low-energy stimulation - displaying strong potential for closed-loop adaptive neuromodulation.

## 11:55 – 12:05 Evoked Resonant Neural Activity for Target Identification in Parkinson Disease

Ashwin Viswanathan, MD

### Introduction

Evoked resonant neural activity (ERNA), a resonant response after stimulation which dampens over time, has emerged as a new biomarker in Parkinson Disease. Contacts with the largest ERNA amplitude have been correlated with the optimal therapeutic stimulation contact. One key advantage of ERNA is the large signal amplitude. However, limited data exists on ERNA acquired from asleep subjects during DBS surgery.

### Objectives

Determine whether ERNA can be reliably obtained in the asleep state, and hence be used to confirm optimal lead placement during asleep DBS.

### Methods

Seven patients (4 STN, 3 GPi) undergoing bilateral DBS surgery were evaluated intraoperatively in the asleep and awake states during deep propofol anesthesia, and after anesthetic washout. Stimulation (130 Hz, 3mA) was delivered through the bottom contact of the implanted lead while recording local field potentials sampled at 15 kHz. Stimulation was delivered in bursts of 20 pulses, followed by a 200-millisecond gap, where ERNA may be present. Time-frequency maps (TFM) for each gap were created and averaged (Figure 1c,e).



## Results

Figure 2 illustrates findings in asleep and awake states, with ERNA extending for 40 to 70 milliseconds in the time domain. TFM reveals ERNA extends close to 100 milliseconds after stimulation ends. No significant difference was seen in peak to peak amplitude ( $177.11 \pm 287.36$  versus  $152.35 \pm 256.66$   $\mu\text{V}$ ,  $p=0.18$ ) or RMS ( $16.37 \pm 30.18$  versus  $14.83 \pm 24.43$   $\mu\text{V}$ ,  $p=0.65$ ) of the ERNA in the asleep and awake states respectively (Wilcoxon signed rank test). Contacts located within the target structure determined by Lead-DBS had significantly higher ERNA amplitude ( $475.4 \pm 294.9$  versus  $179.5 \pm 76.8$   $\mu\text{V}$ ,  $p\text{-value} = 4.3e-5$ , two-sample  $t$ -test) and area under the curve ( $8216.52 \pm 5177.3$  versus  $3006.5 \pm 1796.6$   $\text{nWb}$ ,  $p\text{-value} = 4.9e-5$ , two-sample  $t$ -test) compared with contacts outside the target.

## Conclusion

ERNA may be a robust signal for validating DBS lead placement in both awake and asleep DBS patients.

## 12:05 – 12:15 Gene Therapy in Huntington’s Disease: Analysis of Real-time Magnetic Resonance Imaging Multi-site Delivery

James Elder, MD

### Introduction

Current gene therapy clinical trials often use real-time MR-imaging during CED to monitor infusions, aiming to optimize target coverage. However, intraoperative imaging features of these surgical strategies in HD are not well described.

### Objectives

To characterize real-time magnetic resonance (MR)-imaging properties of convection enhanced delivery (CED) of multiple gene therapy infusions in patients with Huntington’s Disease (HD).

### Methods

Consecutive HD patients enrolled in a clinical trial of CED of recombinant adeno-associated viral vector serotype 5 expressing microRNA targeting human HTT (rAAV5-miHTT) co-infused with 1 mM gadoteridol were included. Maximum infusion volumes were 3000 microliters per patient - 1000 microliters (2 injections) per putamen and 500 microliters (1 injection) per caudate. Surgical and intraoperative MR-imaging features were analyzed.

### Results

Ten early manifest HD patients underwent real-time MR-imaging during CED of bilateral striatum using a trans-frontal approach (60 total infusions; 20 caudate, 40 putamen) at one surgical center. Volume of distribution ( $V_d$ ) increased linearly with increasing volume of infusion ( $V_i$ ). Mean  $V_d/V_i$  ratio at infusion completion was similar for each target (Table 1). Targeting accuracy (percent infusion within target) was  $35.1 \pm 15.6\%$  (13.7-62.4%) for caudate infusions,  $45.6 \pm 12.7\%$  (30.9-65.3%) anterior putamen,  $42.5 \pm 8.0\%$  (29.1-56.8%) posterior putamen. Mean percent target structure infused was  $18.4 \pm 3.0\%$  (14.3-22.6%) per caudate and  $34.0 \pm 7.0\%$  (19.9-43.0%) per putamen. Gadoteridol hyperintensity dissipated in a defined manner after stopping infusion (mean  $T1/2$  95.3  $\pm$  37.2 minutes). Off target infusion occurred via infusion volume expansion past target structure borders into anatomically-adjacent white/grey matter structures in 60/60 (100%) of infusions and via low resistance pathways (e.g., perivascular spaces) in 50/60 (83%) infusions.

### Conclusion

In HD patients, gene therapy CED followed definable volume of distribution and target structure coverage patterns. After infusion completion, gadoteridol dissipation occurs in a defined manner. Additional imaging analysis and correlation with clinical outcomes is critical for shaping future clinical trials.

## **12:15 – 12:25 Gait Phase Triggered Adaptive Deep Brain Stimulation Device Using Machine Learning for Seizure Prediction and Treatment**

**Doris Wang, MD**

### Introduction

Human gait is a complex movement that entails the dynamic coordination of synchronized neural activities across the locomotor network. Gait disturbances are particularly debilitating motor impairments in Parkinson's disease (PD), and unlike symptoms of bradykinesia and rigidity, are often refractory to conventional deep brain stimulation (DBS). This is likely because the therapeutic desynchronization effects of continuous DBS may impair the neural network's ability to dynamically synchronize during normal gait. Therefore, stimulation that can dynamically change during the gait cycle may overcome this limitation.

### Objective

1) To identify neural biomarkers of left and right leg swing from chronically implanted cortical and basal ganglia electrodes and 2) To develop and test personalized adaptive DBS (aDBS) that alters stimulation using these biomarkers.

### Methods

Two PD patients underwent bilateral globus pallidus DBS implantation, with subdural cortical paddles overlying the primary motor and premotor cortices connected to bidirectional sensing neural stimulators (Summit RC+S). Local field potentials (LFP) from cortical and subcortical electrodes were wirelessly streamed and synchronized to movement kinematic data while the subjects walked overground. Biomarkers specific to contralateral leg swing were identified utilizing LFP spectral power. aDBS program that alters stimulation amplitude during contralateral leg swing were embedded into the subjects' stimulator and tested for accuracy and effects on gait.

### Results

In both subjects, compared to continuous DBS, aDBS significantly decreased step time and step length, improved step time and step length symmetry, and decreased variance in step time and step length.

### Conclusion

Adaptive DBS triggered by gait phase is feasible and can improve gait parameters.

## **12:25 – 12:35 Development of an Ultrasound Powered Brain Stimulation Device Using Machine Learning for Seizure Prediction and Treatment**

**Joseph Neimat, MD**

### Introduction

The past decade has seen significant advances in central neuromodulation to treat epilepsy. Further improvement will be enabled by devices that enable closed loop communication, multifocal stimulation, and seizure prediction. We anticipate that wireless communication and AI based prediction will be key components of these systems.

### Objective

To develop a novel stimulation device powered by low-energy ultrasound and employing a deep learning Convolutional Neural Networks (CNN) for efficient seizure prediction and treatment.

### Methods

Hardware: Closed-loop DBS prototypes were designed and fabricated using Ultrasonic Wide Band (UsWB) communication technology and miniaturized custom electronics. These systems were tested in porcine in vivo models achieving performance comparable to clinical stimulation settings and evaluated for their ability to transmit data through scalp tissue and to recharge the using UsWB.

Software: Personalized seizure prediction algorithms were trained using samples of EEG and ECG data from the EPILEPSIAE dataset (n= 27). Data was used to train CNNs and was paired with a 'Voting' algorithm that substantially enhanced prediction accuracy. Algorithms that used iEEG and ECG signals by themselves or a combination were assessed for sensitivity and specificity of detection using data from a separate iEEG/ECG dataset.

### Results

The prototype hardware achieves stimulation at standard clinical settings and wirelessly communicates between devices at rates of 64 kbit/s with no meaningful throughput degradation. Our CNN based algorithm achieved a sensitivity, and specificity > 99% in predicting a seizure 1hr before its onset using iEEG or ECG alone. A combined iEEG/ECG approach improved sensitivity, specificity, and accuracy to > 99.8%. False positives with this method were 0.23 per hour. Power consumption of this algorithm is compatible with the capabilities of the designed hardware.

### Conclusion

It is possible to implement high-accuracy epileptic seizure prediction models on miniaturized processing hardware that can be wirelessly powered.

**12:35 - 12:45 Closing Remarks & Meeting Adjourn**  
Sander Connolly



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<b>IMAD N. KANAAN</b> (Huda) King Faisal Specialist Hospital <a href="mailto:dr.imad.kanaan@gmail.com">dr.imad.kanaan@gmail.com</a>	2008	CORRESPONDING
<b>TAKESHI KAWASE</b> (Mieko) Keio University, School of Medicine <a href="mailto:kawase@sc.itc.keio.ac.jp">kawase@sc.itc.keio.ac.jp</a>	1997	CORRESPONDING
<b>ANDREW H. KAYE</b> (Judith) University of Melbourne <a href="mailto:andrewk@hadassah.org.il">andrewk@hadassah.org.il</a>	1996	CORRESPONDING
<b>HARUHIKO KIKUCHI</b> (Yuriko) Kobe City Medical Center	1993	CORRESPONDING
<b>NEIL D. KITCHEN</b> (Amanda) National Hospital for Neurology and Neurosurgery <a href="mailto:neilkitchen@nhs.net">neilkitchen@nhs.net</a>	2016	CORRESPONDING
<b>SHIGEAKI KOBAYASHI</b> (Hideko) Shinshu University <a href="mailto:shigek0305@gmail.com">shigek0305@gmail.com</a>	1998	CORRESPONDING
<b>BYUNG DUK KWUN</b> (Eun Joo Lee) ASAN Medical Center <a href="mailto:bdkwun@amc.seoul.kr">bdkwun@amc.seoul.kr</a>	2005	CORRESPONDING
<b>MARC LEVIVIER</b> (Cinthia) CHUV Lausanne <a href="mailto:Marc.Levivier@chuv.ch">Marc.Levivier@chuv.ch</a>	2016	CORRESPONDING
<b>LUIGI MARIANI</b> (Susanne) University Hospital Basel, Switzerland <a href="mailto:luigi.mariani@usb.ch">luigi.mariani@usb.ch</a>	2020	CORRESPONDING
<b>RAUL MARINO, Jr.</b> (Angela) Instituto Neurologico De Sao Paulo <a href="mailto:raulmarino@uol.com.br">raulmarino@uol.com.br</a>	1977	CORRESPONDING

<b>EDWARD W. MEE</b> (Jane Elliott) Auckland City Hospital <a href="mailto:edward.mee@xtra.co.nz">edward.mee@xtra.co.nz</a>	2005	CORRESPONDING
<b>A. DAVID MENDELOW</b> (Michelle Davis) University of Newcastle <a href="mailto:a.d.mendelow@ncl.ac.uk">a.d.mendelow@ncl.ac.uk</a>	2005	CORRESPONDING
<b>JORGE S. MENDEZ</b> (Soledad) Catholic University Medical School <a href="mailto:jorgemendez@manquehue.net">jorgemendez@manquehue.net</a>	1997	CORRESPONDING
<b>BASANT K. MISRA</b> (Sasmita) P.D. Hinduja National Hospital & MRC <a href="mailto:basantkmisra@gmail.com">basantkmisra@gmail.com</a>	2008	CORRESPONDING
<b>MICHAEL K. MORGAN</b> (Elizabeth) Royal North Shore Hospital <a href="mailto:michael.morgan@mq.edu.au">michael.morgan@mq.edu.au</a>	1999	CORRESPONDING
<b>M. NECMETTIN PAMIR</b> (Feriha) Marmara University <a href="mailto:pamirmn@yahoo.com">pamirmn@yahoo.com</a>	2006	CORRESPONDING
<b>JOHN D. PICKARD</b> (Mary) University of Cambridge <a href="mailto:jdpsecretary@medschl.cam.ac.uk">jdpsecretary@medschl.cam.ac.uk</a>	2001	CORRESPONDING
<b>WAI SANG POON</b> (Gillian Kew) Chinese University of Hong Kong <a href="mailto:wpoon@surgery.cuhk.edu.hk">wpoon@surgery.cuhk.edu.hk</a>	2008	CORRESPONDING
<b>ANDREAS RAABE</b> Inselspital <a href="mailto:andreas.raabe@insel.ch">andreas.raabe@insel.ch</a>	2019	CORRESPONDING
<b>JEAN M. REGIS</b> Hospital d'adulte de la Timone <a href="mailto:jean.regis@ap-hm.fr">jean.regis@ap-hm.fr</a>	2019	CORRESPONDING
<b>HANS-JUERGEN REULEN</b> University of Munich and Mainz <a href="mailto:hjreulen@gmx.de">hjreulen@gmx.de</a>	1998	CORRESPONDING

<b>MADJID SAMII</b> International Neuroscience Institute <a href="mailto:samii@inihannover.de">samii@inihannover.de</a>	1996	CORRESPONDING
<b>TOMIO SASAKI</b> Kyushu University School of Medicine <a href="mailto:tsasaki@ns.med.kyushu-u.ac.jp">tsasaki@ns.med.kyushu-u.ac.jp</a>	2012	CORRESPONDING
<b>GABRIELE SCHACKERT (Hans)</b> University of Technology, Dresden <a href="mailto:gabriele.schackert@uniklinikum-dresden.de">gabriele.schackert@uniklinikum-dresden.de</a>	2003	CORRESPONDING
<b>JOHANNES SCHRAMM (Dorothea)</b> University of Bonn <a href="mailto:johannes.schramm@gmx.net">johannes.schramm@gmx.net</a>	2002	CORRESPONDING
<b>VOLKER SEIFERT (Doris Faust-Seifert)</b> Johann Wolfgang Goethe-University <a href="mailto:v.seifert@em.uni-frankfurt.de">v.seifert@em.uni-frankfurt.de</a>	2009	CORRESPONDING
<b>FRANCO SERVADEI</b> Azienda Ospedailero Universitaria <a href="mailto:franco.servadei@gmail.com">franco.servadei@gmail.com</a>	2016	CORRESPONDING
<b>CHARAS SUWANWELA (Nitaya)</b> Chulalongkorn University <a href="mailto:charas.s@chula.ac.th">charas.s@chula.ac.th</a>	1972	CORRESPONDING
<b>TAKASHI TAMIYA</b> Kagawa University <a href="mailto:tamiya@kms.ac.jp">tamiya@kms.ac.jp</a>	2019	CORRESPONDING
<b>GRAHAM M. TEASDALE</b> NHS Quality Improvement Scotland <a href="mailto:y.mitchell@clinmed.gla.ac.uk">y.mitchell@clinmed.gla.ac.uk</a>	2004	CORRESPONDING
<b>DAVID G. T. THOMAS (Hazel)</b> Institute of Neurology, Univ. Coll, London <a href="mailto:Roseann.Mccrea@uclh.nhs.uk">Roseann.Mccrea@uclh.nhs.uk</a>	1995	CORRESPONDING
<b>JOERG CHRISTIAN TONN (Karin)</b> University of Munich LMU <a href="mailto:joerg.christian.tonn@med.uni-muenchen.de">joerg.christian.tonn@med.uni-muenchen.de</a>	2010	CORRESPONDING

<b>YONG-KWANG TU (Charlotte)</b> National Taiwan University Hospital <a href="mailto:yktu@ntu.edu.tw">yktu@ntu.edu.tw</a>	2007	CORRESPONDING
<b>UGUR TURE</b> Yeditepe University School of Medicine <a href="mailto:drture@yahoo.com">drture@yahoo.com</a>	2016	CORRESPONDING
<b>ANDREAS W. UNTERBERG</b> University of Heidelberg <a href="mailto:andreas.unterberg@med.uni-heidelberg.de">andreas.unterberg@med.uni-heidelberg.de</a>	2014	CORRESPONDING
<b>PETER VAJKOCZY</b> Charité - Universitätsmedizin <a href="mailto:peter.vajkoczy@charite.de">peter.vajkoczy@charite.de</a>	2023	CORRESPONDING
<b>TOSHIHIKO WAKABAYASHI (Midori)</b> Nagoya University Graduate SOM <a href="mailto:wakabat@med.nagoya.u.ac.jp">wakabat@med.nagoya.u.ac.jp</a>	2013	CORRESPONDING
<b>M. GAZI YASARGIL</b> dianne9182@gmail.com	1975	CORRESPONDING
<b>SANJAY GUPTA (Rebecca)</b> Emory University <a href="mailto:sanjay.gupta@emory.edu">sanjay.gupta@emory.edu</a>	2019	HONORARY



IN MEMORIAM  
DECEASED MEMBERS

	ELECTED	DECEASED
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
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
ALBINO BRICOLO	2002	2015
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
EVANDRO DE OLIVEIRA	2002	2021
JACQUES C. DE VILLIERS	1986	2015
RICHARD L. DESAUSSURE, JR.	1962	2008
HERMANN DIETZ	1980	2016
PEARLON 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
SALVADOR GONZALEZ- CORNEJO	1982	-
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
WOLFF M. KIRSCH	1971	2023
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
DONLIN M. LONG	1983	2023
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
BURTON M. ONOFRIO	1975	2022
PIETRO PAOLETTI	1989	1991
TAE SUNG PARK	1975	2024
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
R. MICHAEL SCOTT	1991	2023
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
BENNETT M. STEIN	1970	2022
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
KINTOMO TAKAKURA	1988	2020
SUZIE CUNNINGHAM TINDALL	1990	2016
RUSSELL L. TRAVIS	1994	2022
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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