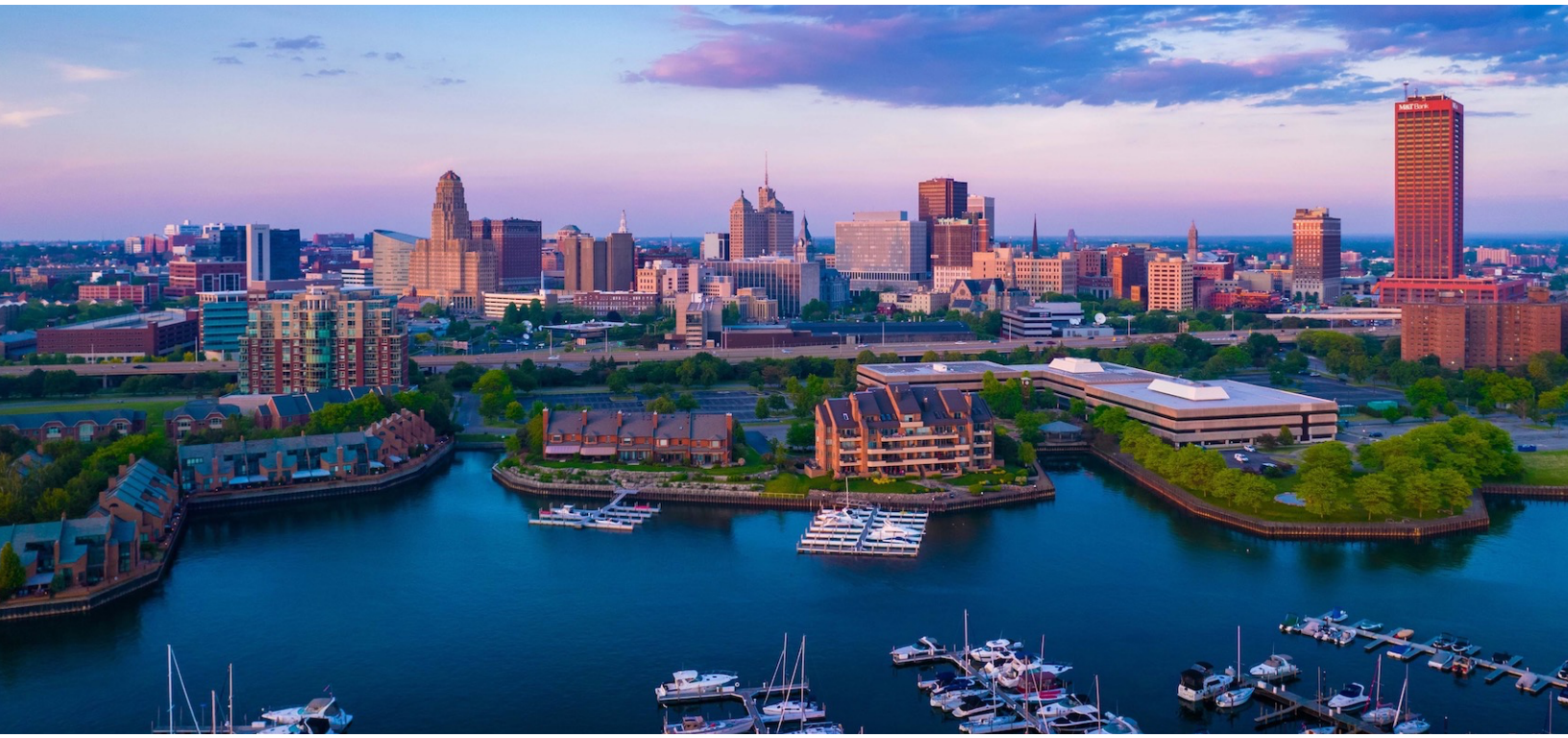
ICS2025
Buffalo, NY, USA

The 21st Interdisciplinary Cerebrovascular Symposium



July 31st – August 2nd

Visit: ICS2025Buffalo.com

Hosted at the:

Jacobs School of Medicine and Biomedical Sciences
University at Buffalo

955 Main Street, Buffalo, NY 14203

Welcome to ICS2025 in Buffalo!

Vincent M. Tutino, PhD

- Assistant Professor of Pathology and Anatomical Sciences, University at Buffalo
- CEO, Neurovascular Diagnostics, Inc.
- CFO, QAS.AI, Inc.
- Director, Hemodynamics and Vascular Biology Lab, Canon Stroke and Vascular Research Center

Dear Symposium Attendees,

Welcome to the 21st Interdisciplinary Cerebrovascular Symposium (ICS), taking place in Buffalo, NY this year. Building on the legacy of its predecessors, the ICS was established in 2004 by Prof. Daniel A. Rüfenacht, Prof. Pedro Lylyk, and Dr. Makoto Ohta as the IntraCranial Stent meeting. This gathering brought together clinicians, scientists, engineers, and industry professionals, united by a shared goal: to explore, develop, and implement innovative treatment strategies for intracranial vascular diseases.

Over the years, the symposium has evolved significantly. The initial focus on flow-diverting stents and intracranial aneurysms has expanded to encompass a broader range of cerebrovascular conditions, including ischemic stroke, hemorrhage, intracranial atherosclerotic disease, and arteriovenous malformations. Indeed, the ICS has become a platform that nurtures interdisciplinary dialogue, fostering collaboration among experts from diverse fields, all dedicated to enhancing our understanding and treatment of cerebrovascular disease.

The uniqueness of this symposium lies in the deep engagement it fosters among participants. Each session is designed to explore a specific topic from multiple perspectives, featuring presentations from clinicians, engineers, and basic scientists. This multidisciplinary approach is complemented by dynamic discussions that encourage the exchange of ideas and insights, allowing attendees to gain a richer understanding of the subject matter.

As we gather in Buffalo, a city renowned for its innovation and collaborative spirit, we invite you to join us on this journey of learning and discovery. Together, we can push the boundaries of knowledge and explore new frontiers in cerebrovascular research. Welcome to the 21st ICS!

Vincent M. Tutino, PHD

ICS2025 Chairman

ICS2025 Organization

Co-Hosts:

Ciprian Ionita, PhD

- Assistant Professor of Biomedical Engineering, University at Buffalo
- CEO, QAS.AI, Inc.

Daniel Woo, MD

- Professor and Irvin and Rosemary Smith Endowed Chair of Neurology, University at Buffalo

John E. Tomaszewski, MD, MASCP

- SUNY Distinguished Professor and Peter A. Nickerson, PhD Professor and Chair of Pathology and Anatomical Sciences, University at Buffalo

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- University Hospital of Geneva

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Linh Khanh To

Thank You to Our Sponsors!

On behalf of the ICS steering committee, we extend our deepest gratitude to our generous sponsors for their invaluable support in making this year's meeting a success. This event would not be possible without your commitment to innovation, education, and collaboration. Your contributions have helped us bring together a diverse group of professionals, thought leaders, and changemakers for a meaningful exchange of ideas and knowledge.

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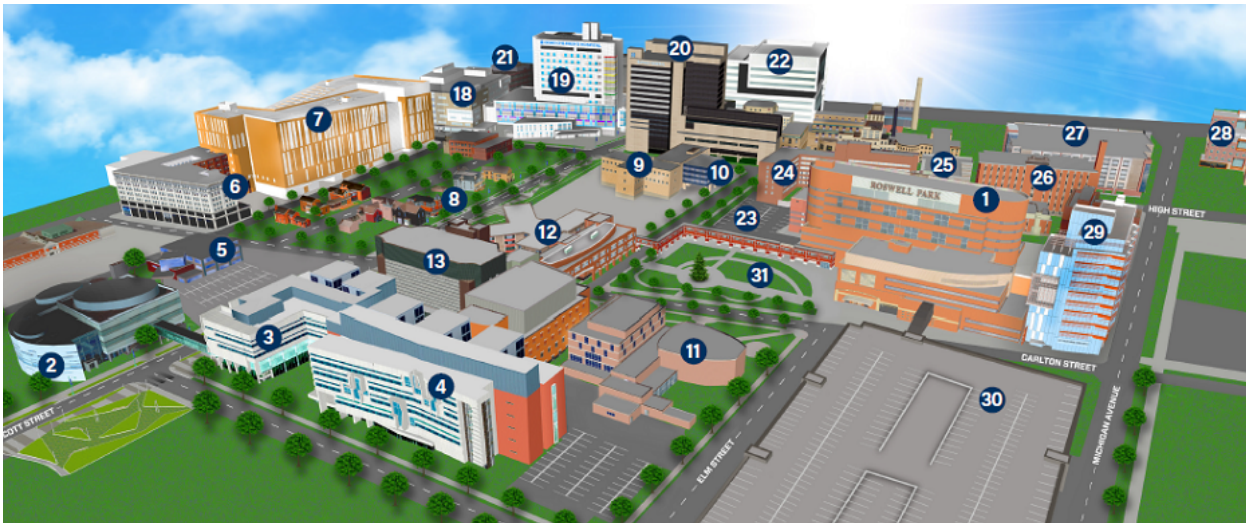

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21. UB Research Institute on Addictions
22. Kaleida Health – Gates Vascular Institute: UB Clinical and Translational Research Center and the Jacobs Institute
23. Roswell Park – Radiation Medicine Parking Lot
24. Roswell Park – Basic Science Building
25. **Wyndham Garden Hotel**
26. Roswell Park – Cell and Virus Building
27. MiGO Garage (Employee Parking)
28. Kaleida Health – HighPointe on Michigan
29. Roswell Park – Clinical Sciences Center
30. Roswell Park Parking Garage (Patient and Visitor)
31. Roswell Park – Kaminski Park

Conference Details

ICS2025 will focus on the intersection of artificial intelligence and precision medicine in the treatment of cerebrovascular diseases. As AI technologies rapidly advance, the symposium will explore their potential to uncover disease mechanisms, improve biomarker diagnostics, and enable personalized treatment strategies. In parallel, it will address critical challenges in data integrity, fusion, and harmonization—essential for making sense of complex, large-scale datasets that drive modern medical discoveries. The program will also feature innovations in next-generation medical devices that are expanding treatment possibilities. Other sessions will examine the genetic and cellular foundations of neurovascular diseases, offering deeper insight into the molecular drivers of these conditions and informing future therapies.

Meeting Rooms in the Jacobs School of Medicine and Biomedical Sciences:
ICS2025 will be held in the Jacobs School of Medicine and Biomedical Sciences (JSMBS), located at 955 Main Street, Buffalo, NY 14203.

- Scholarly presentations and keynote talks will be held on the 2nd floor of the JSMBS in rooms 2220 A&B and 2120 A&B.
- Coffee/refreshments will be provided in the 2nd floor atrium outside the lecture halls.
- Sponsor tables will also be in the 2nd floor atrium outside of the lecture halls.
- Lunch, poster sessions, and the Entrepreneurship Panel (on August 1st) will be held in the Active Learning Center on the 1st floor of the JSMBS, in Room 1220.

Conference Parking:
Parking is available in the 854 Ellicott Street Garage (at Ellicott and North Streets) for approximately $6–9/day. Street parking is also available but is metered. Payments can be made via the Buffalo Roam app (https://www.buffaloroamapp.com).

Other Important Locations:
The Opening Reception on July 31st will be at The VUE Rooftop Lounge at 6:30 PM.

- 210 Franklin Street, Buffalo, NY 14202. Valet parking is included.
- https://vuebuffalo.com.

The Closing Reception on August 2nd will be at The Terrace at Delaware Park at 6:00 PM.

- 199 Lincoln Pkwy, Buffalo, NY 14222. Street parking is available but limited.
- https://terracebuffalo.com.

Optional Excursions:
An optional excursion to Niagara Falls on August 1st will leave from the JSMBS entrance at approximately 5:00 PM.

- For those partaking in the Maid of the Mist Tour, complimentary ponchos are provided, however, you may still get wet from the spray of the Falls. Please dress accordingly.

An optional tour of the AKG Art Museum is available prior to the Closing Reception from 3:00 to 6:00 PM (this is across the street from the reception venue).

- 1285 Elmwood Avenue, Buffalo, NY 14222. Street parking is available but limited.
- https://buffaloakg.org.

Thursday, July 31, 2025

7:30-8:00a	**Registration and Coffee with Light Breakfast (2nd Floor Atrium)**	
8:00-8:30a	**Welcome and Opening Remarks (JSMBS 2220 A&B)**	**Vincent Tutino, Introduction**
8:30-10:00a	***Session 1: Stroke Biology and Genetics (JSMBS 2220 A&B)***	**Chairs: Marc Halterman, Christopher Kellner, Vincent Tutino**
8:30a	*Hemorrhagic Infarct Before Thrombectomy on MRI is Associated with Worse Outcome After Treatment: A Multicenter Bayesian Analysis*	**Robert Regenhardt** University of Texas at Houston
8:45a	*Lung-Brain Coupling in Post-Ischemic Cerebral Reperfusion Injury*	**Marc Halterman** University at Buffalo
9:00a	*Multi-Omic Characterization of Thrombectomy-Retrieved Clots Identifies Signatures of Ischemic Stroke Etiology in the INSIGHT Registry*	**Carina Seah** Mt. Sinai Health System
9:15a	*Intra-Arterial Thrombolytics After Successful Recanalization in Endovascular Thrombectomy for LVO Acute Ischemic Stroke: A Comprehensive Meta-Analysis of Randomized Controlled Trials*	**Wei Jun Lee** Boston Medical Center
9:30a	*Immunologically Muted TICI 3 Emboli: Proteomic and Functional Insights into Stroke Reperfusion*	**Santiago Mendoza-Ayus** University of Rochester
9:45a	*The Single Cell Immune Landscape of Human Clots*	**Daniela Renedo** Yale University
10:00-10:15a	**Coffee and Snack Break (2nd Floor Atrium)**	
10:15-11:45a	***Session 2: New Advancements in ICAD (JSMBS 2120 A&B)***	**Chairs: Sepideh Amin-Hanjani, Nandor Pinter, Sricharan Veeturi**
10:15a	*ICAD Imaging Biomarker Development Using Real World Data – Introducing the Buffalo ICAD Database*	**Nandor Pinter** University at Buffalo
10:30a	*Flow Measurements as a Biomarker of Stroke in Intracranial Stenosis*	**Ali Alaraj** University of Illinois Chicago
10:45a	*Oral Dysbiosis and Impaired Immune Response is Associated with Aneurysmal Disease*	**Josh Geiger** University of Rochester
11:00a	*Imaging Biomarkers for Stroke Risk in ICAD*	**Sepideh Amin-Hanjani** University Hospitals Cleveland Medical Center
11:15a	*Theranostics in Intracranial Atherosclerosis – Leveraging Diagnostics for Therapeutics*	**David Liebeskind** UCLA **Virtual*
11:30a	*Hemodynamic Evaluation of Plaque Phenotypes in Patients with Intracranial Atherosclerotic Disease*	**Sricharan Veeturi** University at Buffalo

11:45a-12:35p	**Lunch and Poster Session (JSMBS 1220)**	
12:35-2:25p	***Session 3: Innovations in Understanding & Treating ICH (JSMBS 2120 A&B)***	**Chairs: Jason Davies, Dan Hanley, Daniel Woo**
12:35-1:05p	**Keynote Session 1: ICH Gaps in Knowledge about Minimally Invasive Surgery (JSMBS 2120 A&B)**	**Dan Hanley** Johns Hopkins University
1:05p	*RNA Sequencing Acutely After ICH*	**Stacie Demel** University of Cincinnati
1:20p	*Continuous Irrigation with the IRRAflow System for Intraventricular Hemorrhage: A Single-Center Pilot Study*	**Kimberly Agosto** Mt. Sinai Health System
1:35p	*Longitudinal Differential Tractography After ICH*	**Daniel Woo** University at Buffalo
1:50p	*Minimally Invasive Evacuation of Intracranial Hemorrhages Using the Aurora Surgiscope System: Preliminary Experience*	**Kenneth Snyder** University at Buffalo
2:05-2:25p	**Keynote Session 2: Comparisons between MISTIE, ENRICH and MIND trials of MIS for ICH (JSMBS 2120 A&B)**	**Wendy Ziai** Johns Hopkins University
2:25-2:40p	**Coffee and Snack Break (2nd Floor Atrium)**	
2:40-3:55p	***Session 4: Hemorrhagic Stroke (JSMBS 2220 A&B)***	**Chairs: David Hasan, Devin McBride, Robert Regenhardt**
2:40p	*Intraarterial Encephalography from an Acutely Implanted Aneurysm Embolization Device in Awake Humans*	**David Hasan** Duke University
2:55p	*Late-Onset Vasospasm in Aneurysmal Subarachnoid Hemorrhage*	**Justin Granstein** Beth Israel Deaconess Medical Center
3:10p	*Thromboinflammation Causes Delayed Cerebral Ischemia after SAH*	**Devin McBride** University of Texas at Houston
3:25p	*Widespread Neuroinflammation in the Cerebral Cortex Following Subarachnoid Hemorrhage*	**Hiroki Yamada** Mihara Memorial Hospital
3:40p	*Adropin-Mediated Neurovascular Protection Following Subarachnoid Hemorrhage*	**Koji Hosaka** University of Florida
3:55p	**Close of Day 1 (JSMBS 2220 A&B)**	**Vincent Tutino** University at Buffalo
6:30-9:30p	**6:30p Cocktail Hour at the VUE Rooftop Bar and Restaurant** **7:30p Dinner at the VUE Rooftop Bar and Restaurant**	

Friday, August 1, 2025

7:30-8:00a	**Registration and Coffee with Light Breakfast (2nd Floor Atrium)**	
8:00-8:30a	**Keynote Session 3: Sharing the Voice of the Impacted, and How We Can Make a Difference Together (JSMBS 2220 A&B)**	**Christine Buckley** Brain Aneurysm Foundation
8:30-10:00a	***Session 5: Intracranial Aneurysm Biomechanics (JSMBS 2220 A&B)***	**Chairs: Yongho Bae, John Kolega, Anne Robertson**
8:30a	*High Wall Shear Stress and Cyclic Stretch Synergize to Trigger Intracranial Aneurysm Induction*	**Tomohiro Aoki** Jikei University
8:45a	*Protect Finns: An Ongoing Study to Develop Personalized Medicine for Persons with Intracranial Aneurysms*	**Juhana Frösén** Tampere University
9:00a	*The Role of Vasa Vasorum in Cerebral Arteries and Aneurysms – Friend and/or Foe?*	**Anne Robertson** University of Pittsburgh
9:15a	*CFD-Based Assessment of Hemodynamics in Intracranial Aneurysms Following the Virtual Deployment of the Contour Neurovascular System*	**Gabor Janiga** Otto Von Guericke University
9:30a	*Investigation of Vasa Vasorum and Hypoxia in Human Intracranial Aneurysms*	**Yasutaka Tobe** University of Pittsburgh
9:45a	*The Role of Survivin in Vascular and Cerebrovascular Mechanobiology and Pathology*	**Yongho Bae** University at Buffalo
10:00-10:15a	**Coffee and Snack Break (2nd Floor Atrium)**	
10:15-11:30a	***Session 6: Intracranial Aneurysm Natural History and Biology (JSMBS 2120 A&B)***	**Chairs: Tomohiro Aoki, Juhana Frösén, Kerry Poppenberg**
10:15a	*The Specific Molecular Mechanisms Regulating the Rupture of Intracranial Aneurysm Distinct from the Initiation or the Growth*	**Masahiko Itani** Kyoto University
10:30a	*Pharmacological Clearance of Senescent Cells Prevents Intracranial Aneurysm Rupture in Aged Mice*	**Hiroki Sato** Barrow Neurological Institute
10:45a	*Lymphatic Vessels in the Intracranial Aneurysm Wall: A Novel Component in Aneurysm Pathobiology*	**Nora Firtser** Kuopio University Hospital
11:00a	*Fibrin Accumulation after Flow Diversion: Aneurysm and Jailed Branches*	**Juan Cebral** George Mason University
11:15a	*Analysis of Symptomatic Brain Aneurysm Presentation with Three-Dimensional Aneurysm Wall Enhancement*	**Edgar Samaniego** University of Iowa
11:30a-12:00p	**Keynote Session 4: The Innovation Ecosystem (JSMBS 2120 A&B)**	**Adnan Siddiqui** University at Buffalo

12:00-1:00p	**Lunch and Entrepreneurship Panel: Navigating the Path from Academic Innovation to Commercial Reality (JSMBS 1220)**	**Moderator: Olga Petrova** Panel Participants: Canon, JI, Neurovascular Diagnostics, QAS.AI, Siemens
1:00-2:35p	***Session 7: Advancements in IA Treatment and Devices (JSMBS 2120 A&B)***	**Chairs: Nicole Cancelliere, Adam Dmytriw, Adnan Siddiqui**
1:00-1:20p	**Keynote Session 5: Emerging Tech, Novel Stroke Devices, and FDA Regulation – A Roadmap to Getting Devices to the US Marketplace (JSMBS 2120 A&B)**	**Carlos Peña** The Jacobs Institute
1:20p	*The Association between Low Fractional Flow (FF) and Revascularization Efficacy in Symptomatic Intracranial Atherosclerotic Stenosis*	**Jianping Xiang** ArteryFlow Technology Co.
1:35p	*Enhanced Super Large Bore Aspiration Catheter Navigability in Complex Neurovascular Anatomy*	**Naoki Kaneko** University of California, Los Angeles
1:50p	*Development of a First Coil Prediction Model for Unruptured Cerebral Aneurysms Using Surgeon-Oriented Morphological Features and Machine Learning*	**Takuto Furukawa** Tokyo University of Science
2:05p	*International Study of Intracranial Aneurysm Embolization using the Woven EndoBridge (WEB) Device: Synopsis of the WorldWideWEB Consortium*	**Adam Dmytriw** Oxford University
2:20p	*An Update on Robotics in NeuroIR: Are We Ready for Remote?*	**Nicole Cancelliere** Unity Health Toronto
2:35-2:50p	**Coffee and Snack Break (2nd Floor Atrium)**	
2:50-4:05p	***Session 8: Flow in Cerebrovascular Disease and Aneurysm (JSMBS 2220 A&B)***	**Chairs: Ali Alaraj, Juan Cebral, Gabor Janiga**
2:50p	*Quantifying the Harmful Oscillatory Shear Index Threshold for Endothelial Cells: A Preliminary Assessment*	**Makoto Ohta & Hanif Saifurrahman** Tohoku University
3:05p	*Cerebral Flow Dynamics Assessment with 4D Flow MRI*	**Vitaliy Rayz** Purdue University
3:20p	*The Forgotten Culprit, the Role of Hemodynamic Stresses in Draining Veins of AVMs*	**Ali Alaraj** University of Illinois Chicago
3:35p	*Role of Flow Stagnation and Vasa-Vasorum in Aneurysm Growth*	**Juan Cebral** George Mason University
3:50p	*Modelling Point-Spread Functions in Microcirculation: A Proof-of-Concept Study Based on Fick's Law and 1D Navier-Stokes Simulations of Arterial Aneurysm*	**Maryam Samavaki** Tampere University
4:05p	**Close of Day 2 (JSMBS 2220 A&B)**	**Vincent Tutino** University at Buffalo
5:00-8:00p	**Optional Excursion to Niagara Falls (Attendees responsible for own dinner arrangements)**	

Saturday, August 2, 2025

7:30-8:00a	**Registration and Coffee with Light Breakfast (2nd Floor Atrium)**	
8:00-8:30a	**Keynote Session 6: The Role of Biobanking in Facilitating Cerebrovascular Research (JSMBS 2220 A&B)**	**John Tomaszewski** University at Buffalo
8:30-10:00a	***Session 9: AI, Big Data, and Predictive Analytics (JSMBS 2220 A&B)***	**Chairs: Philippe Bijlenga, Tatsat Patel, John Tomaszewski**
8:30a	*Topologically Accurate Circle of Willis Modeling from TOF-MRA and CTA Data*	**Norman Juchler** University of Zurich
8:45a	*Intervention Planning using Functional Atlases, fMRI, TMS, DTI, Planning Tools and Intra-Operative Mixed Reality*	**Philippe Bijlenga** University of Geneva
9:00a	*The Florida Familial Brain Aneurysm Study*	**Montserrat Lara Petrick** Baptist Health
9:15a	*Disruptive AI Interventions Along the Entire Acute Stroke Care Pathway*	**Sunil Sheth** University Texas at Houston **Virtual*
9:30a	*A Machine Learning Approach to Predicting Device Size in Endovascular Treatment of Intracranial Aneurysms for Logistics Optimization*	**Soichiro Fujimura** Tokyo University of Science
9:45a	*Management Model Based on the Patient's Initial Condition and the Factors of an Incidentally Discovered Solitary Saccular Aneurysm*	**Abiram Sandralegar** University of Geneva
10:00-10:15a	**Coffee and Snack Break (2nd Floor Atrium)**	
10:15-11:45a	***Session 10: Imaging Biomarkers and Radiomics (JSMBS 2120 A&B)***	**Chairs: Ciprian Ionita, Mahmud Mossa-Basha, Edgar Samaniego**
10:15a	*Quantitative MRA in Complications of Aneurysmal Subarachnoid Hemorrhage*	**Mahmud Mossa-Basha** University of Washington
10:30a	*Imaging-Guided Precision Thrombectomy: Predicting Optimal Device Choice Using Clot Radiomics and Vascular Morphometrics for AIS Patients*	**Tatsat Patel** University at Buffalo
10:45a	*3D Volumetric Analysis for Detecting Subtle Growth in Unruptured Intracranial Aneurysms*	**Navami Shenoy** University of Iowa
11:00a	*Differences in Aneurysm Wall Enhancement on Vessel-Wall MRI using Standard and Advanced Flow Suppression Modules*	**Owais Khan** Toronto Metropolitan University
11:15a	*Next-Generation Imaging: 1000 fps High-Speed X-ray Angiography for Real-Time Blood Flow Assessment in Endovascular Diagnosis and Treatment of Aneurysms*	**Swetadri Vasan Setlur Nagesh** University at Buffalo
11:30a	*Real-Time Prognosis for Aneurysm Occlusion: Clinical Translation of the QAS.AI Decision-Support System*	**Ciprian Ionita** University at Buffalo

11:45a-12:35p	**Lunch and Poster Session (JSMBS 1220)**	
12:35-2:20p	***Session 11: Venous Disease and Glymphatics (JSMBS 2220 A&B)***	**Chairs: Jason Davies, Josh Geiger, Rashad Hussain**
12:35-1:05p	**Keynote Session 7: Post-TBI Pathophysiological Changes in Vasculature and Glymphatic Impairment (JSMBS 2220 A&B)**	**Rashad Hussain** University of Rochester
1:05p	*Insights into Venous Congestive Disorders from High-Fidelity CFD*	**Gurnish Sidora** University of Toronto
1:20p	*Under Pressure: Unraveling the Central Venous Influence in Idiopathic Intracranial Hypertension*	**Vinay Jaikumar** University at Buffalo
1:35p	*In-silico Investigation of Hemodynamic Fluctuations in Transverse Sinus Stenosis: Pitfalls in Computational Modeling*	**Gabor Janiga** Otto Von Guericke University
1:50p	*Who Gets Better? Predictors of Symptom Improvement Following Venous Sinus Stenting in Idiopathic Intracranial Hypertension*	**Jason Davies** University at Buffalo
2:05p	*Endovascular and Surgical Treatment of CSF Leak Types 1, 2, and 3*	**Matthew Bender** University of Rochester
2:20-2:40p	**Closing Session – Summary (JSMBS 2220 A&B)**	**Vincent Tutino, Ciprian Ionita, Daniel Woo** Conference Co-Hosts
2:40-5:00p	**Free Time for Attendees**	
3:00-6:00p	**AKG Art Museum Tour (Optional)**	
6:00-9:00p	**6:00p Cocktail Hour at the Terrace on Hoyt Lake** **7:00p Dinner at the Terrace at Hoyt Lake**	

Keynote Speakers

Keynote Session 1: ICH Gaps in Knowledge about Minimally Invasive Surgery

Dan Hanley Jr., MD

- Professor of Neurology, Johns Hopkins University
- Division Director, BIOS Clinical Trials Coordinating Center
- Deputy Director for Support and Innovation in Multicenter Trials at the Johns Hopkins Institute for Clinical and Translational Research
- Jeffrey and Harriet Legum Professor of Acute Care Neurology

Dr. Daniel F. Hanley has been a professor of neurology, neurosurgery, and anesthesiology/critical care medicine at the Johns Hopkins University School of Medicine since 1996 and awas named the Jeffrey and Harriet Legum Chair of Acute Care Neurology. Dr. Hanley founded and directed the Johns Hopkins Neurocritical Care Unit, one of the first critical care units dedicated solely to neurosurgical and neurological patients. In 1999, he founded and continues to direct the BIOS Clinical Trials Coordinating Center (BIOS CTCC). Under his leadership the BIOS CTCC has organized and completed more than 20 large clinical trials. He has been awarded over 70 clinical and basic research grants, predominantly from the National Institutes of Health and the FDA Orphan Products Grants Program.

Dr. Hanley's 40-year career in medicine has focused on clinical trial design, the organization and interpretation of drug and device trials, the development of strategic research plans, and FDA regulatory compliance. He has led international, NIH-sponsored trials including the MISTIE III and CLEAR III trials investigating minimally invasive neurosurgical techniques to treat hemorrhagic stroke. As principal investigator for the National Center for Advancing Translational Sciences (NCATS) Johns Hopkins Trial Innovation Center, Dr. Hanley leads collaborative efforts to advance education and therapeutics through innovative CTSA clinical trials. He is currently the PI on several ongoing trials, including a multisite phase 2/3 randomized controlled dementia prevention trial (MAP), a large multicenter clinical trial involving automated monitoring of atrial fibrillation (REACT AF), and a first-in-patient phase 2a biomarker and edema attenuation in intracerebral hemorrhage trial (BEACH).

Dr. Hanley has published over 400 peer-reviewed articles and book chapters, received the Humboldt Research Prize for accomplishments in brain injury research, and mentored nearly 100 researchers. His trainees, which include many trialists, have led 25 brain intensive care units, and over 40 have been named full professors, program leaders, or department chairs. He has served on public boards including the American Academy of Neurology, National Stroke Association, and NIH National Institute of Nursing Research.

Keynote Session 2: Comparisons between MISTIE, ENRICH and MIND trials of MIS for ICH

Wendy C. Ziai, MD

- Professor of Neurology, Johns Hopkins University
- Medical Director, Neurovascular Laboratory
- Co-Director, Johns Hopkins Bayview Neurocritical Care Unit

Dr. Wendy Ziai is a Neurologist based in Baltimore, MD, with a subspecialty in Critical Care Medicine. She obtained her MD from Queen's University at Kingston in 1993, holds a BA in Economics from Carleton University, and an MPH from Johns Hopkins Bloomberg School of Public Health.

Dr. Ziai is Professor of Neurology, Neurosurgery, and Anesthesia/Critical Medicine at the Johns Hopkins Medical Institutions in Baltimore Maryland. She is Co-Chair of the Grants Committee of Neurocritical Care Research Central (NCRC) in the Neurocritical Care Society. She is an Associate Editor for the journal Neurocritical Care. Her research is focused on mechanisms of injury and recovery in intracerebral hemorrhage with focus on multicenter clinical trials of minimally invasive surgery to treat hemorrhagic stroke.

Dr. Ziai is PI of the NIH-funded REASSESS ICH study (Repeated Assessment of Survivors in Intracerebral Hemorrhage). Dr. Ziai is involved in various clinical trials, serving as the Principal Investigator for the BEACH study and participating in multiple studies related to intracerebral hemorrhage and neurologic injury.

Keynote Session 3: Sharing the Voice of the Impacted, and How We Can Make a Difference Together

Christine Buckley

- Executive Director & Board President, The Brain Aneurysm Foundation

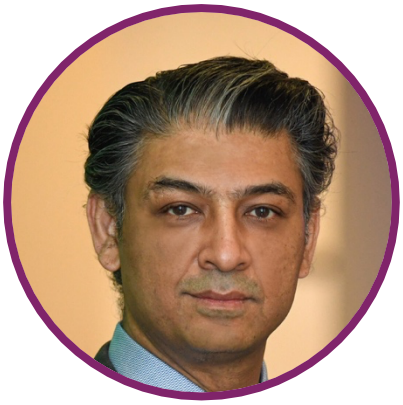
Christine Buckley is widely recognized as a steadfast advocate for brain aneurysm patients and their families. She has been associated with the Brain Aneurysm Foundation for the past 22 years, first as a volunteer and Board President, and as Executive Director since 2006.

Throughout her tenure with this national nonprofit organization, Buckley has demonstrated her commitment to the scientific and medical communities. Her ability to engage business leaders, government officials, and the public in efforts to raise awareness and much-needed research funds is respected by colleagues and philanthropists alike. Under Buckley's leadership, the Brain Aneurysm Foundation, currently the largest private funder of vital research in the US and Canada, has invested almost $5 million in support of groundbreaking studies focused on early detection, improved treatment modalities, and the technological advances that will ultimately improve outcomes.

Despite the widespread availability of brain imaging technology that can detect an aneurysm before it ruptures, misdiagnoses and delays in treatment occur in 25% of patients seeking medical care. And in three out of four cases, the primary cause of a misdiagnosis is a failure to order noninvasive diagnostic imaging. That's why Buckley, a graduate of the Isenberg School of Management at the University of Massachusetts, Amherst, created the Scan2Save initiative, a public health campaign designed to help physicians and patients quickly recognize the early signs of a brain aneurysm.

A tireless ambassador and effective fundraiser, Buckley has launched a number of successful initiatives including an annual road race that has to date generated well over $1,000,000. She is currently a member of Chief, the only private leadership network focused on connecting and supporting high level women executives and was recently elected Chairman of the South Shore Chamber of Commerce's Non-Profit group.

Keynote Session 4: The Innovation Ecosystem



Adnan Siddiqui, MD, PhD, FAHA

- CEO & CMO, Jacobs Institute
- UB Distinguished Professor & Vice Chairman, University at Buffalo
- Director Neuroendovascular Fellowship & Research, Department of Neurosurgery
- Director, Canon Stroke and Vascular Research Center
- Director Neurosurgical Stroke Service, Kaleida Health

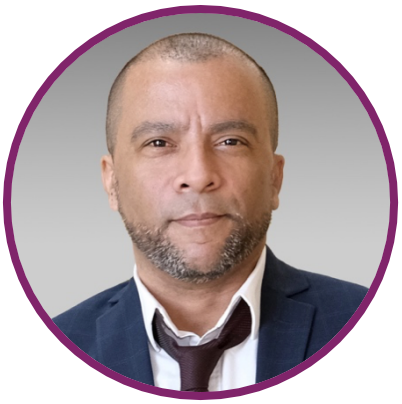
Dr. Adnan Siddiqui is Professor and Vice Chairman in the Department of Neurosurgery at the University at Buffalo's Jacobs School of Medicine and Biomedical Sciences. He joined UBNS in December 2006. Dr. Siddiqui completed fellowship training in Interventional Neuroradiology, Cerebrovascular Surgery and Neurocritical Care from Thomas Jefferson University in Philadelphia. He completed his Neurosurgical residency at Upstate Medical University and received his PhD in Neuroscience from the University of Rochester and medical degree from Aga Khan University in Pakistan. He is a Fellow of the American Association of Neurological Surgeons, American College of Surgeons and American Heart Association. He is profoundly indebted to his mentors Nick Hopkins, Robert Rosenwasser, Charles Hodge and Shirley Joseph in shaping his career as a dual-trained cerebrovascular surgeon, clinician scientist and entrepreneur.

Dr. Siddiqui has special interest and expertise in the performance of complementary microsurgical, radiosurgical and endovascular techniques for the comprehensive management of cerebrovascular conditions. This spectrum of disease includes aneurysms and arteriovenous malformations, as well as dural, cavernous and spinal fistulae. He has interests in endovascular management of acute ischemic stroke, as well as endovascular and microsurgical management of extracranial and intracranial vascular occlusive disease. Other clinical interests include endovascular management of intractable epistaxis, facial vascular malformations, head, neck, and brain tumor embolization and microsurgical resection of skull base tumors.

Dr. Siddiqui has authored over 450 peer reviewed publications and more than 50 book chapters. He is proud of representing Buffalo and the US at most major cerebrovascular conferences around the world with over 200 international presentations to date. He has designed, conducted and lead multiple major national and international clinical trials and currently serves as National and International PI for multiple major funded multi-site trials. These efforts have significantly contributed to the success of the department, which was ranked 7th in academic impact in North America by the Journal of Neurosurgery.

He is the CEO and CMO of the Jacobs Institute, which is focused on entrepreneurship, development and education opportunities with partners in the medical technology industry including strategics, startups and individual inventors to advance the care of patients with vascular diseases. He now assumes the role of Chief Executive Officer of the JI with the goal to improve the current offerings and translate technologies being developed at the Institute's idea to reality into therapeutic and commercial successes. Dr. Siddiqui also leads the Canon Stroke & Vascular Research Center at University at Buffalo. This multi-disciplinary center with multiple concurrent major NIH grants, houses neurosurgeons, physicists and engineers working collaboratively on cutting edge research focused on neurovascular biology and pathology. In addition, he serves as the Director of the Neurosurgical Stroke Service at the Gates Vascular Institute in Buffalo, one of the busiest Comprehensive Stroke Services in New York State and the United States.

Keynote Session 5: Emerging Tech, Novel Stroke Devices, and FDA Regulation – A Roadmap to Getting Devices to the US Marketplace



Carlos Peña, PhD

- Chief Regulatory Officer & Chief Quality Officer, The Jacobs Institute

Carlos Peña, PhD, MS, is Chief Regulatory Officer and Chief Quality Officer, leading the newly established Offices of Regulatory Services and Office of Quality Services at the Jacobs Institute. Carlos served 20+ years as a public servant in federal government. Prior to the JI, Carlos served as the Director of the Office of Neurological and Physical Medicine Devices, at the Center for Devices and Radiological Health (CDRH), at the U.S. Food and Drug Administration (FDA), based in the Washington, DC area.

He was responsible for providing leadership in the development of safe and effective neurological and physical medicine devices to support FDA's mission. Carlos provided oversight over several cross-functional teams and was the lead contact for the agency's strategic plans and implementation efforts for neuro devices.

Before his FDA Director role, Carlos served as Assistant Director of Emerging Technologies in the White House Office of Science & Technology Policy (OSTP) in the Executive Office of President Obama for two years (2012-2014). Prior to the White House, Carlos began his career ascension at the FDA, starting out as a Lead Reviewer, Team Leader, and Acting Branch Chief for CDRH, before moving into the role of Science Policy Analyst and, finally, the Director of Emerging Technology Programs in the FDA Commissioner's Office.

Carlos holds a PhD in Neuroscience from Case Western Reserve University and a Master's degree in Comparative Physiology from the University of Connecticut. He holds an Emergency Medical Technician license in West Virginia and currently volunteers as a Fire Fighter and for Emergency Medical Services in both New York and West Virginia.

Keynote Session 6: The Role of Biobanking in Facilitating Research Cerebrovascular Research

John E. Tomaszewski, MD, MASCP

- SUNY Distinguished Professor and Peter A. Nickerson, PhD Professor and Chair of Pathology and Anatomical Sciences, University at Buffalo

Dr. Tomaszewski received his undergraduate education from LaSalle College in Philadelphia in 1973. He attended the University of Pennsylvania School of Medicine and received his MD in 1977. After finishing medical school Dr. Tomaszewski did an internship in Internal Medicine at Pennsylvania. He completed his Pathology Residency in anatomic and clinical pathology at the Hospital of the University of Pennsylvania in 1982, during which he had special concentrations in immunology, HLA testing, and nephropathology. He was a Fellow in Surgical Pathology in 1982-1983, then joined the faculty in the Department of Pathology and Laboratory Medicine at the University of Pennsylvania in 1983. In his first year of appointment, Dr. Tomaszewski was given the opportunity to do a specially arranged fellowship with Dr. Conrad Pirani in nephropathology at Columbia University. At Penn he rose through the ranks to become Professor, Vice Chair for Anatomic Pathology-Hospital Services, and Interim Chair of the Department of Pathology and Laboratory Medicine

Dr. Tomaszewski's research interests are translational and have been focused on the domain of genitourinary pathology. Over the last decade he has had the opportunity to work collaboratively with a group of image scientists in the development of quantitative image analysis tools tailored to the needs of the digital pathology community. His overall vision has been to create a new analytic paradigm fusing the data from the quantitative analysis of high-resolution images with multidimensional molecular data. This "fused diagnostics" approach will support personalized predictive modeling of disease and its response to therapy. His collaborative group is funded and is working hard to develop platforms which will support this new way of addressing complex multivariable testing.

Over the years Dr. Tomaszewski has had the great good fortune to teach many classes of undergraduate medical students in nephropathology and genitourinary pathology. He has been Program Director of the Surgical Pathology and Immunopatholgy Fellowships at the Hospital of the University of Pennsylvania and instructed 56 Fellows. He has also been a member of 9 PhD and 1 MS candidates' thesis committees and continues to instruct at the UME, GME, and Graduate student levels. Dr. Tomaszewski has also been active in the work of many Pathology societies both as a speaker and in varied leadership roles. His volunteer work has been with the ACSP, USCAP, CAP, Pathology Informatics, ASIP, ICPI, and APC. He was a member of the ASCP Board of Directors for many years and rose through the leadership sequence to be elected ASCP President for 2010-2011.

Keynote Session 7: Post-TBI Pathophysiological Changes in Vasculature and Glymphatic impairment

Rashad Hussain, PhD

- Research Associate Professor - Department of Neurology, Center for Translational Neuromedicine, University of Rochester

Dr. Hussain is a distinguished neurobiologist and associate professor at the Centre for Translational Neuromedicine, and the Department of Neurology at the University of Rochester, NY. He got Ph.D. from the University of Paris-Saclay, France, and extensive postdoctoral training at prestigious institutions including University of Strasbourg (France), Temple University in Philadelphia, and the University of Colorado, Denver, Dr. Hussain has established a multifaceted research portfolio focused on glial function and brain health.

Dr. Hussain's work delves into the cellular and molecular mechanisms underlying neural regeneration, white matter pathology—such as oligodendrocyte degeneration and myelin deficits—sleep disorders, traumatic brain injury (TBI), and glymphatic system dysfunction leading to Alzheimer's disease-related dementias (ADRD). This comprehensive approach bridges fundamental neuroscience and translational medicine, addressing critical gaps in understanding brain health and disease.

A recent highlight of Dr. Hussain's groundbreaking research centers on cerebral edema, a major contributor to morbidity and mortality in TBI patients. Their team's discovery of the role of cervical lymphatic vessels in resolving cerebral edema, enhanced through noradrenergic inhibition, represents a significant advancement in the field. This research has led to the development of innovative quantitative techniques for assessing brain fluid clearance, with implications for both health and injury contexts (Hussain et al., 2023, Nature, 623(7989):992).

Dr. Hussain's research is consistently supported by competitive funding from the National Institutes of Health (NIH) and the Department of Defense (DOD), underscoring the high impact and translational potential of their work. Current research interests include investigating glial function in health and disease with a focus on glymphatic flow, elucidating cellular and molecular mechanisms and signaling pathways that sustain glial homeostasis in the brain, exploring glia-neuron interactions and the influence of environmental factors, and examining the neuromodulatory effects of tDCS and TMS in restoring or augmenting cerebrospinal fluid (CSF) flow within the brain. Dr. Hussain's contributions continue to shape the field of neuroscience, offering innovative insights into brain function and paving the way for novel therapeutic approaches.

SCHOLARLY PRESENTATIONS

Session 1, Abstract 1

HEMORRHAGIC INFARCT BEFORE THROMBECTOMY ON MRI IS ASSOCIATED WITH WORSE OUTCOME AFTER TREATMENT: A MULTICENTER BAYESIAN ANALYSIS

Authors: Thiago O Goulart[3], Markus D Schirmer[2], Anna K Bonkhoff[2], Erik L Bogdanoff[5], Penina Krieger[1], Ben Teasdale[2], Alvin S Das[4], Adam A Dmytriw[1], James D Rabinov[1], Christopher J Stapleton[1], Aman B Patel[1], Michael I Nahhas[6], Sunil A Sheth[6], Claus Z Simonsen[5], Robert W Regenhardt[6]

Affiliations: [1]Massachusetts General Hospital; [2]Harvard Medical School; [3]University of São Paulo; [4]Beth Israel Deaconess; [5]Aarhus University; [6]University of Texas

Background: Hemorrhagic infarction (HI), defined as petechial bleeding within infarcted tissue, is often considered benign. However, its clinical significance before mechanical thrombectomy (MT) remains unclear. We sought to evaluate whether HI, detected on sensitive pre-MT MRI, is associated with 90-day functional outcome, post-MT parenchymal hematoma (PH), and symptomatic intracerebral hemorrhage (SICH).

Methods: We conducted a retrospective multicenter study of consecutive patients with anterior circulation large vessel occlusion (LVO) who underwent MT and had pre-treatment MRI. HI was assessed on T2*-weighted imaging by ECASS III criteria. Outcomes included 90-day functional dependence (modified Rankin Scale [mRS] 3–6), ipsilateral PH, and SICH. Multivariable logistic regression, Firth regression, and Bayesian models were used to assess associations, adjusting for clinical and procedural covariates.

Results: Among 491 patients, median age was 72.0y (IQR:59.0–80.0), 46.4% were female, median NIHSS was 16.0 (IQR:11.0–20.0), 49.9% received IVT, and 83.1% achieved successful reperfusion (mTICI2b–3). Pre-MT HI was present in 5.5%, post-MT PH in 9.2%, SICH in 3.1%, and 47.3% achieved functional independence. HI was independently associated with functional dependence (Bayesian OR=6.09, 95%CrI:2.07–20.29). HI also showed high posterior probability of association with ipsilateral PH (OR=2.38, 95%CrI:0.61–7.70; Pr[OR>1]=0.91) and SICH (OR=4.40, 95%CrI:0.55–24.06; Pr[OR>1]=0.93), though CrIs included unity.

Conclusions: HI on pre-treatment MRI is associated with worse 90-day outcomes and may signal increased risk of subsequent hemorrhage. While not a contraindication to MT, HI may not be completely benign and may serve as a prognostic marker and future therapeutic target for neuroprotective strategies to prevent reperfusion injury.

Session 1, Abstract 2

LUNG-BRAIN COUPLING IN POST-ISCHEMIC CEREBRAL REPERFUSION INJURY

Authors: Marc Halterman[1]

Affiliations: [1]University at Buffalo, Buffalo, NY

Evidence indicates that reciprocal signaling between the lung and brain modulates post-ischemic cerebral injury. This presentation will explore the concept of lung-brain coupling in the context of cerebral reperfusion injury, with a focus on how systemic inflammatory signaling, pulmonary vascular responses, and circulating immune mediators influence neurovascular stability following stroke. Using preclinical models and translational data, we investigate the bidirectional crosstalk between the injured brain and peripheral organs, emphasizing the lung as both a target and amplifier of post-ischemic injury. Insights from these studies highlight new avenues for therapeutic intervention aimed at improving neurologic recovery through systemic modulation.

Session 1, Abstract 3

MULTI-OMIC CHARACTERIZATION OF THROMBECTOMY-RETRIEVED CLOTS IDENTIFIES SIGNATURES OF ISCHEMIC STROKE ETIOLOGY IN THE INSIGHT REGISTRY

Authors: Carina Seah[1], Alex Devarajan[1], James M. Vicari[1], Graham Branscom[1], Alan R. Dabney[2], Selva Baltan[3], Farida Sohrabji[4], Keith R. Pennypacker[5], Ashish Nanda[6], Keith Woodward[7], Dennis Rivet[8], Justin F. Fraser[9], and Christopher P. Kellner[1] (on behalf of the INSIGHT Study Investigators)

Affiliations: [1]Department of Neurosurgery, Mount Sinai Health System, New York, NY, USA; [2]Department of Statistics, Texas A&M University, College Station, TX, USA; [3]Department of Anesthesiology & Perioperative Medicine/Research, Oregon Health & Science University, Portland, OR, USA; [4]Department of Neuroscience and Experimental Therapeutics, Texas A&M University, Bryan, TX, USA; [5]Department of Neurology, University of Kentucky, Lexington, KY, USA; [6]Department of Stroke and Interventional Neurology, SSM Neuroscience Institute, SSM Health, St. Clare Hospital, Fenton, MO, USA; [7]Tennessee Neurovascular Institute, Knoxville, TN, USA; [8]Virginia Commonwealth University, Richmond, VA, USA; [9]Department of Neurological Surgery, University of Kentucky College of Medicine, Lexington, KY, USA

Introduction: Precise determination of the etiology of acute ischemic stroke is crucial in long-term management. Identification of etiology-associated molecular biomarkers following thrombectomy may allow for personalized intervention. Here, we examine the transcriptomics and proteomics of thrombi with known etiology of atrial fibrillation, identifying distinct -omic signatures relative to other causes of ischemic stroke.

Methods: The INSIGHT registry is a prospective, multicenter, multi-omic registry focused on elucidating the molecular underpinnings of stroke from analysis of clot and intraarterial blood. RNA sequencing of 10,990 genes and mass spectrometry of 4148 proteins from 292 thrombi from patients undergoing thrombectomy was analyzed. 111 patients had a determined etiology of atrial fibrillation. Controls had unknown, atheroembolic, or coagulopathy stroke etiology. Association of thrombus gene and protein expression with atrial fibrillation were conducted using the Wilcoxon Rank Sum test using Benjamini-Hochberg multiple testing correction (FDR <5%). Gene set enrichment was performed using FGSEA. Co-expression modules were detected using WGCNA and analyzed using cytoscape.

Results: 33 genes (27 upregulated, 6 downregulated) within thrombi were significantly associated with atrial fibrillation etiology. These included upregulated endothelial cell markers associated with cardiac endothelial cells (e.g. KDR, AMDHD2, IL18BP) compared to thrombi of other etiologies. In addition, upregulated genes were associated with oxidative stress (FDR= 8.46x10-8) and metabolism (1.04x10-6), while adaptive T-cell immunity was downregulated (FDR= 7.52x10-3). The protein AGAP3, associated with ion channel stability and gap junction morphology, is upregulated in atrial fibrillation, only in clots but not peripheral blood.

Conclusion: Cardioembolic thrombi possess distinct molecular signatures consistent with collection and retainment of cardiac endothelial tissue at origin, and display immunomodulatory activity associated with innate vs adaptive immune balance. This signature suggests the active role thrombi likely play in the vascular microenvironment and may ultimately be used to identify atrial fibrillation as an etiology in strokes of unknown origin.

Session 1, Abstract 4

INTRA-ARTERIAL THROMBOLYTICS AFTER SUCCESSFUL RECANALIZATION IN ENDOVASCULAR THROMBECTOMY FOR LARGE-VESSEL OCCLUSION ACUTE ISCHEMIC STROKE: A COMPREHENSIVE META-ANALYSIS OF RANDOMIZED CONTROLLED TRIALS

Authors: Wei Jun Lee[1], Rafaela Correia Maciel[2], Henrique Alexsander Ferreira Neves[3], Luana Miyahira Makita[3], Ong Zhi Inn[4], Tam QM Tran[5], Tan Ya Wen[6], Robert Regenhardt[7]

Affiliations: [1]Department of Medicine, Boston Medical Center - Brighton, Boston, Massachusetts, USA; [2]School of Medicine, Jundiaí Faculty of Medicine, Jundiaí, São Paulo, Brazil; [3]School of Medicine, Federal University of Paraná, Paraná, Brazil; [4]IMU University, Bukit Jalil, Federal Territory of Kuala Lumpur, Malaysia; [5]John T. Milliken Department of Medicine, Washington University, Saint Louis, Missouri, USA; [6]Penang General Hospital, Georgetown, Penang, Malaysia; [7]Department of Neurology, UTHealth Houston, Houston, Texas, USA

Background: Intra-arterial thrombolytics (IAT) as adjunctive therapy for large vessel occlusion acute ischemic stroke (LVO-AIS) after successful endovascular thrombectomy (EVT) may improve outcomes. This meta-analysis evaluates the efficacy and safety of IAT in this context.

Methods: We identified randomized controlled trials (RCTs) comparing IAT versus placebo or no IAT in LVO-AIS patients with successful recanalization post-EVT, including published studies and recent conference data. The primary outcome was excellent functional outcome (modified Rankin Scale [mRS] 0–1) at 90 days. Secondary outcomes included other mRS 0-2 at 90 days, symptomatic intracranial hemorrhage (sICH), and 90-day mortality. Pooled risk ratios (RRs) with 95% confidence intervals (CIs) were calculated using a random-effects model; heterogeneity was assessed using the I^2 statistic.

Results: Six RCTs comprising 1,972 patients (990 IAT, 982 control) were included. IAT was associated with a higher likelihood of excellent outcome at 90 days (RR = 1.25; 95% CI: 1.07–1.46; p = 0.01; I^2 = 16%). There were no significant differences in functional independence (mRS 0–2) (RR = 1.06; 95% CI: 0.97–1.17; p = 0.15), sICH (RR = 1.14; 95% CI: 0.70–1.85; p = 0.52), or 90-day mortality (RR = 1.00; 95% CI: 0.79–1.27; p = 0.99); heterogeneity was low across these outcomes (I^2 = 0%).

Conclusion: Adjunct IAT after successful EVT significantly improves excellent functional outcomes at 90 days without increasing sICH or mortality. However, its effect on broader disability outcomes is uncertain. Further trials are needed to refine patient selection.

Session 1, Abstract 5

IMMUNOLOGICALLY MUTED TICI 3 EMBOLI: PROTEOMIC AND FUNCTIONAL INSIGHTS INTO STROKE REPERFUSION

Authors: Santiago Mendoza Ayus[1], Sajal Medha Akkipeddi[1], Pablo Valdes Barrera[1], Rohin Singh[1], Prasanth Romiyo[1], Catherine Jay[1], Stephen Susa[1], Derrek A Schartz[1], Kevin Welle[2], Vincent Nguyen[1], Tarun Bhalla[1], Thomas K Mattingly[1], Craig Morrell[3], Matthew T Bender [1]

Affiliations: [1]Department of Neurosurgery, University of Rochester Medical Center, Rochester, New York, USA; [2]Mass Spectrometry Resource Laboratory, University of Rochester Medical Center, Rochester, New York, USA; [3]Department of Medicine, Cardiovascular Research Institute, University of Rochester Medical Center, Rochester, New York, USA

Introduction: Complete reperfusion (TICI 3) is associated with better functional outcomes after endovascular thrombectomy. Whether this may be partially attributable to intrinsic features of the thrombus is unclear. We aimed to characterize the proteomic composition and identify the biological pathways enriched in thromboemboli retrieved from patients with TICI 2B versus TICI 3 reperfusion.

Methods: Fifty-nine retrieved stroke thromboemboli were analyzed, 67.8% TICI 3 Vs 32.2% TICI 2B. Proteomic profiling was performed using mass spectrometry, and protein enrichment and their associated biological functions were compared between TICI 2B and TICI 3 samples. A heat map of differentially enriched proteins was developed to visualize hierarchical clustering of retrieved thrombi. The differentially expressed proteins between the two groups were displayed and identified in a volcano plot. Gene Ontology (GO) enrichment analysis was used to identify over-represented GO terms associated with thrombi in the two groups.

Results: A total of 2790 proteins were identified. 37 (1.32%) showed differential expression based on recanalization status. The heat map showed separate clustering of TICI 2B and TICI 3 clots, indicating within-group similarity. The volcano plot displayed differentially expressed proteins, with Aldo-keto reductase family 1 member B1 (AKR1B1), known for its role in oxidative stress response and inflammation, as the most enriched in TICI 2B clots ($p = 0.002$). Functional analysis revealed that TICI 2B clots were enriched in inflammatory and stress-related processes, extracellular and vesicle-associated proteins, hydrolase activity, and pathways related to innate immunity and neutrophil degranulation, suggesting a distinct inflammatory and immune activation profile compared to TICI 3 clots.

Conclusion: The immunological silence of TICI 3 clots may explain the better reperfusion and functional outcomes observed in these patients. These insights into the biological characteristics of thrombi highlight the need for novel therapeutic targets for stroke management.

Session 1, Abstract 6

THE SINGLE CELL IMMUNE LANDSCAPE OF HUMAN CLOTS

Authors: Daniela Renedo[1-2], Tanyeri Barak[1], Jonathan DeLong[2], Julian N. Acosta[2], Nanthiya Sujijantarat[1], Andrew Koo[1], Cyprien Rivier[2], Santiago Clocchiatti-Tuozzo[2], Shufan Huo, MD[2], Joseph Antonios[1], James Giles[2], Guido J Falcone[2-3], Kevin N Sheth[1-2-3], Ryan Hebert[1], Murat Gunel[1], Lauren H Sansing[2], Dhasakumar S Navaratnam[2], Charles Matouk[1]

Affiliations: [1]Department of Neurosurgery, Yale School of Medicine, New Haven, CT; [2]Department of Neurology, Yale School of Medicine, New Haven, CT; [3]Yale Center for Brain & Mind Health, Yale School of Medicine, New Haven, CT

Background: Ischemic stroke causes substantial morbidity and mortality. We aimed to explore the cellular and molecular landscape of stroke clots to better understand disease mechanisms and uncover potential biomarkers of stroke etiology.

Methods: We performed single-cell RNA sequencing on 10 thrombi retrieved from patients with large vessel occlusion stroke. We analyzed immune cell composition and gene expression patterns, comparing clots from strokes attributed to atrial fibrillation (AF) and carotid atherosclerosis (CA). Using MAGMA and GIGASTROKE GWAS summary statistics, we assessed genetic associations between stroke-related variants and cell type–specific gene expression.

Results: Both AF and CA clots exhibited heterogeneous immune cell populations, including monocytes, macrophages, dendritic cells, neutrophils, and T-cells. Differential gene expression analysis revealed distinct molecular signatures across etiologies. CA clots showed enrichment of atherosclerosis-related genes such as **CD74**, **HLA-DRB1*01**, **HTRA1**, **C1Q**, **CD81**, and **CR1**. In contrast, AF clots demonstrated upregulation of cytotoxic and inflammatory genes in CD8 T-cells and NK cells (e.g., **GZMH**, **GZMB**, **S100A4**, **HLA-A**, **IFITM2**, **TIMP1**, **CLIC1**), suggesting enhanced immune-mediated injury. MAGMA analysis identified strong genetic enrichment in leukocyte lineages—especially B-cells, T-cells, and macrophages—in both stroke subtypes.

Conclusions: Our single-cell analysis reveals distinct immune and transcriptional profiles in stroke clots of different etiologies. These findings highlight the potential of thrombus-level transcriptomics to inform stroke mechanisms and support ongoing efforts to integrate multi-omic approaches for biomarker discovery and precision stroke prevention.

Session 2, Abstract 1

ICAD IMAGING BIOMARKER DEVELOPMENT USING REAL WORLD DATA – INTRODUCING THE BUFFALO ICAD DATABASE

Authors: Nandor Pinter[1,2]

Affiliations: [1]DENT Neurological Institute, Buffalo, NY; [2]Department of Radiology, University at Buffalo, Buffalo, NY

Neuroimaging biomarker development in recent years has mostly focused on acute stroke, while fewer studies have investigated the role of such biomarkers in the chronic course of ICAD. Multi-center, prospective cohorts are needed to understand how current and proposed biomarkers can explain pathophysiology, map the longitudinal evolution of disease, and assist in risk assessment and outcomes prediction for not only recurrent stroke but also more chronic conditions such as silent ischemia or cognitive decline. Between 2019 and 2024, we have implemented a multi-parametric MRI protocol for longitudinal follow-up of patients with prior cerebral ischemia. The protocol included vessel wall imaging, arterial flow quantification, arterial spin labeling for perfusion and flow delay assessment, as well as structural brain scans. 334 patients were scanned with this protocol, 30% of them with multiple time points. In this presentation, will give an overview of the initial findings of this cohort, and review the most important MRI biomarkers and their potential value in understanding ICAD in longitudinal cohorts.

Session 2, Abstract 2

FLOW MEASUREMENTS AS A BIOMARKER OF STROKE IN INTRACRANIAL STENOSIS

Authors: Ali Alaraj[1]

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Quantitative flow measurements are emerging as important biomarkers in the evaluation and management of cerebrovascular disease, particularly ischemic stroke. Among these, Quantitative Magnetic Resonance Angiography (QMRA) offers a non-invasive and radiation-free technique to assess blood flow in major intracranial and extracranial arteries. Unlike conventional imaging modalities that provide anatomical detail, QMRA directly quantifies volumetric blood flow, offering critical physiological information about cerebral perfusion. This is especially valuable in patients with intracranial stenosis, atherosclerotic disease, or arterial dissection, where traditional imaging may not reveal the full extent of hemodynamic compromise.

There is a well-established relationship between reduced cerebral blood flow and increased risk of stroke, particularly in cases of impaired collateral circulation or progressive vessel narrowing. QMRA allows for the early detection of hemodynamic insufficiency before clinical symptoms occur, supporting timely interventions such as intensified medical therapy, revascularization procedures, or surgical bypass. Studies have demonstrated that patients with low flow in the posterior or anterior circulation have significantly higher stroke recurrence rates, underscoring the prognostic value of flow-based assessment.

In addition to its diagnostic utility, QMRA enables longitudinal monitoring of patients to assess disease progression and response to therapy, making it a valuable tool in both acute stroke triage and chronic disease management. As stroke care continues to evolve toward more individualized, physiology-driven approaches, QMRA provides a critical bridge between anatomical imaging and functional insight. Its integration into clinical workflows can improve risk stratification, guide therapeutic decision-making, and ultimately enhance outcomes for patients at risk of stroke.

Session 2, Abstract 3

ORAL DYSBIOSIS AND IMPAIRED IMMUNE RESPONSE IS ASSOCIATED WITH ANEURYSMAL DISEASE

Authors: Joshua T Geiger[1], Joel Kruger[1], Ann L Gill[2], Baqir Kedwai[1], Daniel Lehane[1], Benjamin Ford[1], Michael C Stoner[1], Steven R Gill[2], Doran Mix[1]

Affiliations: [1]Division of Vascular Surgery; [2]Department of Microbiology and Immunology; University of Rochester Medical Center, Rochester, New York

Introduction: Inflammation is a hallmark of abdominal aortic aneurysms (AAA), but the underlying etiology remains unclear. Previous work suggests that bacteria common in the oral cavity may contribute to disease, but no clear mechanism has been identified. Bacterial extracellular vesicles (EVs) can modulate the host immune system, providing a possible mechanism.

Methods: Bacterial sequencing targeting the V1–V3 16S rRNA gene was performed on oral wash samples from 69 pre-operative patients. Bulk RNA sequencing was performed on EVs isolated from oral wash samples and matching plasma samples on 15 patients. Linear differential abundance analysis identified oral taxonomies associated with AAA. Pathway analysis was applied to differentially expressed EV RNA sequences, and multi-omic factor analysis (MOFA) identified microbial and EV RNA features jointly associated with AAA.

Results: Several bacterial taxonomies were differentially abundant between AAA and Atherosclerosis. Patients with atherosclerosis had a higher alpha diversity of bacterial taxonomies compared to patients with aortic disease. EV RNA sequencing revealed a downregulation of the host immune system within the oral cavity, specifically in the innate response to bacterial products. Interestingly, no differentially regulated pathways were identified within EV RNA sequences from the plasma. Finally, MOFA identified 2 factors that explained 55.1% of the variability within the 15-subject dataset and factor 2 could discriminate AAA from atherosclerotic controls ($p=0.02$).

Conclusions: AAA is associated with oral dysbiosis and a down-regulation of the host response to bacterial products in the oral cavity. This is not seen in the plasma. This phenomenon may be used to screen patients with AAA and suggests interactions between the oral microbiome and the host immune system may contribute to the disease.

Session 2, Abstract 4

IMAGING BIOMARKERS FOR STROKE RISK IN ICAD

Authors: Sepideh Amin-Hanjani[1,2]

Affiliations: [1]University Hospitals Cleveland Medical Center; [2]Case Western Reserve University School of Medicine, Cleveland, OH

Intracranial atherosclerotic disease (ICAD) and resultant intracranial stenosis (ICAS) is a global leading cause of stroke, and poses an ongoing treatment challenge. Among patients with ICAD, the mechanisms of stroke can be broadly categorized into hypoperfusion from hemodynamic compromise, artery-to-artery embolic and perforator branch atheromatous etiology, with each mechanism having a different anticipated response to medical vs. interventional strategies.

Imaging biomarkers of ICAD, beyond severity of stenosis, demonstrate promise in identifying high risk patients. Plaque characteristics on MR-based vessel wall imaging (VWI) such as enhancement and intraplaque hemorrhage correlate with symptomatic status and recurrent risk. Various forms of hemodynamic assessment, focused on local or regional hemodynamics have been evaluated in ICAD patients. Local hemodynamic alterations reflected in pressure and wall shear stress ratios on computational fluid dynamics (CFD) modeling has been associated with elevated recurrent stroke risk.

Other markers of hypoperfusion are present in up to 50% of symptomatic ICAD patients and have also been shown to correlate with a high risk of recurrent stroke. In particular, regional hypoperfusion assessed by cerebral large vessel flow measurements using quantitative magnetic resonance angiography (QMRA) has proven to be a robust predictor of stroke in symptomatic vertebrobasilar disease, based on prospective observational data from the VERiTAS study. In VERiTAS, patients classified as low flow based on evaluation of relevant distal territory regional flow had a four-fold higher risk of subsequent stroke. Similar to the predictive value in the posterior circulation, analysis of anterior circulation flow data from the prospective observational MYRIAD study showed a five-fold increased stroke risk in patients categorized as low flow.

Validation of imaging biomarkers in ICAD is currently underway in the ongoing CAPTIVA MRA trial, focusing on QMRA, CFD and VWI. Identification of high-risk flow-compromised patients has important implications for future investigation of therapeutic interventions.

Session 2, Abstract 5

THERANOSTICS IN ICAD - LEVERAGING DIAGNOSTICS FOR THERAPEUIOTCS

Authors: David Liebeskind[1]

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Session 2, Abstract 6

HEMODYNAMIC EVALUATION OF PLAQUE PHENOTYPES IN PATIENTS WITH ICAD

Authors: Sricharan S Veeturi[1,2], Nandor Pinter[1,2], Elad Levy[1,2], Adnan Siddiqui, [1,2] Vincent Tutino[1,2,3]

Affiliations: [1]Canon Stroke and Vascular Research Center; [2]Department of Neurosurgery, University at Buffalo, Buffalo, NY; [3]Department of Pathology and Anatomical Sciences, University at Buffalo, Buffalo, NY

Background: Intracranial atherosclerotic disease (ICAD) is a systemic disease that causes buildup of plaques in the intracranial arteries. Although statins are standard care, plaques either progress or have no change in ~35% of the cases. We evaluated differences in hemodynamics between progressing, stable and regressing plaques longitudinally on vessel wall imaging.

Methods: We included patients with ICAD who underwent vessel wall imaging protocol and PC-MRI (NOVA) in this study. We segmented the lumen on both time points and calculated the WASID and a change >10% was used to determine if a plaque was regressing, remained stable or was progressing. We also used PC-MRI data to perform steady state computational fluid dynamics simulations across the vicinity of the plaque. We computed the wall shear stress (WSS), Normalized WSS (WSSNorm), WSS divergence (WSSD), WSS gradient (WSSG) and volume-based parameters (Vorticity, strain rate, helicity, lambda2 criterion, Q criterion and average velocity) at the plaque location and downstream of the plaque location. We also quantified the pressure drop across the plaque. We performed univariate analysis and AUC analysis of binary classification of all the plaques for different features.

Results: We analyzed 12 patients with 29 plaques (6 regressing, 18 stable and 5 progressing plaques). We observed that although none of the hemodynamics variables were significantly different, progressing plaques had a higher drop in pressure across the plaque as regressing and stable plaques (Regressing= 1206 Pa, stable=1267.84 Pa and progressing=1978.6 Pa). We also observed through AUC analysis that although WSS based parameters at the plaque location had a higher AUC in delineating plaque progression, vorticity parameters had a higher AUC for downstream location suggesting that downstream hemodynamics affects plaque behavior as well.

Conclusions: Our findings suggest that wall shear stress–based features at the plaque site and volumetric hemodynamics downstream may have potential to distinguish progressing plaques in ICAD patients, warranting further validation in larger cohorts.

Session 3, Abstract 1

RNA SEQUENCING ANALYSIS ACUTELY AFTER ICH

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Session 3, Abstract 2

CONTINUOUS IRRIGATION WITH THE IRRAFLOW SYSTEM FOR INTRAVENTRICULAR HEMORRHAGE: A SINGLE-CENTER PILOT STUDY

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Background: Intraventricular hemorrhage (IVH) is associated with poor outcomes due to persistent elevated intracranial pressure and inflammation. Standard external ventricular drainage (EVD) is limited by frequent catheter occlusion and incomplete hematoma evacuation. The IRRAflow catheter system offers continuous irrigation and drainage, aiming to enhance hematoma clearance and reduce complications. The DIVE trial is a phase 1, prospective and retrospective single-center study evaluating the safety and feasibility of IRRA flow treatment in IVH patients, with primary endpoints of serious adverse event (SAE) rates and hematoma volume reduction at day 5 post-bleed.

Methods: A retrospective cohort of the last 60 traditional EVD placements was compared to a prospective IRRAflow arm at a single center. Comprehensive clinical and imaging data were collected, including demographics, catheter duration, segmented IVH volumes, and serious adverse events. This feasibility study analyzed IVH volumes from radiographic imaging data at 5 days post-bleed to quantify hematoma clearance, and SAEs were monitored throughout treatment.

Results: Ten patients (mean age = 68.5 ± 14.3 years) were treated with IRRAflow between 2022 and 2024. The average duration of catheter placement for the IRRAflow arm was 7.4 days, a 16.7% reduction compared to 8.9 days in EVD patients. The mean IVH volume at day 5 showed a 35% reduction with IRRAflow versus EVD (11.3 ± 8.4 mL vs. 17.4 ± 22.6 mL, $p = 0.073$), representing a trend toward improved clearance. No device-related serious adverse events have been observed to date.

Conclusions: These early results demonstrate that the IRRAflow active fluid exchange system is a feasible and safe option for IVH management, with trends toward improved ventricular hematoma clearance and reduced catheter duration compared to standard EVD. Ongoing enrollment toward our target of 60 patients in the prospective study will enhance statistical power and clarify the clinical impact of these findings.

Session 3, Abstract 3

LONGITUDINAL DIFFERENTIAL TRACTOGRPHY AFTER ICH

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Session 3, Abstract 4

MINIMALLY INVASIVE EVACUATION OF INTRACRANIAL HEMORRHAGES USING THE AURORA SURGISCOPE SYSTEM: PRELIMINARY EXPERIENCE

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Background: Minimally invasive neuroendoscopic techniques for intracranial hemorrhage (ICH) have advanced, emphasizing reduced tissue damage, effective clot removal, and smaller incisions. We assessed the clinical utility of the Aurora® Surgiscope System (Integra LifeSciences, Princeton, NJ) by analyzing volumetric reduction and outcomes associated with ICH clot evacuation.

Methods: We retrospectively reviewed the records of patients undergoing ICH evacuation with the Surgiscope between July 1, 2019 and August 31, 2024. Demographics, comorbidities, symptoms, procedural details, and outcomes data were analyzed. Hemorrhage volumes were quantified pre- and post-evacuation using 3D Slicer software. Volumes and outcomes in lobar (superficial) versus basal ganglia or thalami (deep-seated) ICH were statistically evaluated.

Results: Thirty-one patients (median age:68 years [interquartile range [IQR]:56.5-75]; 54.8% women) harboring 17 superficial and 14 deep-seated ICH were included. Using the Surgiscope, technical success was achieved in all cases, reducing median hemorrhage volume from 33.5mL (IQR:25.2-67.4) to 6.7mL (IQR:3.5-17.7), an 80.9% reduction (IQR:54.4-89.9). The evacuation success rate was 93.5%, with 2 patients requiring reoperation. Superficial ICH demonstrated delayed Glasgow Coma Scale (GCS) score improvement after day 7 postprocedure (p=0.01), whereas deep-seated ICH showed GCS score improvement as early as 48 hours (p=0.04). Compared to patients with deep-seated ICH, patients with superficial ICH exhibited a dichotomous clinical course, experiencing neurological decline leading to a higher in-hospital death rate (47.1% vs. 7.1%, p=0.02) or excellent GCS scores among survivors at discharge (15 [15–15] vs. 14 [13.8-15], p=0.03). Post-evacuation ICH volume remained the sole predictor of in-hospital mortality (adjusted odds ratio:1.135 [95% confidence interval:1.016-1.268], p=0.03) on multivariable regression.

Conclusion: The Surgiscope showed promise as a safe, effective tool for minimally invasive ICH evacuation, with early neurologic improvement in deep-seated hemorrhages, and substantial volumetric reduction and a high evacuation success rate in all hemorrhages, providing valuable insights to guide minimally invasive ICH evacuation surgeries.

Session 4, Abstract 1

INTRAARTERIAL ENCEPHALOGRAPHY FROM AN ACUTELY IMPLANTED ANEURYSM EMBOLIZATION DEVICE IN AWAKE HUMANS

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Objective: Endovascular electroencephalography (evEEG) uses the cerebrovascular system to record electrical activity from adjacent neural structures. The safety, feasibility, and efficacy of using the Woven EndoBridge Aneurysm Embolization System (WEB) for evEEG has not been investigated.

Methods: Seventeen participants undergoing awake WEB endovascular treatment of unruptured cerebral aneurysms were included. After WEB deployment and before detachment, its distal deployment wire was connected to an EEG receiver, and participants performed a decision-making task for 10 minutes. WEB and scalp recordings were captured.

Results: All patients underwent successful embolization and evEEG with no complications. Event-related potentials were detected on scalp EEG in 9/17 (53%) patients. Of these 9 patients, a task-related low-gamma (30-70 Hz) response on WEB channels was captured in 8/9 (89%) cases. In these 8 patients, the WEB was deployed in 2 middle cerebral arteries, 3 anterior communicating arteries, the terminal internal carotid artery, and 2 basilar tip aneurysms. Electrocardiogram artifact on WEB channels was present in 12/17 cases.

Conclusions: The WEB implanted within cerebral aneurysms of awake patients is capable of capturing task-specific brain electrical activities. Future studies are warranted to establish the efficacy of and support for evEEG as a tool for brain recording, brain stimulation, and brain-machine interface applications.

Session 4, Abstract 2

LATE-ONSET VASOSPASM IN ANEURYSMAL SUBARACHNOID HEMORRHAGE

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Background: Current risk prediction scores are limited in their ability to accurately predict the onset of cerebral vasospasm. Significant variability in vasospasm onset exists, as a subset of patients will experience delayed-onset vasospasm (≥10 days post-rupture) for unclear reasons. This study aims to determine the clinical and radiologic predictors of delayed-onset vasospasm in aneurysmal subarachnoid hemorrhage (aSAH).

Methods: Patients with vasospasm detected on transcranial doppler ultrasound (mean flow velocity >120 cm/s) or CT angiogram (using neuroradiology interpretation) were selected from a prospectively maintained database of consecutive aSAH patients admitted to a tertiary care center (October 2008 to May 2024). Clinical and neuroimaging variables were compared in univariate tests between patients with and without delayed-onset vasospasm. Variables with significant differences ($p < 0.1$) were included in a multivariable regression model to determine predictors of delayed-onset vasospasm.

Results: Among 435 aSAH patients, 194 (45%) experienced vasospasm (mean age 54 ± 9, 62% female), of which 29 (15%) developed delayed-onset vasospasm. Compared to those without delayed-onset vasospasm, patients with delayed-onset vasospasm were older (58 vs. 53 years, $p < 0.01$), had a higher frequency of posterior circulation aneurysms (31% vs. 18%, $p = 0.03$) and fusiform/dissecting aneurysms (14% vs. 7%, $p = 0.04$), and were more likely to undergo late (> 24 hours post-rupture) aneurysm treatment (10% vs. 7%, $p = 0.05$) and external ventricular drain (EVD) weaning within 24 hours of vasospasm onset (31% vs. 12%, $p = 0.06$). In the multivariable regression, treatment delay (adjusted odds ratio [aOR] 1.03, 95% confidence interval [CI] 1.00–1.07, $p = 0.03$) and EVD weaning (aOR 1.22, 95% CI 1.06–1.41, $p = 0.01$) were associated with delayed-onset vasospasm.

Conclusions: Delayed aneurysm securement and EVD weaning were associated with the development of vasospasm beyond the typical risk window, underscoring the importance of early aneurysm treatment and cautious EVD weaning.

Session 4, Abstract 3

THROMBOINFLAMMATION CAUSES DELAYED CEREBRAL ISCHEMIA AFTER SAH

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Delayed cerebral ischemia (DCI) is the highest cause of morbidity in subarachnoid hemorrhage (SAH) patients surviving aneurysm rupture. DCI has multiple contributing factors including vasospasm, inflammation, and microthrombi, and more recently, microvasculature occlusion has been suggested as a cause of DCI. Recent work implicates platelets and neutrophils in the pathogenesis of DCI in rodents, and both are considered therapeutic targets. However, anti-platelet drugs have not been successful in clinical trials and drugs targeting neutrophils have not been tested in patients. We hypothesize that thromboinflammation, the interaction between platelets and neutrophils, can result in microvascular occlusion. Utilizing mice, SAH was induced, and various interventions targeting platelets neutrophils were evaluated. Mice were examined for neurobehavior deficits daily up to 7 days. SAH causes microvascular occlusion by clots that contain platelets and neutrophil extracellular traps, suggesting thromboinflammation is elevated. The thromboinflammatory cascade was attenuated by depletion of either platelets or neutrophils. Our data suggests that thromboinflammation might be a cause of DCI after SAH in mice.

Session 4, Abstract 4

WIDESPREAD NEUROINFLAMMATION IN THE CEREBRAL CORTEX FOLLOWING SUBARACHNOID HEMORRHAGE

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Background: Early brain injury (EBI) following subarachnoid hemorrhage (SAH) is a critical determinant of poor prognosis. Neuroinflammation is recognized as a key mechanism of EBI, yet the extent and spatial distribution of inflammation in the cerebral cortex remain unclear. We aimed to investigate how SAH-induced inflammation spreads throughout the cortex and contributes to neuronal cell death.

Methods: A mouse model of SAH was created by controlled hematoma injection into the prechiasmatic cistern, allowing us to model both mild and severe SAH. Twenty-four hours post-injury, brain sections were obtained at anterior, intermediate, and posterior levels to assess neuroinflammation using Iba1 immunostaining for microglia, and neuronal cell death via NeuN and TUNEL co-staining. Cortical layers I–VI were analyzed to determine the depth and regional spread of inflammation.

Results: Activated microglia were markedly increased in all cortical layers and regions in SAH mice, with a significant correlation between SAH severity and the extent of microglial infiltration. Neuronal cell death was also observed across all cortical regions, most notably in superficial layers of the anterior cortex. Although the hippocampus showed some increase in microglia, the inflammatory response was milder than in the cortex. The results suggest that neuroinflammation is not limited to areas adjacent to the hemorrhage but extends throughout the entire cerebrum.

Conclusions: SAH induces widespread neuroinflammation across the cerebral cortex, leading to severity-dependent neuronal death. Targeting global inflammation in the acute phase may be crucial for improving outcomes.

Session 4, Abstract 5

ADROPIN-MEDIATED NEUROVASCULAR PROTECTION FOLLOWING SUBARACHNOID HEMORRHAGE

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Background: Acute neural injury resulting from subarachnoid hemorrhage (SAH)-associated cerebral infarction frequently leads to long-term disability, including significant cognitive deficits. Effective treatment options, however, remain limited. The secreted peptide adropin, encoded by the energy homeostasis-associated (Enho) gene, is highly expressed in the brain and liver, and has neuroprotective effects in brain injury models. Adropin is a regulator of lipid metabolism and modulates endothelial cell function, including the maintenance of blood-brain barrier integrity via an endothelial nitric oxide synthase (eNOS)-dependent mechanism. We believe that the peptide hormone adropin is a promising therapeutic target for post-SAH cerebral infarction. Using our murine SAH model, we investigated whether synthetic adropin treatment could reverse the deleterious effects post-SAH.

Methods: SAH was induced in wild-type (C57BL/6) or Enho–/– mice by stereotactic injection of heterologous blood into the subarachnoid space. Mice were randomized to receive intraperitoneal injections of either synthetic adropin or a control vehicle at 6 or 12 hours post-SAH, followed by additional doses every 24-48 hours for up to 10 days. Animals were euthanized at 24 hours, 10 days, and 30 days post-SAH to assess outcomes. Immunofluorescent staining, vasospasm assessment, and neurobehavioral testing were performed.

Results: Adropin treatment reduced delayed cerebral vasospasm at 5 days post-SAH. Immunofluorescent staining demonstrated that adropin significantly decreased microthrombus formation and neuronal apoptosis compared to the control. Neurobehavioral tests, including novel object recognition, Y-maze, and open field tests, showed that adropin improved neurocognitive performance compared to the control at 30 days post-SAH.

Conclusions: These findings suggest that adropin may play a key role in mitigating both vascular and neural damage post-SAH, thereby promoting recovery in our murine SAH models. Although further studies are needed, adropin holds promise as a potential therapeutic target for clinical use.

Session 5, Abstract 1

HIGH WALL SHEAR STRESS AND CYCLIC STRETCH SYNERGIZE TO TRIGGER INTRACRANIAL ANEURYSM INDUCTION

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Background: Intracranial aneurysm is formed at the unique morphology, bifurcation site of intracranial arteries, and becomes balloon-like unique shape during the progression of the disease, indicating the crucial role of hemodynamic-loading in its pathogenesis. The experimental finding that only the alternation of hemodynamic-loading in animals could induce intracranial aneurysm has supported this notion.

Methods: Experimental Intracranial aneurysm model of rats were used. Immunohistochemical analyses, histopathological analyses including electron microscopic ones, live-imaging analyses or cell culture experiments loading stress mimicking hemodynamics were done to explore machineries mediating hemodynamic- loading mediated induction of intracranial aneurysms.

Results: High wall shear stress loaded on endothelial cells activated NF-kB that functions as a major transcription factor regulating intracranial aneurysm induction via mediating inflammatory responses. Because the genetic deletion of P2rx4 in rats which converts the strengthen of shear stress into the concentration of Ca influx in endothelial cells could suppress the incidence of intracranial aneurysms but only a half, not only confirming the role of high walls shear stress in the induction of the diseases but also the presence of another hemodynamic factor contributing to. Here, live-imaging analyses detected the cyclic stretch at the prospective site of the intracranial aneurysm formation in response to heat beats and activated fibroblasts could be observed there. Because, in in vitro studies, stretched fibroblasts could recruited more macrophages, cyclic stretch by heart beats might function as another hemodynamic force to facilitate the formation of intracranial aneurysm via recruiting macrophages synergistically with endothelial cells activated under high wall shear loading.

Conclusions: High wall shear stress and cyclic stretch synergize to recruit macrophages at the prospective site of intracranial aneurysm formation and trigger the disease induction.

Session 5, Abstract 2

PROTECT FINNS: AN ONGOING STUDY TO DEVELOP PERSONALIZED MEDICINE FOR PERSONS WITH INTRACRANIAL ANEURYSMS

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Background: Personalized decision making and treatment is very much needed by patients with unruptured intracranial aneurysms. To achieve this, high quality patient-specific follow-up data is needed from a large number of patients.

Methods: Protect Finns is an ongoing prospective follow-up study of patients with unruptured aneurysms in Finland. At baseline, blood & microbiome samples are taken following with the participating patients are followed with clinical controls every 6 months to collect data of risk factor management, and with yearly control MRAs.

Results: Less than 10% of the aneurysms being followed have grown during the first 2 years of study, and no ruptures have occurred. There is high variation in the composition of the microbiome of the aneurysm patients, as well as in their living habits and blood pressure. Somewhat surprisingly, spontaneous occlusion of an aneurysm has been observed.

Conclusions: Conservative management of unruptured aneurysms deemed to have a low risk of rupture by expert clinicians, seems very safe at least in short-term, provided that the aneurysms are followed with repeated imaging. Large prospectively collected datasets are needed to determine how microbiome, living habits, and blood pressure affect the clinical course of unruptured aneurysms, which may even heal spontaneously.

Session 5, Abstract 3

THE ROLE OF VASA VASORUM IN CEREBRAL ARTERIES AND ANEURYSMS- FRIEND AND/OR FOE?

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Vasa vasorum, or "vessels of the vessels," are essential for supplementing the physiological transport of nutrients from the blood-filled lumen into the arterial wall. These vessels are particularly important in areas of the vasculature with increased metabolic demands and longer diffusions distances such as coronary vessels and the aorta. Intracranial arteries are generally thinner and situated in the subarachnoid space, where their abluminal surface is bathed in cerebrospinal fluid, providing an additional pathway for nutrient delivery and waste removal. In cerebral arteries, the vasa vasorum are thought to develop in adulthood, largely in pathological situations such as in regions of atherosclerotic thickening. Similarly, the presence of vasa vasorum in the IA wall has generally been viewed negatively due to an association with inflammation. Moreover, the vasa vasorum of IAs has been implicated in continued growth of large and giant aneurysms after apparently successful endovascular treatment - ultimately necessitating surgical intervention.

In this study, we addressed the role of the vasa vasorum in both cerebral arteries and aneurysms using combined scanning immunofluorescent multiphoton imaging (SI-MPM) and high-resolution micro-computed tomography (μCT). We contrasted the vasa vasorum architecture and location, identifying, for the first time, an extensive vasa vasorum plexus in the IA wall, in contrast to the distinct vessels in cerebral arteries. In IAs, our quantitative analyses show significant colocalization of vasa vasorum with calcifications, a strong correlation with increased wall thickness (R^2=0.96, $P<0.001$), and associations with well-defined biaxially oriented collagen fibers. Contrary to the prevailing view of vasa vasorum as solely adverse, these findings highlight an additional protective remodeling role, both of which may critically impact IA rupture risk. This study underscores the contribution to IA wall stability and the need to elucidate their functional impact on IA pathophysiology. Hence, these micro-vessels warrant further investigation for therapeutic targeting.

Session 5, Abstract 4

CFD-BASED ASSESSMENT OF HEMODYNAMICS IN INTRACRANIAL ANEURYSMS FOLLOWING THE VIRTUAL DEPLOYMENT OF THE CONTOUR NEUROVASCULAR SYSTEM

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Background: The Contour Neurovascular System (Contour) is an emerging endovascular device designed to treat wide-necked bifurcation aneurysms (WNBAs). The precise hemodynamic impact of this intervention remains poorly understood. This study aims to provide detailed in vitro and in vivo analyses of blood flow alterations following Contour deployment, using advanced imaging and simulation techniques.

Methods: Fourteen patient-specific basilar tip aneurysm models, with and without Contour devices (sizes 5–14 mm), were analyzed via 4D flow MRI and image-based numerical simulations. A sophisticated virtual pipeline replicated the experimental positioning and deformation of the Contour. Additionally, 28 patient-specific simulations were conducted using medical imaging from eight patients across pre- and post-intervention, and first and second follow-up time points.

Results: In vitro simulations revealed that the Contour significantly reduced intra-aneurysmal flow velocity by 67% ($p = 0.002$) and wall shear stress (WSS) by over 87% ($p = 0.002$). Flow reduction was also confirmed via decreased neck inflow rate, kinetic energy, and inflow concentration index. Device size had a stronger influence on intra-aneurysmal flow reduction than positioning, though positioning affected posterior cerebral artery (PCA) flow. In vivo analyses supported these findings: mean time-averaged velocity decreased from 0.10 m/s pre-intervention to 0.04 m/s post-intervention, and mean WSS decreased from 0.57 Pa to 0.16 Pa. Longitudinal follow-up confirmed sustained reductions. Moreover, insufficient ostium coverage correlated with higher flow metrics over time, indicating its importance in long-term treatment efficacy.

Conclusions: In conclusion, the Contour device significantly reduces intra-aneurysmal flow and wall shear stress, supporting its efficacy in treating WNBAs. Proper device sizing and positioning—particularly to ensure ostium coverage—are crucial for maximizing both immediate and long-term treatment outcomes. Further studies are needed to assess flow changes in PCAs following Contour deployment.

Session 5, Abstract 5

INVESTIGATION OF VASA VASORUM AND HYPOXIA IN HUMAN INTRACRANIAL ANEURYSMS

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Vasa vasorum (VV) are microvascular networks nourishing the outer layers of large extracerebral arteries, commonly present in thicker vessel walls. In contrast, cerebral arteries typically lack significant VV, presumably due to their thinner vessel walls, receiving nutrients primarily via luminal diffusion. A recent study has suggested that VV proliferation in intracranial aneurysm (IA) walls may be linked to localized hypoxic conditions, indicated by hypoxia-inducible factor-1 alpha (HIF-1α) expression. Understanding the development and function of VV in IA could elucidate mechanisms underlying IA enlargement and its impact on wall thickness, thus aiding in the assessment of aneurysm wall vulnerability.

Three unruptured human IAs from hypertensive patients with an average age of 68 will be examined. Specimens will be fixed in 4% paraformaldehyde and whole-stained for endothelial cells (CD31), calcification (Osteosense 680), and hypoxia (HIF-1α). Each sample will undergo micro-computed tomography scanning at 3 μm resolution and be three-dimensionally reconstructed using IMARIS software. Wall thickness analysis will be performed using 3-Matic software. Subsequently, the abluminal surfaces of aneurysms will be imaged using scanning multiphoton microscopy, enabling visualization and three-dimensional reconstruction of VV, calcification, and hypoxic areas using IMARIS software. The primary objective of this study is to investigate the correlation between VV density, aneurysm wall thickness, and both general vascular ischemic conditions and those caused by calcific occlusion. By analyzing these variables in human IA samples, we aim to better understand the pathophysiological role of VV within IA walls. Understanding this mechanism may allow for the detection of VV using advanced wall enhancement (AWE) imaging techniques, potentially establishing VV as a biomarker for aneurysm wall vulnerability. The findings could enhance pathological understanding and identify critical factors influencing IA progression.

Session 5, Abstract 6

THE ROLE OF SURVIVIN IN VASCULAR AND CEREBROVASUCALR MECHANBIOLOGY AND PATHOLOGY

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Arterial stiffening contributes to the development and progression of vascular and cerebrovascular diseases such as atherosclerosis and cerebral aneurysms, both of which increase stroke risk. Stiffening alters the biophysical cues sensed by resident vascular smooth muscle cells (VSMCs), driving a phenotypic shift from a contractile to a synthetic state characterized by aberrant migration, proliferation, and extracellular matrix (ECM) production and remodeling, all of which are key processes in pathological plaque development, aneurysm formation, and vascular dysfunction. These phenotypic transitions are strongly regulated by arterial stiffness. While current therapies reduce plaque burden and vascular tone, none specifically target the signaling pathways associated with arterial stiffening. A clearer understanding of how stiffness regulates VSMC migration, proliferation, and ECM remodeling may identify more effective therapeutic targets. Survivin, a protein with pro-proliferative and anti-apoptotic functions, is downregulated in differentiated adult tissues but upregulated in vascular pathologies such as vascular injury, atherosclerosis, and hypertension, both in animal models and in proliferating VSMCs within the neointima and media of human atherosclerotic plaques and stenotic vein grafts. Moreover, we observed elevated survivin mRNA levels in animal models of cerebral aneurysms and stroke, further supporting its clinical relevance in vascular and cerebrovascular diseases. We therefor hypothesize that survivin functions as a molecular lynchpin in regulating the vascular injury response, atherosclerosis, cerebral aneurysms, and stroke. To examine how stiffness influences VSMC behavior in vitro, we used stiffness-tunable polyacrylamide hydrogels that mimic physiological and pathological vascular stiffness and found that increased substrate stiffness enhances VSMC proliferation, migration, and ECM production. These effects were attenuated by survivin inhibition. Together, our findings suggest a novel mechanism by which survivin modulates stiffness-dependent VSMC behavior, potentially reducing or preventing vascular and cerebrovascular disease.

Session 6, Abstract 1

THE SPECIFIC MOLECULAR MECHANISMS REGULATING THE RUPTURE OF INTRACRANIAL ANEURYSM DISTINCT FROM THE INITIATION OR THE GROWTH

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Background: Rupture of intracranial aneurysm can cause devastating subarachnoid hemorrhage, making the precise understanding of mechanisms regulating the rupture being important. Recent experimental findings mainly from animal models have revealed some potential but crucial mechanisms regulating the rupture of the lesions.

Methods: Experimental Intracranial aneurysm model of rats were used. Immunohistochemical analyses, traditional histopathological analyses and ones with tissue transparency, comprehensive analyses or in vitro experiments were done to explore machineries regulating the rupture of intracranial aneurysms.

Results: Animal model of rats in which induced intracranial aneurysm spontaneously ruptures resulting in subarachnoid hemorrhage was used. The histopathological examinations revealed the induction of neovessels, vaso vasorum, exclusively around the site of rupture from surround brain surface. Through studies using dye sensitive to hypoxia in tissue, histopathological examinations, applying ectopic administration of VEGF, hypoxic microenvironment in aneurysm walls might contribute to the induction of neovessels toward the prospective site of rupture via VEGF. Comprehensive gene expression analyses and histopathological analyses identified neutrophils as a specific type of cells present in ruptured lesions, indicating the contribution of this type of cellsto rupture. Indeed, the increase in the number and the activation of neutrophils could induce rupture of the lesions in rats, confirming the crucial role of neutrophils in the rupture. Next, factors to recruit neutrophils into the prospective site of rupture was examined. C5a produced through enzymatic cleavage of complement C5 by Plasmin released from thrombus in lesions was picked up as a promising candidate.

Conclusions: The rupture of intracranial aneurysms might require two machineries, vaso vasorum formation due to inflammation-induced hypoxia in aneurysm walls and the migration of neutrophils recruited by C5a produced by the Plasmin-mediated enzymatic cleavage of C5, distinct from the initiation or the growth of the lesions. In this sense, inflammatory microenvironment dramatically changes when intracranial aneurysm ruptures.

Session 6, Abstract 2

PHARMACOLOGICAL CLEARANCE OF SENESCENT CELLS PREVENTS INTRACRANIAL ANEURYSM RUPTURE IN AGED MICE

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Background: Aging is associated with a risk of intracranial aneurysm rupture. Aging disrupts cellular homeostasis, notably through the accumulation of senescent cells. Among markers of cellular senescence, p16 is widely used due to its elevated expression in senescent cells. Our preliminary data demonstrated p16-positive senescent cell accumulation in human intracranial aneurysms, implicating cellular senescence in aneurysm pathophysiology. Similarly, aged mice exhibited higher aneurysm rupture rates, correlated with senescent cell accumulation. Based on these findings, we investigated whether pharmacological elimination of senescent cells could reduce the rate of aneurysm rupture.

Methods: We used p16-3MR mice (18-month-old males and females), transgenic mice in which the p16 promoter drives truncated herpes simplex virus-1 thymidine kinase and two reporter proteins. The expression of herpes simplex virus-1 thymidine kinase under the control of the p16 promoter enables the selective elimination of senescent cells through ganciclovir treatment. Intracranial aneurysms were induced by combining elastase injection with the induction of hypertension. Following aneurysm induction, mice were treated either with ganciclovir to eliminate senescent cells or with phosphate-buffered saline as a vehicle control. We compared the two groups' aneurysm formation rates, rupture rates, and survival curves.

Results: Pharmacological elimination of senescent cells reduced the rupture rate of intracranial aneurysms compared to vehicle treatment in both male and female mice ($P < 0.05$). There was no significant difference in the rate of aneurysm formation between the groups in either sex. Survival rates were significantly higher in the ganciclovir-treated group compared to the vehicle group in both sexes ($P < 0.05$).

Conclusions: The pharmacological elimination of senescent cells effectively prevented the rupture of intracranial aneurysms. These findings suggest that aging and cellular senescence may represent novel therapeutic targets for preventing aneurysm rupture.

Session 6, Abstract 3

LYMPHATIC VESSELS IN THE INTRACRANIAL ANEURYSM WALL: A NOVEL COMPONENT IN ANEURYSM PATHOBIOLOGY

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Background: Intracranial aneurysm (IA) rupture remains a devastating cause of subarachnoid hemorrhage, with limited treatment options beyond invasive interventions. Although the pathophysiological mechanisms underlying IA formation and rupture are not yet fully understood, chronic inflammation and wall degeneration are recognized as central contributors. The aneurysm wall is known to undergo structural remodeling characterized by loss of smooth muscle cells, extracellular matrix degradation, neovascularization, and infiltration of inflammatory cells such as macrophages and lymphocytes. These changes contribute to wall thinning, increased fragility, and ultimately rupture. However, the role of lymphatic structures in this context has remained understudied.

Methods: This review describes the presence and characteristics of lymphatic vessels in human IA walls, based on immunohistochemical and immunofluorescent stainings using established lymphatic markers (including LYVE-1, podoplanin, and Prox1) and explores their association with inflammatory infiltrates and degenerative wall features.

Results: Recent findings identified lymphatic vessels within the walls of IAs, introducing a novel aspect to IA pathobiology. Their presence positively associated with aneurysm rupture as well as characteristics of chronic inflammation in the IA wall. The identification of lymphatic structures in the IA walls challenges previous assumptions about the absence of lymphatic structures in cerebrovascular pathology and aligns with earlier discoveries of meningeal lymphatics in the brain.

Conclusion: Dysfunctional or inadequate lymphatic-like drainage may contribute to the chronic inflammatory environment seen in aneurysm walls and thus may have a role in the aneurysm instability and rupture risk.

Session 6, Abstract 4

FIBRIN ACCUMULATION AFTER FLOW DIVERSION: ANEURYSM AND JAILED BRANCHES

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Aneurysm occlusion after flow diversion is traditionally thought to occur primarily by intra-saccular thrombus formation. However, new evidence shows that fibrin accumulation across the flow diverter wires crossing the aneurysm neck can be equally or more important (and may occur earlier), not only by further disrupting the flow stream into the aneurysm but also by providing a scaffold for tissue growth across the neck. Similarly, fibrin accumulation across flow diverters jailing small arteries and perforators usually results in substantial occlusion of the jailed branch ostium and although in most cases the arteries remain angiographically patent in some cases it can lead to full blockages. In this talk I will describe these findings in more detail, which have been obtained with a combination of in vitro experiments, animal models, and corresponding computational models recently developed that couple the flow and fibrin dynamics. In particular, these studies show that fibrin production from fibrinogen caused by exposure to high flow shear stress can explain the patterns of fibrin accumulation observed in in-vitro experimental models of idealized aneurysms, as well as the partial occlusion of side branches jailed by flow diverters implanted into superior mesenteric arteries of rabbit models.

Session 6, Abstract 5

ANALYSIS OF SYMPTOMATIC BRAIN ANEURYSM PRESENTATION WITH THREE-DIMENSIONAL ANEURYSM WALL ENHANCEMENT

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Background: Aneurysm wall enhancement (AWE) has been associated with intracranial aneurysms (IAs) instability and symptomatic presentation. This cross-sectional international multi centric study aims to test if three-dimensional quantitative assessment of AWE can enhance existing scoring systems for identifying high-risk IAs.

Methods: Patients with saccular IAs prospectively underwent 3T high-resolution MRI at the University of Iowa, Changhai and Ren Ji hospitals between 2018 and 2023. IAs were classified as symptomatic, defined by ruptured presentation, sentinel headaches, cranial nerve palsy, and neurological symptoms due to mass effect. AWE quantification was performed using three-dimensional aneurysm wall mapping. The performance of the PHASES (Population, Hypertension, Age, Size, Earlier subarachnoid hemorrhage, and Site) score to estimate symptomatic and asymptomatic presentation of IAs was compared with the performance of a comprehensive multivariate logistic regression model selected through all subsets regression from the PHASES variables, sex, smoking, aneurysm morphology and AWE data.

Results: A total of 334 patients with 387 saccular IAs were included, being 73 (19%) symptomatic at presentation. In multivariate analysis, three-dimensional circumferential AWE (3D-CAWE) was independently associated with IA symptomatic presentation (OR 6.1, CI 2.1–19.5, $p<0.001$). The PHASES score achieved an area under the curve (AUC) of 0.64 (CI 0.56-0.71) for classifying IA as symptomatic. A comprehensive model that included age, smoking, size ratio, and 3D-CAWE achieved an AUC of 0.80 (CI 0.74-0.85), outperforming the PHASES score (difference in AUC -0.16, CI -0.23-0.09, $p < 0.001$).

Conclusion: In a large international multi-center cohort of saccular IAs imaged with high-resolution MRI, AWE was significantly associated with symptomatic presentation. The comprehensive model incorporating clinical variables, aneurysm morphological metrics, and 3D-CAWE outperformed the PHASES score estimating symptomatic presentation.

Session 7, Abstract 1

THE ASSOCIATION BETWEEN LOW FRACTIONAL FLOW (FF) AND REVASCULARIZATION EFFICACY IN SYMPTOMATIC INTRACRANIAL ATHEROSCLEROTIC STENOSIS

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Background: The application of fractional flow (FF) in assessing symptomatic intracranial atherosclerotic stenosis (ICAS) has been explored, but its clinical value remains unverified. This study aims to evaluate the utility and prognostic value of FF in guiding management of symptomatic ICAS.

Methods: We retrospectively included 111 symptomatic ICAS patients who were hospitalized between March 2019 and December 2023. FF was measured before and after digital subtraction angiography (DSA) and endovascular procedures using AccuICAS software (AccuICAS V1.0; ArteryFlow Technology, Hangzhou). Patients were divided into four groups based on a preoperative FF threshold of 0.76. Similarly, patients were categorized into four groups based on the degree of stenosis (DS%) (≥70% and <70%) to assess the role of DS% in treatment decisions. We assessed the National Institutes of Health Stroke Scale (NIHSS) and modified Rankin Scale (mRS) scores at discharge and 12 months post-treatment. Statistical analyses included T/U tests and between-group comparisons.

Results: In patients with FF≤0.76, the mRS scores in the surgical group significantly differed from those in the conservative group within one year ($P = 0.034$). However, in patients with FF>0.76, no significant difference was observed between the surgical control group and the delayed control group ($P > 0.05$). For patients categorized by DS%, no significant differences were observed in treatment outcomes between the surgical and conservative groups, regardless of whether DS% was≥70% or <70% ($P > 0.05$).

Conclusions: A preoperative FF threshold of 0.76 holds significant value in guiding revascularization decisions.

Session 7, Abstract 2

ENHANCED SUPER LARGE BORE ASPIRATION CATHETER NAVIGABILITY IN COMPLEX NEUROVASCULAR ANATOMY

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Background: Mechanical thrombectomy is the standard treatment for acute ischemic stroke from large vessel occlusion. Super large bore aspiration catheters (SLBAC) improve rates of successful recanalization on the first attempt but their navigation through complex aortic arch and tortuous intracranial anatomies presents challenges due to SLBAC tracking difficulties and the ledge effect, potentially impacting procedural times and outcomes. We investigated the trackability of a newer flexible SLBAC and impact of inner devices on SLBAC navigation including a ledge-reducing delivery catheter (LRDC).

Methods: Two silicone flow models reproduced physiologic temperature and flow. Model one simulated the aortic arch to the internal carotid artery origin, model two replicated tortuous intracranial internal carotid artery to middle cerebral artery. Tested devices were Infinity guide catheter, Zoom88, and Toro88 (Toro Neurovascular). Catheters advanced at one centimeter per second in seven independent trials. Maximum tracking force, inner device displacement, and catheter tip advancement were recorded and analyzed with variance statistics. Methods: Two studies conducted under laboratory conditions utilized physiologically relevant silicone models of 1) the aortic arch to internal carotid artery and 2) tortuous intracranial internal carotid artery. The trackability of a conventional guide catheter (Infinity, Stryker Neurovascular) and two super large bore aspiration catheters such as Zoom88 (Imperative Care), Toro88 (Toro Neurovascular) was assessed using the aorta model. Intracranial navigation of Zoom88 and Toro88 was further evaluated with microguidewire only, microguidewire plus aspiration catheter, and the LRDC Tenzing8 (Route92 Medical).

Results: In the aortic arch model, the newer, flexible SLBAC (Toro88) demonstrated significantly lower track force and greater guiding distance compared to conventional devices. In tortuous intracranial segments, the flexible SLBAC (Toro88) consistently showed significantly lower track forces and greater guiding distances across all techniques. The usage of LRDC markedly reduced track forces for both catheters, particularly in highly tortuous segments, and minimized inner device displacement. Toro88 combined with LRDC achieved optimal navigability, reaching distal segments where Zoom88 failed.

Conclusions: The flexible SLBAC and the usage of LRDC, profoundly improved navigability in challenging vascular anatomies. These findings suggest that combining optimized catheter design with innovative delivery techniques can improve mechanical thrombectomy procedural efficiency and safety.

Session 7, Abstract 3

DEVELOPMENT OF A FIRST COIL PREDICTION MODEL FOR UNRUPTURED CEREBRAL ANEURYSMS USING SURGEON-ORIENTED MORPHOLOGICAL FEATURES AND MACHINE LEARNING

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Background: In coil embolization for unruptured cerebral aneurysms, the selection of the first coil is crucial to prevent postoperative recurrence or incomplete occlusion. Recently, AI-based prediction models have been developed using aneurysm size and volume; however, these models have not sufficiently explored feature designs that consider aneurysm orientation or the neck plane. As a result, they struggle to capture the complex 3D morphology of aneurysms and fail to reflect morphological factors prioritized by clinicians. This study aimed to improve prediction accuracy by introducing shape features based on the neck plane, which surgeons consider critical during coil selection.

Methods: We analyzed 1,005 cases of unruptured cerebral aneurysms treated at Jikei University Hospital. The aneurysm and parent vessel were annotated separately from DSA images to construct STL models. The contact area between the aneurysm and vessel was defined as the neck plane, and the aneurysm height was measured along the normal vector of this plane. The height was divided into 5 to 15 segments, and elliptical fitting was applied to cross-sections parallel to the neck plane. Features such as major/minor axes, rotation angle of each ellipse, and aneurysm height were used as input. The ground truth was defined as the actual size and length of the first coil used in surgery. Prediction models were constructed using seven machine learning algorithms. Accuracy was evaluated within ±1 mm for size and ±5 cm for length. We explored the best combination of segmentation count, feature standardization, and algorithm.

Results: For size prediction, the SVR model with 11 slices achieved the highest accuracy (90.0%). For length prediction, the SVR model with 6 slices and standardized features achieved 90.6%, both outperforming conventional models.

Conclusion: Using neck-based elliptical features enables high-accuracy prediction of first coil selection. Surgeon-informed feature design may enhance explainability and support broader device selection.

Session 7, Abstract 4

INTERNATIONAL STUDY OF INTRACRANIAL ANEURYSM EMBOLIZATION USING THE WOVEN ENDOBRIDGE (WEB) DEVICE: SYNOPSIS OF THE WORLDWIDEWEB CONSORTIUM

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Background: Pooled data from several registries have shown satisfactory efficacy of the WEB for the treatment of wide-neck bifurcation aneurysms. The present study is the real-world largest collaboration that endeavors to investigate the angiographic and clinical outcomes of intracranial aneurysms treated with the device.

Methods: The WorldWideWEB consortium is a retrospective multicenter collaboration of international centers with no limitation on aneurysm location or rupture status. Angiographic and clinical outcomes, as well as complications were assessed among all consecutive patients along with determinants of adequate aneurysm occlusion (i.e. complete occlusion or neck remnant).

Results: The cumulative population comprised 674 patients (median age 61.3 years; 71.3% female) with 686 intracranial aneurysms. MCA bifurcation (30.2%) was the most common location. Adequate occlusion was observed in 85.7% of aneurysms at last follow-up. Aneurysm retreatment was required in 7.9% of aneurysms. Thromboembolic complications were encountered in 7.6% of patients but only 2.0% had permanent neurological deficits, while 3.1% had hemorrhagic complications. We identified subarachnoid hemorrhage, immediate remnant aneurysm (as compared to complete occlusion), and minor compaction or major compaction (as compared to no compaction) as significant determinants for lower rates of adequate aneurysm occlusion on last follow up ($p<0.01$).

Conclusion: This report suggests an adequate efficacy and safety profile of the WEB comparable with the currently available endovascular techniques and devices designed for bifurcation intracranial aneurysms.

Session 7, Abstract 5

AN UPDATE ON ROBOTICS IN NEUROIR: ARE WE READY FOR REMOTE

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Robotic technologies are rapidly reshaping the landscape of neurointerventional radiology (NeuroIR), offering the promise of enhanced precision, radiation protection, and improved safety. As platforms evolve, the prospect of remote neurointerventions—once considered aspirational—is now entering real technical and strategic discussions.

We will provide a review of the current landscape of robotic systems in NeuroIR, highlighting key challenges to adoption and presenting original research from our lab focused on the technical feasibility of remote procedures. Specifically, we will share results from simulated remote robotic testing, analyzing connectivity parameters such as latency, packet loss, jitter, and bandwidth across a range of network conditions.

We will also critically evaluate the broader readiness of these technologies for remote operation, including infrastructure and system requirements, safety considerations, and site selection.

Finally, we explore the implications of remote robotic interventions for global neurovascular care delivery, rural access, and the future of procedural training. As the field advances, the central question shifts from feasibility to ecosystem readiness for remote robotic NeuroIR.

Session 8, Abstract 1

QUANTIFYING THE HARMFUL OSCILLATORY SHEAR INDEX THRESHOLD FOR ENDOTHELIAL CELLS: A PRELIMINARY ASSESSMENT

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Background: Stent deployment is the frontline therapy to reestablish narrowed arteries, yet the flow disturbance and pattern change induced by stent deployment can lead to endothelial dysfunction, and subsequently restenosis. Hemodynamic metrics such as Time-Averaged Wall Shear Stress (TAWSS) and Oscillatory Shear Index (OS)—which captures temporal directional reversals of shear— have been used to assess this risk. However, while the former has a well-defined threshold, the latter still lacks a validated biological limit. Studies define them at arbitrary numbers, most commonly used OSI 0.1. This study intends to provide early biological validation of what OSI values affect cells harmfully. This would narrow the critical range for clinical device assessment.

Methods: A self-made two-step flow chamber was created to simulate the strut gaps, and CFD simulation was conducted to optimize the design so that OSI can be varied without significantly changing the TAWSS, thus isolating OSI effect from TAWWSS. Three experimental configurations with differing OSI values below the commonly used OSI threshold 0.01 (0, 0.02, and 0.08) was created. Human carotid artery endothelial cells (passages 5–9) were cultured on gelatin-coated dishes and exposed for 24 h under pulsatile flow (0.4 s period) generated by a roller-pump system with an inline dampener, reservoir, and flow chamber. Post-exposure, cell density and F-actin morphology were quantified by laser scanning microscopy.

Results: The result shows that cell shrinkage were negligible under OSI 0 but soon become apparent (OSI 0.02) and becomes more distinct at OSI 0.08.

Conclusions: This shrinkage is suspected to be signs of early apoptosis, which implicates that even at values below the current accepted threshold oscillating flow are still capable of inducing pro-atherogenic behavior, and that the current threshold needs to be reassessed in future study.

Session 8, Abstract 2

CEREBRAL FLOW DYNAMICS ASSESSMENT WITH 4D FLOW MRI

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Background: Local flow dynamics play a key role in driving vascular adaptations and influencing neurovascular health and disease. 4D flow MRI enables in vivo assessment of cerebral blood flow and cerebrospinal fluid (CSF) dynamics, providing quantitative metrics to inform the diagnosis and treatment of various neurovascular pathologies. However, the accuracy of 4D flow measurements can be compromised by the broad range of velocities, limited spatial and temporal resolution, and partial volume effects. These limitations are especially significant in small cerebral vessels and CSF passages, reducing the reliability of clinically relevant flow biomarkers.

Methods: We developed a framework for estimating the local error in 4D flow MRI measurements based on statistical analysis and principles of flow physics. The uncertainty in MRI-measured velocity at each voxel is estimated using local spurious flow divergence and the error correlations across velocity components inferred from velocity in background voxels.

Results: The error analysis framework was validated using synthetic 4D flow datasets generated from high-resolution CFD simulations and applied to both in vitro and in vivo 4D flow MRI measurements in cerebral aneurysms. While 4D flow MRI captured the dominant flow patterns, near-wall errors increased by up to an order of magnitude. These errors were further amplified when computing velocity-derived metrics such as wall shear stress, substantially compromising their reliability. Wall shear stress estimates derived from CFD and 4D flow MRI showed substantial discrepancies, despite overall agreement in global flow patterns across modalities.

Conclusions: Our findings indicate the need for super-resolution methods to increase reliability of velocity-derived metrics in cerebral vasculature and CSF passages. The error analysis of 4D flow MRI measurements of cerebral flow provides framework for quality assessment of the data. This can improve reproducibility of the data and facilitate comparison of 4D flow datasets acquired in longitudinal studies or with different MRI systems.

Session 8, Abstract 3

THE FORGOTTEN CULPRIT, THE ROLE OF HEMODYNAMIC STRESSES IN DRAINING VEINS OF AVMS

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Arteriovenous malformations (AVMs) are typically defined by abnormal connections between arteries and veins, bypassing the capillary network. While the nidus and arterial feeders have traditionally been the focus of AVM evaluation and treatment, the role of draining veins has received comparatively little attention. These veins are chronically exposed to high-flow, high-pressure arterialized blood, which can lead to wall remodeling, venous dilation, and increased risk of rupture. Hemodynamic stresses—including elevated shear stress and turbulent flow—are increasingly recognized as contributors to venous wall weakening and AVM instability.

Emerging imaging techniques have revealed that draining veins are not merely passive conduits but dynamic structures that may influence AVM behavior. Vessel wall enhancement on high-resolution MRI has been identified as a non-invasive marker of venous inflammation, correlating with histopathologic evidence of vessel wall degradation. This inflammatory response may further reduce venous integrity and increase the risk of hemorrhage, even in the absence of large or complex nidus morphology.

Quantitative Magnetic Resonance Angiography (QMRA) adds physiologic context by measuring volumetric flow through arterial and venous segments. When used in combination with vessel wall imaging, QMRA can identify abnormal venous hemodynamics and regions of flow-related stress that may contribute to AVM rupture risk.

Current treatment paradigms often focus solely on the nidus, but a comprehensive assessment that includes draining vein pathology may improve risk stratification and therapeutic decision-making. Recognizing the draining vein as a critical and potentially inflamed component of AVMs could lead to more individualized treatment strategies and better outcomes. Further research is needed to establish imaging biomarkers of venous vulnerability and integrate them into AVM management algorithms.

Session 8, Abstract 4

ROLE OF FLOW STAGNATION AND VASA-VASORUM IN ANEURYSM GROWTH

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Understanding the conditions that promote aneurysm growth and subsequent wall failure and rupture is important for precise and personalized risk assessment but remains a challenging problem.

It is widely accepted that low flow conditions affect the pathophysiology of the aneurysm wall, and flow stagnation has been proposed as a risk factor for aneurysm rupture. In a previous study, we demonstrated that aneurysm flow stagnation can be quantified by exceeding contrast retention in the aneurysm compared to the parent artery during angiography, in both real DSA sequences as well as virtual angiograms created from CFD models. More recently, we found that flow stagnation is associated with aneurysm instability and growth in a pilot study restricted to a single location (PCOM aneurysms). The first part of the talk will describe these findings.

During aneurysm growth, the wall needs to remodel to be able to withstand increased stresses due to its enlargement or it will rupture. This typically leads to wall thickening, especially in regions of low or stagnant flows. However, as the wall thickens, hypoxia conditions may be created towards the outer layers, which may prevent further remodeling. On the other hand, development of vasa vasorum in these regions can complement the oxygen supply from the lumen and meet the metabolic oxygen consumption demands of the wall. The second part of the talk will present a recent computational model of oxygen transport in the walls of an aneurysm that developed a vasa vasorum network on its external layer. A realistic model was constructed from micro-CT images of a resected tissue sample, and high-resolution multi-photon microscopy segmentations of the vasa vasorum network. These results support the idea that vasa vasorum development may aid in the positive remodeling of the wall allowing the aneurysm to continue its enlargement.

Session 8, Abstract 5

MODELLING POINT-SPREAD FUNCTIONS IN MICROCIRCULATION: A PROOF-OF-CONCEPT STUDY BASED ON FICK'S LAW AND 1D NAVIER–STOKES SIMULATIONS OF ARTERIAL ANEURYSM

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Background: Modelling blood flow in arteries is crucial for understanding hemodynamics and associated physiological processes. In this proof-of-concept study, we employ one-dimensional (1D) Navier–Stokes equations (NSEs) to simulate arterial blood flow dynamics, coupled with Fick's law to characterize blood flow concentration diffusion in the microcirculation. We apply the Lax-Wendroff scheme, a second-order finite difference method, to solve the mass and momentum conservation equations under realistic physiological conditions, including pulsatile heart rates. Our focus is on modelling the point-spread function of blood concentration in both healthy arteries and arteries affected by aneurysms.

Methods: Blood flow is modeled considering convective acceleration, pressure gradients, and constant viscosity in a cylindrical domain. Fick's law is discretized to simulate concentration diffusion from arteries into surrounding tissues. The 1D arterial system is linearized around a steady-state solution to analyze wave propagation and mass transport. Numerical simulations are conducted using MATLAB, with boundary conditions linking pressure and cross-sectional area.

Results: Simulations successfully capture pressure wave propagation and concentration diffusion dynamics under normal and aneurysmal conditions. The results reveal how vascular wall expansion in aneurysms alters wave speed, damping behavior, and concentration profiles. The Lax-Wendroff method demonstrates enhanced numerical accuracy and stability, with reduced artificial diffusion compared to first-order methods.

Conclusions: This study shows the feasibility of combining 1D blood flow modelling with Fick's law diffusion to investigate microcirculatory transport and point-spread functions. The proposed numerical framework provides insights into how aneurysms affect hemodynamic patterns, offering a foundation for future computational studies of pathological vascular conditions.

Session 9, Abstract 1

TOPOLOGICALLY ACCURATE CIRCLE OF WILLIS MODELING FROM TOF-MRA AND CTA DATA

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The Circle of Willis (CoW) represents a critical arterial structure in the cerebrovascular domain, exhibiting significant morphological and topological variations among individuals. Approximately 85% of intracranial aneurysms originate within the arteries of the CoW, and its configuration significantly influences the distribution pathways of emboli in ischemic strokes. Due to its clinical relevance, the precise characterization and accurate reconstruction of the CoW from medical imaging are crucial for both acute interventions and elective surgical planning.

In this presentation, we explore automated methods for extracting the CoW and its constituent vascular segments from diagnostic imaging modalities, specifically TOF-MRA and CTA. We critically analyze the challenges and performance differences encountered during intermodal comparisons of CoW extraction techniques, highlighting the importance of topological accuracy in the reconstructed arterial networks.

Furthermore, we outline our ongoing research involving the application of these methodologies to the Geneva aneurysm dataset. This dataset facilitates the systematic evaluation and validation of automated extraction processes, thereby contributing to the development of robust clinical tools for the detection, monitoring, and intervention planning of cerebrovascular pathologies associated with the CoW.

Session 9, Abstract 2

INTERVENTION PLANNING USING FUNCTIONAL ATLASES, FMRI, TMS, DTI, PLANNING TOOLS AND INTRA-OPERATIVE MIXED REALITY

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Effective surgical planning relies on precise visualization, integration of multimodal data, and structured team coordination. Drawing inspiration from aviation, where flight planning involves atlases, protocols, and crew resource management, this talk explores how similar principles can be applied to neurosurgery—particularly in the treatment of arteriovenous malformations (AVMs).

Population-based functional and structural atlases, such as those from the EBRAINS project, offer a foundation for understanding brain connectivity. These can be enriched with patient-specific data from functional MRI (fMRI), transcranial magnetic stimulation (TMS), and diffusion tensor imaging (DTI), co-registered to high-resolution anatomical datasets (e.g., angioCT, TOF, T1-Gad). Together, they form a digital twin of the patient's brain, enabling preoperative simulation in virtual reality (VR) to define surgical targets, reference landmarks, and optimal approaches.

This environment supports designing surgical trajectories, defining patient/head positioning, and rehearsing the procedure with the team. Intraoperatively, mixed reality assists in aligning navigation systems using skin markers and anatomical landmarks. After sterile draping, navigation accuracy is reverified. Bone and vascular structures serve to correct for intraoperative brain shift. Electrophysiological monitoring and cortical or subcortical mapping further guide safe resection near eloquent areas.

For AVMs, feeder vessels are initially targeted to reduce nidus perfusion, monitored via ICG angiography. A >4-second delay in contrast arrival indicates devascularization, allowing for nidus resection with preservation of functional tissue. Intraoperative imaging confirms resection completeness, minimizing reintervention rates.

Postoperative care includes immediate neurological assessment and long-term clinical and imaging follow-up. Complication and outcome registries support continuous improvement. This integrated, data-driven, and team-based approach enhances safety, precision, and surgical outcomes

Session 9, Abstract 3

The Florida Familial Brain Aneurysm Study

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Background: Unruptured Intracranial Aneurysms (UIAs) affect approximately 3.2% of the population, with a higher incidence in females. These vascular abnormalities carry an annual risk of rupture of about 10 cases per 100,000 individuals, influenced by various factors. Aneurysmal subarachnoid hemorrhage (SAH) accounts for roughly 30,000 cases annually in the US, carrying a severe untreated mortality burden of 66.7%. Nonetheless, advancements in minimally invasive technologies have markedly improved the morbidity and mortality associated with UIA treatment. This study aims to conduct a comprehensive analysis of local, retrospective, and prospective data on the diagnosis and treatment of intracranial aneurysms (IAs) in patients. Additionally, it seeks to establish an effective screening protocol for first-degree relatives of IA patients, utilizing non-contrasted magnetic resonance imaging (MRA). Through subsequent blood sample collection and genetic sequencing, the study seeks to identify potential genetic markers, thereby enhancing future screening and treatment strategies.

Methods: A Cohort Registry was established within a high-volume medical institution, with approval from the hospital ethics committee and comprehensive informed consent from all participants. The registry continuously aggregates retrospective and prospective data from individuals diagnosed and/or treated for IAs. It also includes screening of first-degree relatives of IA patients via MRA, interpreted by two independent qualified physicians. These relatives consented to participate in blood sample collection for genetic marker evaluation.

Results: The study enrolled 1,523 individuals, with females comprising 72.3% of the cohort, with an average age of 53 years. Of these, 585 were clinically diagnosed with IAs, and 938 were first-degree relatives. From the family members enrolled, 90% have one, and 10% have two or more first-degree relatives with IAs. Among the screened relatives, 85.6% underwent MRA, revealing a 11.7% positive detection rate for IAs, with an average size of 2.5 mm. Blood samples were collected from 1,354 subjects for genetic sequencing.

Conclusion: Preliminary findings suggest a higher-than-expected incidence of positive IA screenings among first-degree relatives. Genetic analyses hold promise for elucidating IA occurrence and inheritance patterns. Given the current prevalence of IAs, the relatively low morbidity and mortality rates associated with current treatments, and the unpredictable nature of aneurysm rupture, it may be prudent to consider brain aneurysm screening for first-degree family members of individuals diagnosed with brain aneurysms as a valid measure for stroke prevention.

Session 9, Abstract 4

DISRUPTIVE AI INTERVENTIONS ALONG THE ENTIRE ACUTE STROKE CARE PATHWAY

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Session 9, Abstract 5

A MACHINE LEARNING APPROACH TO PREDICTING DEVICE SIZE IN ENDOVASCULAR TREATMENT OF INTRACRANIAL ANEURYSMS FOR LOGISTICS OPTIMIZATION

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Introduction: Endovascular treatment of intracranial aneurysms requires the preparation of devices such as coils, stents, and Woven EndoBridge (WEB) prior to intervention. However, variations in aneurysm and parent artery anatomy across patients complicate optimal device selection. In Japan, wholesalers manage device logistics, however, appropriate sizing cannot typically be determined preoperatively. As a result, a wide range of devices is delivered per case, leading to inefficiencies due to frequent returns of unused items. This study aimed to develop machine learning models that predict optimal device size from aneurysm morphology, with the goal of improving logistics efficiency.

Methods: We retrospectively analyzed 1,630 unruptured saccular aneurysms in 1,570 patients treated at The Jikei University School of Medicine. Inclusion criteria were: (1) treatment with coils, flow diverter stents (FDS: Pipeline), or WEB; (2) no procedural complications; (3) available procedural records with device size and adjunctive techniques; and (4) complete morphological and patient data. For WEB-treated cases, we included both clinical cases and 112 experimental cases where optimal size was determined using 3D-printed models. Cases were grouped by treatment device, and data split into training and test sets (4:1). Seven machine learning algorithms were used to build device-specific prediction models.

Results: Prediction accuracies within ±1 mm for first, second, and third coils were 83.8%, 84.5%, and 84.5%, respectively. For FDS, including parent vessel morphology yielded 94.7% accuracy within ±0.5 mm. For WEB, width and height predictions within ±0.5 mm reached 52.2% each. These results demonstrate moderate success and area for improvement. In a four-case pilot using the coil model to guide logistics, shipped inventory was reduced by 41.2% on average without impacting procedural success.

Conclusion: Machine learning-based prediction of device size may improve preparation and optimize medical device logistics in aneurysm treatment.

Session 9, Abstract 6

MANAGEMENT MODEL BASED ON THE PATIENT'S INITIAL CONDITION AND THE FACTORS OF AN INCIDENTALLY DISCOVERED SOLITARY SACCULAR ANEURYSM

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Background: The increase in incidental diagnosis of intracranial aneurysms (IAs) and fear associated with such diagnosis opens opportunities optimizing the management by creating tools to monitor the effect of modifiable factors on disease induced disability with the aim reducing the disease impact on society. Preliminary single center observations focusing on patients incidentally diagnosed with asymptomatic solitary saccular IA (ASIS) are reported.

Methods: All consecutive patients diagnosed with IAs between 2007 and 2024 were included in the cohort. Only ASIS cases were included in this study. The effect of patient-related factors (age, gender, family history of IA, hypertension and smoking) and aneurysm-related factors (location and morphological characteristics) was first quantified regarding IA management: observation or intervention, choice of intervention modality (endovascular, microsurgery), Secondly, the impact of these factors was measured on patient's disability (modified Rankin scale) at 1 and 5 years of initial diagnosis. Difference in case's distribution regarding outcomes were assessed between clusters of patients using Wilcoxon and Chi-square statistical tests. The significance threshold was set at $p<0.05$.

Results: The full cohort is populated by 3150 patients. The ASIS cohort includes 554 patients and 64% were observed, 19% treated by microsurgery and 17% treated endovascularly. Outcomes were good for 90% and 92% and disability was present in 9% and 7% of patients, one and five years from diagnosis respectively. 0.5% disease related death was measured. Multiple factor interdependencies were observed and integrated in the disease model.

Conclusions: This study identified a set of relevant factors with strong effects on disease management and a high degree of interaction between factors. A disease model and a tool to interact with the model to visualize options and associated outcomes is proposed. More consecutive data provided by multiple centers is required to refine the model and make it more generalizable and precise.

Session 10, Abstract 1

QUANTITATIVE MRA IN COMPLICATIONS OF ANEURYSMAL SUBARACHNOID HEMORRHAGE

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Background: Complications of aneurysmal subarachnoid hemorrhage, including vasospasm, delayed cerebral ischemia (DCI) and delayed infarct contribute to significant morbidity and mortality. We investigated the correlation between quantitative MRA performed immediately after intervention and subsequent complications.

Methods: We included consecutive patients scanned between 9/1/2016 and 1/14/2022 with either ruptured or unruptured intracranial aneurysms (IA's). Clinical data including sex, age, BMI, smoking history, hypertension and diabetes, hyperlipidemia, baseline CTA/CT features, presence of intracranial arterial calcification, mFisher score, and GCS. Using in-house developed semi-automated software (Vessel Voyager), quantitative MRA features, including total arterial length, number of branches, and tortuosity were extracted. Complication endpoints, including a composite endpoint of DCI/delayed infarct/symptomatic and a secondary endpoint of angiographic vasospasm were determined, with assessment of MRA feature contributors in consideration of co-variates.

Results: 78 patients were included, with 48 having vasospasm and 24 having DCI/infarct/symptomatic vasospasm. the first one is vasospasm vs non vasospasm and second one is DCI/delayed infarct/ AI treatment. Using logistic regression and in the multivariate analysis, total branch number, average tortuosity and age are significantly associated with vasospasm incidence, p=0.005, p=0.001 and p=0.021, respectively. In addition, average tortuosity significantly correlated with DCI/delayed infarct/ AI treatment, p=0.012.

Conclusion: Total branch number and average tortuosity are inversely associated with vasospasm as well as average tortuosity is inversely associated with DCI/delayed infarct/ AI treatment. With further studies those features may serve as predictors and markers for those consequences.

Session 10, Abstract 2

IMAGING-GUIDED PRECISION THROMBECTOMY: PREDICTING OPTIMAL DEVICE CHOICE USING CLOT RADIOMICS AND VASCULAR MORPHOMETRICS FOR ACUTE ISCHEMIC STROKE PATIENTS

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Background: Mechanical thrombectomy (MT) has transformed the treatment of acute ischemic stroke (AIS), yet rates of first-pass effect (FPE) and favorable long-term neurological outcomes remain suboptimal. Increasing evidence suggests that patient-specific anatomical and thrombus-related features—such as vascular tortuosity, clot-vessel angulation, and thrombus composition—may critically influence MT success. Pre-treatment CTA imaging offers a noninvasive opportunity to extract these features, but comprehensive, multivariate modeling to inform device selection is lacking.

Methods: We propose a multicenter retrospective study across the University at Buffalo and UCLA involving over 200 AIS patients treated with MT. Pre-procedural CTA will be analyzed using our validated AI-based pipeline for vessel segmentation [DOI: 10.1117/12.3047390], enabling automated quantification of tortuosity and geometric morphometrics along the access path. Manual thrombus segmentation will support extraction of radiomic features that capture thrombus density, heterogeneity, and morphology, as demonstrated in prior work [DOI: 10.1007/s00234-022-03109-2]. Cases will be stratified by MT technique—stent retriever, aspiration, or combination—and multivariate models will identify imaging-derived feature combinations predictive of device-specific procedural success (e.g., FPE, mTICI ≥2c).

Results: Prior single-center studies from our group provide strong foundational evidence: in a 68-patient cohort, radiomic features predicted MT success with an AUC of 0.83, while tortuosity metrics derived from CTA in a 49-patient cohort yielded an AUC of 0.81 for predicting FPE [DOIs: 10.1007/s00234-022-03109-2, 10.1161/SVIN.122.000646]. However, these studies were preliminary, lacked device-specific stratification, and were underpowered for generalizable inference. Our proposed work will address these limitations through a significantly larger, multi-institutional dataset and expanded modeling scope, facilitating robust external validation.

Conclusions: This aims to establish a precision-imaging framework that integrates vascular morphology and thrombus phenotyping to guide MT device selection. By linking imaging features to device performance across a diverse cohort, this work seeks to enable data-driven, patient-specific MT strategies to improve stroke outcomes.

Session 10, Abstract 3

3D VOLUMETRIC ANALYSIS FOR DETECTING SUBTLE GROWTH IN UNRUPTURED INTRACRANIAL ANEURYSMS

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Background: 3D Volumetric Analysis for Detecting Subtle Growth in Unruptured Intracranial Aneurysms Monitoring growth of unruptured intracranial aneurysms (UIAs) is critical for determining the need for treatment, but subtle growth may go undetected using conventional two-dimensional measurements. This study evaluates the effectiveness of three-dimensional volumetric analysis in detecting significant aneurysm growth compared to standard size-based assessment.

Methods: Patients with UIAs who underwent contrast-enhanced magnetic resonance angiography (CE-MRA) at baseline and at least one follow-up visit (≥6 months apart) were retrospectively included. For patients with multiple follow-ups, the latest scan was used. Aneurysm size (the greatest dome diameter, mm) was obtained from clinical records. Baseline and follow-up images underwent rigid registration. Aneurysms and an ipsilateral parent artery were segmented at both timepoints. Aneurysm and arterial volumes were computed using a 3D closed surface quantification method. Registration errors were accounted for by comparing artery volumes. Image processing and volume quantification was performed using 3D Slicer. A paired Wilcoxon signed-rank test was used to determine whether volume and size exhibited significantly different patterns of change from baseline. Modified z-scores were calculated to identify aneurysms with significant growth compared to the study population.

Results: Eighty-eight patients (62 female, mean age 65 ± 13 years) with 113 UIAs were included. The mean percentage change between artery segmentations was 3.72% ± 0.56% (mean ± margin of error, 95% CI). Aneurysm volume exhibited significantly greater change from baseline than size ($p < 0.0001$, effect size = 0.474). A total of 10.6% (12/113) aneurysms had a size increase ≥ 1 mm. In contrast, 15.9% (18/113) exhibited significant volumetric growth (z-score > 3), 11 of which were missed by size criteria for growth.

Conclusions: Volumetric analysis detects aneurysm growth more effectively than size-based measurements, identifying cases overlooked using conventional criteria. Approximately 16% of aneurysms demonstrated growth based on volumetric analysis. This approach may improve risk stratification and management of UIAs.

Session 10, Abstract 4

DIFFERENCES IN ANEURYSM WALL ENHANCEMENT ON VESSEL-WALL MRI USING STANDARD AND ADVANCED FLOW SUPPRESSION MODULES

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Background: Aneurysm Wall Enhancement (AWE) on 3D vessel-wall magnetic resonance imaging (VW-MRI) has emerged as an additional marker to assess aneurysm rupture risk; however, adoptability of VW-MRI has been challenging due to presence of pseudo-enhancements that makes interpretability of VW-MRI findings challenging. The key goal of our study was to quantify the differences in AWE using standard and advanced flow suppression modules in the same patient cohort.

Methods: Patient Data: N=28 patients (16 male, mean age 58 + 12 years) with 30 unruptured intracranial aneurysms were recruited at the Changhai Hospital (China). Imaging: All patients underwent examination on a 3.0-T MRI system. Pre-contrast examination was performed using 3D turbo spin echo sequence (SPACE) with 0.6 mm isotropic resolution followed by Gd-contrast bolus and acquisition of contrast-enhanced MRA with 0.7 mm isotropic resolution. Post-contrast VW-MRI was then performed with four flow suppression sequences: SPACE (0.6mm), SPACE-MSDE (0.6mm), SPACE-DANTE (0.6mm) and SPACE(0.9mm). Post-Processing: We used the AneuSpoke tool developed by our group to project wall intensities onto the 3D model of the aneurysm. Wall intensities were normalized to the signal intensities in the ventricles (referrd to as wall enhancement index or WEI) to allow for inter-patient comparison. Sac-averaged WEI > 1.1 was used to identify aneurysms with pathological or pseudo-enhancements.

Results: Mean WEI for SPACE-0.6mm, DANTE, MSDE and SPACE-0.9mm was 1.83 + 0.022, 1.16 + 0.01, 1.48 + 0.014, and 2.16 + 0.025. Using WEI>1.1 as a threshold, SPACE-0.6mm, DANTE, MSDE and SPACE-0.9mm identified 25/30, 13/30, 23/30, and 28/30 aneurysms as unstable.

Conclusion: DANTE demonstrated the highest blood flow suppression with WEI that was on par with ventricular CSF. Increasing spatial resolution from 0.6mm to 0.9mm led to an increase in WEI intensity, highlighting potential increase in pseudo-enhancement. Future studies should confirm these findings in a larger prospective study.

Session 10, Abstract 5

NEXT-GENERATION IMAGING: 1000 FPS HIGH-SPEED X-RAY ANGIOGRAPHY FOR REAL-TIME BLOOD FLOW ASSESSMENT IN ENDOVASCULAR DIAGNOSIS AND TREATMENT OF CEREBRAL ANEURYSMS

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Endovascular diagnosis and treatment of cerebral aneurysms and other vascular diseases typically use c-arm x-ray imaging systems, which operate at frame rates up to 30 fps. While sufficient for device navigation and treatment deployment, these frame rates are inadequate for detailed flow visualization. 1000 fps High-Speed X-ray Angiography (HSAngio) provides physicians with an unprecedented ability to visualize detailed blood flow patterns within a narrow imaging window of 300 ms to 500 ms, corresponding to the systolic or diastolic phase of a single heartbeat (cardiac cycle), while the patient remains on the intervention table. The quantification of flow such as detailed vascular flow velocity maps, that reflect the true hemodynamics of the blood vessel as influenced by the phase of the cardiac cycle, can be achieved with a higher degree of accuracy immediately following the acquisition of HSAngio images. This provides more accurate assessments of vascular disease severity immediately following image acquisition. Furthermore, HSAngio imaging can also be used to evaluate changes in flow patterns during the course of a treatment procedure. This real-time assessment allows physicians to modify the treatment approach, if necessary, ultimately facilitating better outcomes and enhancing patient care.

In this study, we demonstrate the utility and functionality of HSAngio during flow diversion treatment of four in vitro models of internal carotid artery aneurysm and two in vivo rabbit elastase aneurysm models. HSAngio images of iodine contrast agent flow were acquired during a 300 ms imaging window, both before treatment, during flow divertor deployment, and post-treatment. Following image acquisition, detailed flow velocity maps were generated within one second and made available to the interventionist in real time. HSAngio images were acquired at x-ray doses comparable to conventional lower frame rate Digital-Subtraction-Angiography imaging. Importantly, no modifications were needed to the existing x-ray generator technology of clinically used c-arm systems.

Session 10, Abstract 6

REAL-TIME PROGNOSIS FOR ANEURYSM OCCLUSION: CLINICAL TRANSLATION OF THE QAS.AI DECISION-SUPPORT SYSTEM

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Background: Intracranial aneurysms (IAs) affect up to 6% of the population and are a major cause of hemorrhagic stroke. Flow diverters reduce rupture risk by modifying blood flow, but treatment success still relies heavily on subjective interpretation of angiograms. There are no intraoperative tools to predict outcomes or guide adjustments. This leads to high retreatment rates, up to 30% in some cases.

Methods: We developed QAS.AI, a software as a medical device (SaMD) that integrates quantitative angiography (QA) with machine learning to deliver intraoperative prognostic support during aneurysm treatment. The system processes digital subtraction angiography (DSA) in real time, extracting time-density curves and computing hemodynamic surrogates such as mean transit time and time to peak. These are fed into predictive models trained on multicenter datasets to estimate the likelihood of aneurysm occlusion at 6 months. QAS.AI features a user-facing interface embedded in the clinical PACS workflow, allowing visualization of QA maps and risk indicators directly on live imaging. The system is deployed in a HIPAA-compliant AWS environment that supports real-time processing and model inference with minimal latency. This cloud-based architecture enables scalability and integration across multiple clinical sites.

Results: The software achieved high accuracy in segmenting aneurysms (Dice = 0.84) and predicting 6-month occlusion (AUROC ~78%). QA parameters showed strong correlation ($r > 0.85$) with Doppler ultrasound and CT-based assessments in preclinical studies. In real-time use, QAS.AI flags suboptimal flow conditions that may lead to failure—such as poor device wall apposition or insufficient coverage—allowing for intraoperative correction.

Conclusions: QAS.AI is the first intraoperative tool that offers real-time, data-driven prognosis for IA treatment with flow diverters. It has potential to improve patient outcomes, reduce retreatment, and streamline the adoption of AI-based tools in neurointervention.

Session 11, Abstract 1

INSIGHTS INTO VENOUS CONGESTIVE DISORDERS FROM HIGH-FIDELITY CFD

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Background: Recently, we used high-fidelity CFD to demonstrate high-frequency flow instabilities in venous PT cases, but also the potential to predict stenotic pressure drops using a simple Bernoulli-based formula [Sidora+, JNIS 2024 Jul 29]. The Magdeburg/Chicago group also recently reported excellent prediction of pressure drops in four IIH-PT cases using conventional CFD with patient-specific flow rates [Stahl+, JNIS 2025 Feb 8]. They also highlighted discrepancies between CFD based on flat-panel CT vs. MRV. We now use these cases to validate our Bernoulli formula, and also more generally the need for high-fidelity CFD to understand venous pressure losses and PT sounds.

Methods: High-fidelity CFD simulations were performed on the four Magdeburg/Chicago IIH-PT cases derived from flat panel CT. Pressure drops were also inferred for these cases using our simplified Bernoulli formula. Additionally, three of our own PT cases were simulated using ultra-high fidelity CFD and analyzed using vortex swirl and spectrographic analysis techniques.

Results: High-frequency flow instabilities were evident in 3 of the 4 Magdeburg/Chicago cases when run with our high-fidelity CFD. CFD and Bernoulli pressure drops were consistent but substantially overestimated compared to catheter (and Magdeburg CFD) pressure drops. On the other hand, pressure drops from the Bernoulli formula using MRV-derived stenosis areas were substantially underestimated. In our own PT cases, high pressure fluctuations (ie, potential sound sources) were concentrated at the sigmoid sinus and jugular bulb, and associated with vortex shedding caused by impingement, redirection and relaminarization of the stenotic jet around the sigmoid bend.

Conclusions: High-fidelity CFD suggests that high-frequency vortex shedding, rather than true turbulence, may be prevalent in venous congestive disorders, and the likely cause of PT sounds. Inconsistencies among CFD and clinical pressure drops, and the impact of imaging modality, continue to be investigated.

Session 11, Abstract 2

UNDER PRESSURE: UNRAVELING THE CENTRAL VENOUS INFLUENCE IN IDIOPATHIC INTRACRANIAL HYPERTENSION

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Background: While the role of dural venous stenosis in idiopathic intracranial hypertension (IIH) is well-established, the complex interplay between intracranial and extracranial factors contributing to pathological intracranial venous congestion remains poorly understood. This study explores venous sinus pressure profiles in cohorts with elevated CVP (eCVP) and normal CVP (nCVP).

Methods: We retrospectively included adult patients (≥18 years) who underwent invasive diagnostic venography, with or without therapeutic intent, for a suspected or confirmed diagnosis of IIH. Patient venous dimensions and pressures were extracted and dichotomized into eCVP and nCVP for comparison, with additional subgroup analysis in patient elevated transverse-sigmoid/trans-stenosis pressure gradient (eTSPG) and normal TSPG (nTSPG).

Results: We included 98 patients with CVP measurements. Patients with eCVP exhibited higher opening pressures (OP, 32.3±11.1 vs. 29.9±9.9, p=0.04; Table-1). Unlike patients with stenosis, who demonstrated elevated venous pressures only upstream of the stenotic segment (Table-2), patients with eCVP displayed elevated venous pressures throughout the entire venous sinuses (Table-3). The eCVP cohort not only exhibited a narrower stenotic segment but also significantly narrower sagittal, transverse, and sigmoid sinuses ($p<0.05$; Table-1), suggesting a more global impact of elevated OP rather than just the transverse-sigmoid junction. Furthermore, patients with eCVP demonstrated a delayed onset of gradient formation and a higher pressure gradient before plateauing compared to nCVP for the same OP (Figure-2), indicating increased compliance of the dural venous walls.

Conclusion: This series highlights the variability in venous sinus dimensions, pressures, and gradient profiles with increasing OP, stratified by eCVP and nCVP. While eCVP initially protects against gradient development, it ultimately results in higher venous pressures, which hinder CSF resorption significantly more than for nCVP, thereby increasing OP to a much greater extent. Patients with eCVP demonstrated more compliant dural venous sinus walls, which could influence the long-term effectiveness of venous sinus stenting.

Session 11, Abstract 3

IN-SILICO INVESTIGATION OF HEMODYNAMIC FLUCTUATIONS IN TRANSVERSE SINUS STENOSIS: PITFALLS IN COMPUTATIONAL MODELING

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Computational Fluid Dynamics (CFD) simulations offer a powerful non-invasive method for comprehensive assessment of blood flow in transverse sinus stenosis. These in-silico investigations might provide valuable insights for the better understanding of the occurring pulsatile tinnitus associated with sinus stenosis.

It is hypothesized that the stenosis may induce flow disturbances in the inherently laminar flow. The flow could exhibit turbulent-like structures in the distal located sigmoid sinus region. These unstable flow patterns may only persist for a short duration during each heart cycle. The noise generated by the disorganized flow is likely directly correlated with pulsatile tinnitus. This repetitive effect is expected to be linked only with the peak flow rates at the highest Reynolds numbers within the pulsatile flow.

Accurate detection of flow instabilities requires high-fidelity numerical computations. Low-order numerical discretization methods may fully suppress flow instabilities due to excessive – non-physical – numerical dissipation. Consequently, they are unsuitable for precise numerical computations if the primary objective is the detection of flow instabilities. Additionally, the selection of the mesh generation strategy may also impact the accuracy of computational results.

The present talk will explore various volume mesh generation methods and systematically employs various numerical discretization strategies to study blood flow in patient-specific transverse sinus stenosis. High-fidelity and low-fidelity computational models will be investigated to detect velocity fluctuations in transverse sinus stenosis. Best practice guidelines will be presented for future investigations of pulsatile tinnitus.

Session 11, Abstract 4

WHO GETS BETTER? PREDICTORS OF SYMPTOM IMPROVEMENT FOLLOWING VENOUS SINUS STENTING IN IDIOPATHIC INTRACRANIAL HYPERTENSION

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Background: Chronically elevated venous sinus pressures in idiopathic intracranial hypertension (IIH) may weaken the dural walls covering these sinuses, increasing their compliance and susceptibility to compression by elevated cerebrospinal fluid (CSF) pressures. This may contribute to distal stent- adjacent restenosis following venous sinus stenting (VSS), explaining the persistent or recurrent IIH symptomatology and elevated CSF pressures in some cohorts. We assessed whether increasing the length of transverse sinus stented (TS-Stented) improves IIHsymptom resolution and durability.

Methods: We retrospectively included adult IIH patients who underwent VSS. Pre-stenting TS length (TS-Prestent) was measured from the lateral superior sagittal sinus (SSS) margin to the end of the stenotic segment. Post-stenting, the unstented segment between the torcula and distal stent tip (Torc-Stent) was subtracted from TS-Prestent to derive the TS-Stented. Bivariate logistic regression was used to identify predictors of symptom improvement (complete/partial±recurrence versus no change/worsening), analyzed independently for headache, visual symptoms, tinnitus, and papilledema.

Results: Longer TS-Stented significantly predicted improvement in headaches (aOR:0.78 [95%CI:0.62-0.99]; p=0.041), visual disturbances (aOR:0.581 [95%CI:0.387-0.873]; p=0.009), and tinnitus (aOR:0.881 [95%CI:0.781-0.993]; p=0.039), and showed a trend toward significance for papilledema (aOR:0.521 [95%CI:0.27-1.005]; p=0.052). A longer TS-Prestent was associated with worse visual (aOR:1.543 [95%CI:1.084-2.195]; p=0.016) and papilledema outcomes (aOR:1.621 [95%CI:1.011-2.598]; p=0.045). Factors reflecting an elevated central venous pressure (eCVP, ≥8mmHg) were associated with no change or worsening symptomatology: (1) eCVP-like-physiology (Internal jugular vein pressures≥10.5mmHg): worse visual outcomes (aOR:40.423 [95%CI:3.636-449.433]; p=0.003); (2) higher Anterior 1/3rd SSS venous pressures: worsening headache (aOR:1.141 [95%CI:1.002-1.300]; p=0.047), visual symptoms (aOR:1.099 [95%CI:1.013-1.192]; p=0.023), and papilledema (aOR:1.099 [95%CI:1.013-1.192]; p=0.023); (3) Higher body mass index predicted worse headache (aOR:1.136 [95%CI:1.014-1.272]; p=0.028) and papilledema (aOR:1.204 [95%CI:1.01-1.435]; p=0.038).

Conclusions: This study identifies venous anatomical and pressure factors affecting IIH outcomes and proposes increased dural compliance as a key mechanism, framing VSS as structural reinforcement against chronic collapse.

Session 11, Abstract 5

ENDOVASCULAR AND SURGICAL TREATMENT OF CSF LEAKS TYPES 1, 2, AND 3

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Background: The incidence of spontaneous intracranial hypotension is estimated to be half that of aneurysmal subarachnoid hemorrhage and yet reports of its diagnosis and treatment lag far behind.

Methods: This was a retrospective cohort study including patients referred for a possible diagnosis of spontaneous intracranial hypotension, based on clinical history of orthostatic headaches and suggestive brain MRI, between May 2021 and May 2025.

Results: During this 4-year period, 34 patients were referred with SIH symptoms and brain imaging to warrant evaluation with digital subtraction myelography. CSF leak was identified in 15/34 (44% of patients), including 2 type 1 leaks from ventral bone spur, 2 type 2 leaks from lateral meningocoele, and 11 type 3 leaks from CSF-venous fistula. All type 3 leaks were treated with transvenous onyx embolization, with symptom improvement in 55% and resolution in 45%. There were 4 patients with a spinal longitudinal extradural CSF collection indicating a type 1 or 2 leak. Two patients with lateral meningocoele (type 2) were treated with surgery and both had complete headache resolution, although one experienced bothersome pseudohernia as a consequence of thoracic nerve root sacrifice. One patient with a discogenic bone spur (type 1 leak) was treated with transdural closure and has improving symptoms and imaging in the early phase of recovery while a second patient experienced symptom relief and MRI improvement after epidural blood patching. In the entire cohort of SIH patients treated with surgery or embolization, pre-operative BERN score was 7.4 and was 1.3 on MRI obtained 3 months after treatment.

Conclusions: Spontaneous intracranial hypotension is a disabling postural headache syndrome that is often mis-diagnosed and incompletely treated. Protocolized noninvasive imaging followed by digital spinal myelography yields a diagnosis in ~50% of cases. Tailoring endovascular and surgical treatment to etiology can yield excellent, durable clinical and radiographic outcomes.

POSTERS

Poster 1

DEVELOPMENT OF A DIRECT HEMODYNAMICS PREDICTION METHOD FROM DSA IMAGE INTENSITY DISTRIBUTIONS USING A DEEP LEARNING NETWORK

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Background: Epidemiological analyses of hemodynamics and disease associations require large patient datasets. Computational fluid dynamics (CFD)-based hemodynamic analysis demands extensive preprocessing and iterative computations, hindering its application to large-scale clinical databases. Recent deep learning (DL) networks learn correlations between vascular geometry and hemodynamics, enabling hemodynamic predictions up to 600 times faster than CFD. However, vessel segmentation, mesh generation, and other preprocessing steps remain necessary, limiting use on large datasets. This study aimed to develop a DL network that predicts hemodynamics directly from medical images without preprocessing and iterative computations.

Methods: Hemodynamics depends on vascular morphology, and the geometry is reconstructed from images. Therefore, correlations between intensity distributions in images and hemodynamics were learned by a DL network. Hemodynamics in internal carotid arteries with aneurysms were obtained using CFD from digital subtraction angiography (DSA) images. Then, DSA images were trimmed to the CFD domain. CFD results were resampled to DSA image resolution. DSA intensity value, xyz-velocity (uvw) and static pressure calculated by CFD were normalized to the range from -1 to 1. Rotational augmentation produced 1,292 training samples from 38 cases. The epoch number for training was set as 1,000. Velocity and pressure fields were predicted for four test cases. Prediction errors were evaluated by normalized mean absolute error (NMAE).

Results: Direct prediction of hemodynamics from the images was achieved by DL correlations between intensity distributions and hemodynamics. The errors were limited to NMAE of 8.32 % (SD 1.11) for u, 7.70 % (SD 1.27) for v, 9.91 % (SD 0.90) for w and 14.38 % (SD 1.51) for pressure.

Conclusions: A DL network was developed to learn correlations between intensity distributions in images and hemodynamics. The network achieved direct prediction of hemodynamics from images without preprocessing. Future work will aim to improve prediction accuracy and applicate to large-scale image databases.

Poster 2

EARLY REDUCTION OF PERIVENTRICULAR ANASTOMOSIS FOLLOWING DIRECT BYPASS SURGERY IN ADULT MOYAMOYA DISEASE

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Background: Periventricular anastomosis (PA) is a fragile collateral frequently associated with hemorrhagic events in moyamoya disease. While previous studies have demonstrated delayed radiological reduction of PAs after bypass surgery, little is known about the timing and predictors of early reduction. This study aimed to determine whether PA reduction occurs within 48 hours postoperatively and to identify factors promoting early change.

Methods: This retrospective study included 38 adult patients with moyamoya disease who underwent superficial temporal artery–middle cerebral artery (STA–MCA) anastomosis between 2018 and 2022. A total of 68 PAs were evaluated using sliding thin-slab maximum intensity projection MR angiography (STS-MIP MRA) obtained preoperatively and on postoperative day 1. PA signal ratios were calculated by comparing vessel and parenchymal signal intensity at matched anatomical locations. Multivariate linear regression was used to identify predictors of early PA signal reduction, and ROC analysis was conducted to evaluate its utility for predicting late-phase PA reduction.

Results: The PA signal ratio significantly decreased within 48 hours post-surgery (mean change −0.16 [95% CI −0.21 to −0.11]), whereas cerebellar arteries showed an increased signal. Multivariate analysis revealed that targeted bypass to the PA territory and greater cross-sectional area of the STA were independently associated with early reduction ($p < 0.01$). ROC analysis showed moderate predictive value of early reduction for late-phase reduction (AUC = 0.78). No significant differences in reduction were observed among PA subtypes.

Conclusions: PA can be radiologically reduced within 48 hours following direct bypass surgery, suggesting a potential early preventive effect against hemorrhage. Targeted bypass and STA caliber may promote this reduction. Early PA reduction could serve as a useful imaging biomarker for predicting long-term vascular remodeling and hemorrhage prevention in moyamoya disease.

Poster 3

CTA-BASED VOXEL-WISE RADIOMICS FOR CHARACTERIZATION OF CAROTID INTRAPLAQUE HEMORRHAGE

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Background: Carotid atherosclerosis is a major contributor to acute ischemic stroke. Emerging evidence identifies plaque composition as the key determinant of vulnerability. Computed tomography angiography (CTA) provides a non-invasive alternative to histology for characterizing components like intraplaque hemorrhage (IPH), lipid-rich necrotic core (LRNC), and calcification. This study aims to validate a radiomics-based method for plaque composition quantification and to detect IPH in a stroke registry.

Methods: Patients with symptomatic carotid stenosis (>70%) underwent pre-operative CTA followed by carotid endarterectomy (CEA). Histological slides were matched to CTA slices and analyzed in QuPath. Radiomics features (RF) were extracted from regions of interest and whole plaques using PyRadiomics. Receiver operating characteristic (ROC) analysis identified discriminative RFs for calcification, LRNC, and IPH. A voxel-wise probabilistic classification model estimated plaque composition and was validated against histology. The model was applied to a retrospective stroke registry to assess plaque composition in CTA.

Results: Six patients (mean age 73.7 ± 8.16 years; 3 male) underwent CEA with matched histology and CTA. Radiomics-histology correlation demonstrated substantial agreement (Cohen's kappa = 0.76). Eight optimal first- and second-order RFs achieved AUCs > 0.85 for identifying components. Median histological composition for IPH, LRNC, and calcification were 1.49% (IQR: 0.86), 25.75% (IQR: 8.59), and 19.22% (IQR: 22.57) respectively. Corresponding radiomics estimates were 1.28% (IQR: 2.77), 28.52% (IQR: 59.91), and 69.57% (IQR: 63.03), respectively. Radiomics-based estimates showed high concordance with histology for IPH and LRNC (paired t-test $p > 0.05$; Bland-Altman analysis: no significant bias). In the retrospective analysis of 173 plaques (96 patients), the model detected IPH in 29% (50/173) overall and in 25% (7/28) of plaques ipsilateral to cryptogenic strokes.

Conclusions: Voxel-wise radiomics offers a non-invasive method for quantifying plaque composition and detecting IPH with high concordance to histology, underscoring its potential to enhance stroke risk stratification beyond stenosis severity.

Poster 4

EMERGING EVIDENCE FOR SNCA'S ROLE IN STROKE

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Ischemic stroke involves a complex pathophysiology with both neuronal and vascular components. SNCA, known for its role in neurodegenerative pathologies like Parkinson's disease, has emerged as a potential contributor to vascular morbidity. Yet, how SNCA is involved in the mechanisms behind stroke development and progression is unclear. Transcriptomic studies have suggested that SNCA may exert neuroprotective effects, as music significantly upregulated SNCA expression in individuals with high-music aptitude and in donors with cognitive disorders. Increasing SNCA expression in glioblastoma multiforme (GBM) was also found to reduce tumor growth, indicating that SNCA expression may be favorable in specific disease settings. To elucidate SNCA's role in cerebrovascular pathologies, we compared changes in gene expression across three disease models, vascular injury and ischemic stroke in mouse, and GBM in human, where we found consistent downregulation of SNCA in pathological conditions. 223 differentially expressed genes (DEGs) uniquely shared between GBM and stroke were related to synapse organization and nervous system development. A greater amount of 927 DEGs were exclusively identified between vascular injury and stroke. 129 DEGs were commonly shared across three datasets. Gene Ontology analysis of these DEGs revealed significant enrichment in cell cycle-related terms. We constructed a protein-protein interaction network and found SNCA to be directly involved in stress response and immune system. Predicted pathways of SNCA, DEGs associated with cell cycling and immune system, and potential intermediate players were created with Ingenuity Pathway Analysis. Cell cycling network predicted SNCA inhibition to upregulate CCNA2 through STAT3 and KLF4. Immune players such as VCAM1 and CD44 may be modulated by SNCA through CAV1 and IL6 in our predicted immune system pathway. These findings suggest that SNCA has the potential to be a global regulator of cellular functions and a potential therapeutic target for cerebrovascular disease and GBM.

Poster 5

DESIGNING MULTIMODAL DEEP LEARNING FRAMEWORKS FOR PREDICTING INTRACRANIAL ANEURYSM OCCLUSION: A FOCUS ON DATA INTEGRATION AND FUSION STRATEGY

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Background: Intracranial aneurysm (IA) treatment outcomes remain difficult to predict due to the complex interplay of patient, morphological, and imaging factors. This study investigates the development of a multimodal machine learning (ML) framework to improve the prediction of aneurysm occlusion following treatment, with an emphasis on data harmonization and fusion strategy.

Methods: We analyzed a retrospective dataset of 478 IA patients, combining angiographic parametric imaging (API) from digital subtraction angiography (DSA), aneurysm morphology (location, size class, parent artery diameter), and clinical profiles (hypertension, diabetes, BMI, smoking, subarachnoid hemorrhage, family history, and anticoagulant regimen). Independent preprocessing pipelines were applied to each data stream, using imputation, scaling, and encoding techniques tailored to the feature type. To address class imbalance, synthetic oversampling (SMOTE) was employed.

Results: We trained deep neural networks using both early and late fusion strategies, comparing their efficacy in capturing inter-modal relationships and preserving interpretability. The networks were designed with stream-specific dense layers, activation-specific regularization, and batch-wise class weighting. Across 20 Monte Carlo cross-validation iterations, early fusion yielded higher predictive accuracy (AUC up to 0.80), while late fusion retained clearer attribution of modality-specific influence on outcomes. Confusion matrices and ROC curves were used to evaluate classification quality, and underperforming folds were filtered to focus on generalizable patterns.

Conclusions: Our results demonstrate the importance of fusion architecture and preprocessing design in maximizing the utility of multimodal clinical data. While still in the developmental phase, this approach shows promise for future integration into intraoperative decision-making tools and supports the broader goal of more personalized and interpretable ML in neurovascular care.

Poster 6

ENGINEERED GLIOMA INVASION MODEL USING CEREBRAL ORGANOID

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Background: Glioblastoma (GBM) is a highly invasive brain tumor with poor prognosis, partly due to its dynamic interaction with the brain microenvironment. During invasion, metabolic trafficking such as mitochondria transfer is observed between GBM and astrocytes. While the effects of GBM invasion on surrounding tissue are extensively studied, the role of reactive astrocytes and other tumor-associated cell types on tumor behavior remains unclear. Traditional two-dimensional models and animal studies have limitations in recapitulating the complexity of human brain microenvironments. Here, we introduce a two-phase bioprinting strategy to model and investigate GBM invasion dynamics on cerebral organoids.

Methods: Human cerebral organoids were generated and matured following an established protocol. A two-phase bioprinting technique was employed: fluorescent transfected GBM cells were printed on the surface of cerebral organoids in designated location. Tumor cells spontaneously aggregated and invade brain organoid from the printed spot. After 50 days of invasion, immunofluorescent staining and spatial transcriptomic analysis were performed to evaluate glioma infiltration, metabolic trafficking, and transcriptomic alterations in tumor microenvironment.

Results: The two-phase bioprinted glioma-cerebral organoid model exhibited pronounced and directional invasion of glioma cells into the surrounding organoid matrix, closely mimicking histopathological features in patient-derived tumor samples. Longitudinal fluorescent imaging from day 0 to day 50 revealed persistent and progressive infiltration. Immunostaining showed marked upregulation of FGFR1, GFAP, TOMM20 and additional reactive markers at the GBM tumor-brain tissue interface, indicating a strong astrocytic border response. Quantitative analysis confirmed significantly greater invasion depths and elevated expression of invasion-related genes, including MMP2 and SOX2, compared to early-stage control. Coalignment of GBM cells and reactive astrocytes was observed, suggesting that mechanical stress may guide astrocyte activation and tumor invasion, potentially creating a localized 'hot zone' of interaction.

Conclusions: Our two-phase bioprinted GBM–cerebral organoid model successfully recapitulates key features of glioma invasion and highlights the spatial interplay between tumor cells and reactive astrocytes, offering a powerful platform to study GBM progression and potential therapeutic targets.

Poster 7

RAPID VOLUMETRIC CONTRAST AND HEMODYNAMIC QUANTIFICATION USING SIMULATED DSA AND AI FOR CEREBRAL ANEURYSMS

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Background: 2D digital subtraction angiography (DSA) is used during the placement of stents and coils for cerebral aneurysm treatment. Clinical DSA lacks detailed quantitative hemodynamic information that could impact treatment decisions and patient outcomes. The objective of this work is to develop a pipeline for acquiring 4D (3D+time) patient hemodynamic information in real time based on simulated 2D DSA images. Blood and contrast media transport was simulated in 76 patient-specific cerebral aneurysm models using computational fluid dynamics (CFD). The simulations were modeled over several cardiac cycles until no contrast media remained. The resulting volumetric contrast media transport and velocity data was exported every 10 ms and used as the ground truth. The data was then projected onto 3 orthogonal planes to mimic clinical angiography. The 67 models were used in the training and validation (58/9 split) of a series of convolutional neural networks (CNNs). 9 models were preserved for unseen testing. The first CNN reconstructs the cerebral vascular structure based on three simultaneous orthogonal angiographic images. The second CNN uses time sequences of three orthogonal simulated angiograms and the vascular structure to reconstruct the volumetric contrast media transport in 4D. The final CNN finds the 4D volumetric velocity distribution based on the 4D contrast media transport. Using three orthogonal DSA sequences, the pipeline reconstructed the contrast media transport in 4D (MSE: 5.27 E-04). This allows for better visualization of the contrast media through the depth of the vessel. The pipeline then quantified the 4D patient hemodynamics and compared it to the ground truth CFD cases (MSE: 0.0013). The velocity quantification was successful in all nine unseen testing cases, with a run time of under 3 seconds per case, demonstrating clinical relevance. Quantified 4D contrast and hemodynamic data provided by this pipeline could help guide treatment decisions.

Poster 8

RAPID VOLUMETRIC CONTRAST AND HEMODYNAMIC QUANTIFICATION USING SIMULATED DSA AND AI FOR CEREBRAL ANEURYSMS

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Background: Multiple factors must be considered to optimally manage intracranial aneurysms (IAs). A unicentric study was performed to identify factors associated with the management and patient outcomes and propose a model.

Methods: Patients diagnosed with IA between January 2007 and Decembre 2023 were included. Patient-related factors (age,sex and family history of IA; modifiable:hypertension and smoking status) and aneurysm-specific factors (size, location, rupture, multiplicity) were studied. Patient disability (modified Rankin Scale(mRS)) at one-year post-diagnosis was the primary outcome. The treatment modality was categorized as: endovascular, micro-neurosurgical or observational. Differences in distributions were tested using Wilcoxon-test; and the statistical significancy level fixed at a p-value of 0.05 and corrected for multiple testing.

Results: 3150 patients were included, 590(19%) died over the study period with 206(6.5%) directly disease-related. The analysis cohort included only patients with a complete data set - 1047 (33% of the cohort). An excellent outcome(mRs≤1) was observed in more than 84.3% of cases, impairment in less than 12% and death in less than 2% of patients(95%CI). Aneurysm rupture was the strongest predictor of outcome with 6.65 time more risk of disability and 22.8 more risk of death as compared to unruptured IA. Large dome size strongly associated with increased rates of disability and death. In contrast, younger age, IA multiplicity, positive family history and lower rupture risk IA location had more favorable outcomes. Aneurysm rupture was the strongest predictor for intervention followed by dome size, aspect-ratio, family history of IA, and multiplicity. Treatment modality choice was mostly driven by IA location, IA aspect-ratio, multiplicity and age. A strong interdependency between factors was observed and restricts the use of standard statistics.

Conclusion: This study identified multiple interdependent factors influencing both treatment decisions and outcomes. These findings underscore the need for further analysis. We propose a Bayesian approach comparing posterior probabilities given specific conditions."

Poster 9

3D VESSEL RECONSTRUCTION FROM LIMITED-VIEW DYNAMIC DSA USING A CONVOLUTIONAL NEURAL NETWORK AND MARKOV CHAIN-INSPIRED FRAMEWORK

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3D reconstruction in Digital Subtraction Angiography (DSA) is challenging due to the dynamic nature of blood flow across temporal frames. Traditional 3D-DSA reconstruction methods require continuous X-ray exposure, resulting in high radiation doses. Effective strategies to reduce exposure without compromising image quality remain limited. This study explores the use of truncated dynamic DSA projections for 3D vascular reconstruction via a convolutional neural network (CNN) combined with a Markov chain framework, aiming to minimize radiation while maintaining structural accuracy.

In this study, a total of 45 cerebrovascular DSA datasets were retrospectively obtained from a clinical imaging archive. 3D reconstructions were generated from 30, 50, 70, and 108 projection angles using the Feldkamp-Davis-Kress algorithm. A framework inspired by Markov chain principles was employed: Reconstructions from lower limited projection views were used as inputs for each dataset, while those from higher angular sampling served as reference targets. A series of modified U-Net architectures were developed, with network depth and complexity adjusted according to the level of input sparsity. Each model was trained on 10,700 axial slices, using an 80:20 train-test split stratified by geometry. Finally, the trained U-Nets were applied to 3 DSA patient datasets to evaluate the models' ability to recover full reconstructions from truncated views. Model performance was assessed using mean squared error (MSE).

The trained U-Net models achieved an average mean squared error (MSE) of approximately $4\times10-4$ across the test datasets, demonstrating high reconstruction fidelity. For each test case, the network was able to generate a full 3D reconstruction from truncated input data in approximately 14 seconds, starting from as few as 30 projection views.

This study demonstrates the potential of machine learning algorithms to enable accurate 3D-DSA reconstruction from fewer projections, significantly reducing radiation exposure and supporting the development of safer imaging technologies in clinical practice.

Poster 10

REGIONAL INFARCT TOPOGRAPHY BEFORE THROMBECTOMY AND RISK OF HEMORRHAGIC TRANSFORMATION RISK AFTER TREATMENT: A MULTICENTER BAYESIAN ANALYSIS

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Background: As more patients with stroke are treated with endovascular thrombectomy (EVT), including those with larger cores, understanding the pathophysiology of hemorrhagic transformation (HT) is increasingly important. Pre-EVT infarct topography may have implications for acute decisions (e.g. stenting) and for post-EVT care (e.g. antithrombotics, blood pressure goals). We sought to identify associations between pre-treatment infarct location and HT after EVT.

Methods: Consecutive large vessel occlusion patients with pre-EVT MRI were identified from two centers (2011-2019). Acute infarcts were extracted through a deep learning-enabled pipeline from DWI and spatially normalized. Brains were parcellated (atlas-defined 94 cortical regions, 14 subcortical nuclei, 20 white matter tracts) and reduced to ten principal lesion patterns using unsupervised dimensionality reduction techniques. Binary HT, defined as ECASS PH1 or PH2, was modeled via Bayesian regression, using lesion patterns as inputs, and controlling for total lesion volume, age, sex, initial NIH Stroke Scale (NIHSS), thrombolysis, good reperfusion (TICI 2b-3), acute stenting, last known well-to-puncture time, and other risk factors.

Results: 567 patients (mean age 69 ±15 years; 45% female) had pre-EVT DWI without significant artifacts that underwent lesion segmentation and registration, with median NIHSS 16 (IQR 11-20) and mean total infarct volume was 22.5 ±36.7mL. Thrombolysis was administered in 51%, good reperfusion was achieved in 83%, and HT occurred in 10% of patients. Lesion locations significantly related to HT involved bilateral caudate, putamen, pallidum, and anterior thalamic radiation; and, right more than left thalamus, corticospinal tract, and inferior fronto-occipital fasciculus (area under the curve: 0.73).

Conclusions: These data from a large, multicenter cohort with precise MRI-defined infarcts underscore the risk of specific brain regions infarcted for HT after EVT. Understanding this pathophysiology can inform not only current clinical practice but also the development of future novel therapeutics to prevent HT as more patients with large cores undergo EVT.

Poster 11

COMPARATIVE EFFECTIVENESS OF STANDALONE MIDDLE MENINGEAL ARTERY EMBOLIZATION VERSUS SURGERY IN NON-SEVERE PATIENTS WITH SUBDURAL HEMATOMAS

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Background: Middle meningeal artery embolization (MMAE) has emerged as an alternative to surgical treatment of non-acute subdural hematomas (NASDH). When used in conjunction with surgery, it has previously been shown to reduce recurrence, but its effectiveness as a standalone therapy remains to be established. We aimed to assess the superiority of standalone embolization in comparison to surgery.

Methods: We conducted a propensity score-matched cohort study on 265 consecutive patients with NASDH who underwent either standalone MMAE (sMMAE) or surgical evacuation at our institution. The primary outcome was reintervention due to hematoma resurgence. Secondary outcomes included length of hospital stay, new neurological symptoms, cardiorespiratory, neurological, and other medical adverse events.

Results: After matching, 85 patients were allocated in each group. The median age was 73 years, and 20% were female. There was no baseline difference in clinical characteristics at presentation. During a median follow-up of 72 days in the sMMAE group and 59 days in the surgical group, the incidence rate of reintervention did not differ between the two treatments (IRR 1.38, 95% CI: 0.64 – 3.06, CI p = .41). Median length of hospitalization was shorter in the sMMAE group (4 days vs. 6 days, p =.003). No differences were observed in new neurological symptoms at 30 days (RR 0.85, 95% CI: 0.38 – 1.89, p = .71), nor risk of cardiorespiratory (RR 0.50, 95% CI: 0.12 – 1.93, p = .327), or neurologic adverse events (RR 0.66, 95% CI: 0.11 – 3.88, p = 0.65). Patients treated with sMMAE had a lower risk of other medical adverse events compared to surgery (1.1% vs. 15.2%, RR = 0.07, 95% CI: 0.01 – 0.57, p = 0.013).

Conclusion: Patients who undergo sMMAE may benefit from a shorter hospitalization and a lower risk of medical adverse events compared to open surgical treatment, without experiencing increased risk of recurrence. Larger studies are warranted to establish the effectiveness of sMMAE in the management of NASDH and to identify the subset of patients that are most likely to benefit from this treatment modality.

Poster 12

SPATIAL SEQUENCING OF THE BASILAR TERMINUS DURING EARLY INTRACRANIAL ANEURYSM FORMATION REVEALS POTENTIAL INSIGHTS INTO MECHANISMS DRIVING INITIATION

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Background: Around 1 in 50 Americans have an intracranial aneurysm (IA), a pathologic outpouching of the cerebral vessels. We use an endogenous rabbit model, bilateral common carotid artery ligation (bCCA), to study how changes in blood flow affects gene expression at the basilar terminus (BT) during the early stages of aneurysm formation.

Methods: For our IA model, we performed a bCCA ligation in New Zealand white rabbits in order to increase blood flow through the basilar artery (BA) to initiate an IA at the BT. 24h after the ligation, the BA and BT were dissected and cut into 10-micron frozen sections that are collected onto Capture Slides for spatial sequencing using the 10X Visium. Cell Ranger was used to quantify the cell counts. Then we used Loupe Browser to identify cells on the histology slide that are either in the BA intima, BA media, BT intima or BT media. Differentially expressed genes (DEGs) were identified by extracting the information from Loupe Browser and performing a modified F-test (p,0.1, FC>1.5).

Results: For this rabbit, we identified 33 DEGs expressed in the media (27 lower, 6 higher) between the BA and BT. There were 3 downregulated DEGs of particular interest; PPP1R14A, MYL9, and ISLR. PPP2R14A is associated with enhanced SMC contraction, MYL9 encodes for the myosin light chain protein which affects SMC function, and ISLR that plays a role in tissue regeneration. In the intima, there were 34 DEGs identified (27 lower, 7 higher) between the BA and BT. There was 2 upregulated DEG of interest in the intima: ARPC1B and PFN1. ARPC1B regulates the cytoskeleton which plays a role in cell motility, and PFN1 regulates actin dynamics.

Conclusions: Spatial sequencing reveals DEGs in the intima and media that may play a key role into better understanding the mechanisms of aneurysm formation.

Poster 13

THE EPIGENETIC LANDSCAPE OF MOYAMOYA DISEASE

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Background: "Background: Moyamoya Disease (MMD) is a rare progressive cerebrovascular illness that causes narrowing of blood vessels. Without early detection and treatment, MMD can cause strokes leading to life-long disabilities or death. Etiology is currently unknown, however; there seems to be a familial component. The goal of this study is to expand the on-going research on the genetic component of MMD.

Methods: We acquired single nucleotide polymorphisms (SNPs) from a genome-wide associated study done by Duan et. al and Jeon et al. Histone peak files for human cell types of endothelial cells, macrophages, neutrophils, fibroblast, T-cells, B- cells, monocytes, natural killer cells and smooth muscle cells were downloaded from Cistrome. SNPs were mapped on linkage disequilibrium (LD) blocks using SniPA. In cell types with enriched histone marks, we used Juice Box to identify topological associated domains (TADs) and the genes within them.

Results: Fibroblast and Neutrophils showed significant enrichments within Moyamoya LD Blocks. Fourteen genes were found including RNF213 and TWIST1. RNF213 is known to play a role in angiogenesis and lipid metabolism while TWIST1 plays a role in downregulating the expression of inflammatory cytokines.

Conclusion: Moyamoya associated SNPs have significant enrichments in Fibroblast and Neutrophil cell types. Based on these results, we suspect genetic risk for MMD to operate through fibroblast and neutrophils potentially influencing the expression genes affecting angiogenesis, inflammation and scaffold proteins.

Poster 14

IMPACT OF DENOISING TECHNIQUES ON DEEP LEARNING-DRIVEN INFARCT CORE ESTIMATION FROM CT PERFUSION

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Background: Accurate delineation of infarct core on CT perfusion (CTP) is critical for acute ischemic stroke management, yet vendor-specific denoising routines may alter perfusion map quality and affect automated segmentation. We evaluated whether common noise-reduction strategies influence a convolutional neural network's ability to predict infarcted tissue from baseline CTP.

Methods: Sixty patients with anterior circulation large-vessel occlusion achieved near-complete reperfusion (mTICI 2c–3) after thrombectomy and had baseline CTP and 48-hour diffusion-weighted MRI. Four preprocessing pipelines were compared: principal component analysis filtering, wavelet thresholding, non-local means smoothing, and no denoising. Each produced cerebral blood flow, cerebral blood volume, and mean transit time maps. Infarct labels were derived by co-registration of diffusion-weighted images to CTP and refined with apparent diffusion coefficient thresholds. A data-augmented U-Net convolutional neural network was trained separately on each map set to segment infarct core. Performance was measured by Dice similarity coefficient and examined across denoising methods and infarct volumes.

Results: Median Dice scores were 0.78 ± 0.05 (PCA), 0.77 ± 0.06 (wavelet), 0.79 ± 0.04 (non-local means), and 0.76 ± 0.07 (no denoising). No pairwise differences reached statistical significance ($p > 0.05$). Dice variability showed no correlation with infarct size or specific hemodynamic parameter, and distributions overlapped substantially across methods.

Conclusions: Segmentation accuracy of a U-Net on CTP-derived maps is unaffected by PCA, wavelet, non-local means, or absence of denoising. The ability of deep learning to compensate for noise suggests that complex preprocessing may be unnecessary, supporting streamlined workflows and robust performance of perfusion-based AI across diverse imaging pipelines.

Poster 15

FUNCTIONAL OUTCOMES IN PATIENTS WITH CEREBRAL AMYLOID ANGIOPATHY-RELATED INTRACEREBRAL HEMORRHAGE AND CORTICAL SUPERFICIAL SIDEROSIS

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Background: The recent update to the Boston criteria for cerebral amyloid angiopathy (CAA) adds two non-hemorrhagic neuroimaging markers to increase the sensitivity of CAA detection: multispot white matter hyperintensity (WMH) pattern and severe centrum semiovale enlarged perivascular spaces (CSO EPVS). While these markers, together with well-established hemorrhagic markers such as lobar cerebral microbleeds (CMBs) and cortical superficial siderosis (cSS), increases the likelihood of CAA in intracerebral hemorrhage (ICH) patients, it is unclear whether they impact clinical outcomes.

Methods: Brain MRIs from a prospective database of consecutive non-traumatic ICH patients admitted to a tertiary care center were used to diagnose CAA and determine the frequency of non-hemorrhagic markers. Predictors of an unfavorable discharge outcome (modified Rankin score ≥ 4) were assessed in univariate and multivariable models.

Results: Between 2003 and 2019, 645 (36%) of 1,791 ICH patients were diagnosed with CAA (mean age 74±11 years, 49% female). Multispot WMH pattern occurred in 19%, and severe CSO EPVS occurred in 24%. In univariate analyses, age, hypertension, diabetes, ischemic stroke history, dementia, admission Glasgow Coma Scale (GCS) scores, intubation, external ventricular drain placement, hematoma evacuation, intraventricular extension, and cSS were associated with an unfavorable outcome (all $p < 0.05$). In the multivariable model subjected to backward elimination, age, hypertension, dementia, GCS score, intubation, intraventricular extension, and cSS (aOR 1.75, 95% CI 1.07–2.88) remained significantly associated with an unfavorable outcome.

Conclusions: Although non-hemorrhagic imaging markers are common in CAA patients with ICH, only cSS is significantly associated with unfavorable clinical outcomes.

Poster 16

THERAPEUTIC POTENTIAL OF SURVIVIN IN STROKE

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Stroke remains the fifth leading cause of death worldwide, yet its underlying mechanisms and treatment options remain limited. Intracranial atherosclerotic disease, characterized by plaque formation in cerebral vessels, is a contributor to ischemic stroke. The stiffness of these plaques, which varies with their composition, may contribute to stroke pathology. Survivin, a pro-proliferative and anti-apoptotic protein, is upregulated in pathological vascular conditions such as atherosclerosis and hypertension, promotes plaque development, and is associated with adverse outcomes in patients with cardiovascular disease. Although studies directly linking survivin to stroke are limited, vascular stiffness has emerged as a significant risk factor. Given that dysregulated vascular smooth muscle cells (VSMCs) are associated with vascular stiffening and survivin upregulation, which are also observed in ischemic stroke, we performed RNA sequencing (RNA-seq) on human VSMCs with survivin knockdown cultured on a stiff substrate that mimics the mechanical properties of atherosclerosis or stroke-associated environments. This analysis revealed that survivin regulates key stiffness-responsive processes, including cell proliferation, migration, extracellular matrix deposition, and immune responses. To assess relevance to stroke, we used Ingenuity Pathway Analysis to compare our dataset with RNA-seq data from stroke-induced mice. Differentially expressed genes (DEGs) in both datasets revealed commonly regulated biological processes including angiogenesis, cell differentiation, and cell-cell adhesion. Moreover, survivin knockdown was predicted to inhibit disruption to blood brain barrier and ischemic stroke, through genes such as APP, STAT3, and PLAT. Pathways upregulated in both stroke and stiffness conditions, including FAK Signaling and Extracellular Matrix Organization, were attenuated by survivin knockdown. We identified 30 common DEGs across the stiffness, survivin knockdown, and stroke datasets, including CDK6, E2F8, and TAGLN and overlapping regulators of cell proliferation like HIF-1a, STAT3, PBK, TTK, PLK1, and BUB1. These findings suggest mechanistic links between survivin, vascular stiffness, and stroke, identifying potential targets for therapeutic strategies.

Poster 17

UNDER PRESSURE: UNRAVELING THE CENTRAL VENOUS INFLUENCE INIDIOPATHIC INTRACRANIAL HYPERTENSION

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Background: While the role of dural venous stenosis in idiopathic intracranial hypertension (IIH) is well-established, the complex interplay between intracranial and extracranial factors contributing to pathological intracranial venous congestion remains poorly understood. This study explores venous sinus pressure profiles in cohorts with elevated CVP (eCVP) and normal CVP (nCVP).

Methods: We retrospectively included adult patients (≥18 years) who underwent invasive diagnostic venography, with or without therapeutic intent, for a suspected or confirmed diagnosis of IIH. Patient venous dimensions and pressures were extracted and dichotomized into eCVP and nCVP for comparison, with additional subgroup analysis in patient elevated transverse-sigmoid/trans- stenosis pressure gradient (eTSPG) and normal TSPG (nTSPG).

Results: We included 98 patients with CVP measurements. Patients with eCVP exhibited higher opening pressures (OP, 32.3±11.1 vs. 29.9±9.9, p=0.04; Table-1). Unlike patients with stenosis, who demonstrated elevated venous pressures only upstream of the stenotic segment, patients with eCVP displayed elevated venous pressures throughout the entire venous sinuses. The eCVP cohort not only exhibited a narrower stenotic segment but also significantly narrower sagittal, transverse, and sigmoid sinuses ($p<0.05$), suggesting a more global impact of elevated OP rather than just the transverse-sigmoid junction. Furthermore, patients with eCVP demonstrated a delayed onset of gradient formation and a higher pressure gradient before plateauing compared to nCVP for the same OP, indicating increased compliance of the dural venous walls.

Conclusion: This series highlights the variability in venous sinus dimensions, pressures, and gradient profiles with increasing OP, stratified by eCVP and nCVP. While eCVP initially protects against gradient development, it ultimately results in higher venous pressures, which hinder CSF resorption significantly more than for nCVP, thereby increasing OP to a much greater extent. Patients with eCVP demonstrated more compliant dural venous sinus walls, which could influence the long-term effectiveness of venous sinus stenting.

Poster 18

SELECT THE RIGHT MICROGUIDEWIRE TO OPTIMIZE NAVIGATION IN TORTUOUS ANATOMY

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Background: Rapid and safe endovascular device delivery is often impeded by vessel tortuosity. Microwires are required to have capabilities such as torque, device delivery support, and the pushability to advance. However, there are wires of several stiffness levels, and it is not well understood how stiffness affects each of these respective performance characteristics. This study investigated the impact of microwire stiffness on these abilities within realistic cerebrovascular models.

Methods: Silicone replicas of the intracranial circulation with physiologic flow reproduced moderate and severe tortuosity. The performance of microguidewires with various stiffness (Synchro Select Soft, Standard and Support, Stryker Neurovascular, Fremont, CA) were tested to evaluate the ability of catheter delivery, stress exerted against vessel walls, and distal tip torquability.

Results: Support stiffness demonstrated superior delivery performance by lowering required pushing force by 40% and reduced kickback length by up to 40% compared with Soft stiffness during microcatheter and aspiration catheter delivery ($p < 0.001$). However, stiffer microguidewire exerted significantly greater force on vessel walls, particularly with distal microcatheter tip location ($P < 0.0001$), which may be risk for the vessel perforation. In addition, Support stiffness showed 3 times larger cumulative torque error and frequent lag whip events relative to softer wires ($p < 0.0001$) in the severely tortuous anatomy, while no torque difference appeared in moderate tortuosity.

Conclusions: Microguidewire stiffness presents a complex trade-off. While stiffer wires improve catheter trackability and stability during navigation, stiff wires increase mechanical stress on vessel walls during the advancement and may lose torquability in highly tortuous anatomy for branch selection. Optimal microguidewire selection requires careful consideration of patient-specific anatomical tortuosity and the primary procedural demand to enhance both the efficacy and safety of neuroendovascular interventions.

Poster 19

DISTINCT PROTEIN PROFILES OF ISCHEMIC STROKE PATIENTS BASED ON FIRST PASS EFFECT AND MRS SCORES

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Background: First pass effect in thrombectomy has been well-associated with improved outcomes in patients with acute ischemic stroke. However, not all patients with successful reperfusion experience good functional outcomes. Investigating inflammatory proteins could provide insights into mechanisms influencing stroke outcomes. We hypothesized chemokine and cytokine profiles may indicate thrombectomy success (FPE) and post-thrombectomy outcome.

Methods: Blood samples were collected from 80 patients with ischemic stroke at the time of thrombectomy admitted to Gates Vascular Institute, Buffalo. Procartaplex assays were used to analyze plasma samples for levels of 32 cytokines and chemokines. The relationships between thrombectomy outcome (MRS scores), FPE and analytes were assessed using non-parametric and parametric testing.

Results: Two out of 32 proteins were differential between patients with varying MRS scores. In patients with MRS scores >2, P-Selectin (CD62P) (p=.01) and CD163 (p=.01) were elevated in levels compared to those with scores <2 and were correlated with worsening scores (p=.02 and p=.05). Three out of the 32 proteins were differential when examining thrombectomy pass counts. IL-1 alpha (p=.04) was decreased and both MMP-9 (p=.001) and NGAL (p= .01) were increased in patients who underwent more than one pass and were correlated to increasing amounts of passes (p=.04, p=.01 and p=.02).

Conclusions: Our study suggests distinct proteins may describe patient outcomes according to different clinical metrics. Both P-selectin, a platelet surface adhesion protein, and CD163 a hemoglobin scavenger receptor, were increased in patients with worse outcomes (MRS >2). NGAL and MMP -9 were significantly elevated in patients with >1 pass. The complex is associated with elevation in hemorrhagic stroke and may indicate worsening cerebral infarction. IL-1α plays a complex role in stroke, acting as both a pro-inflammatory and a potential therapeutic agent in post-stroke recovery. The decreased levels of IL-1α in patients with >1 pass could contribute to worsened functional recovery.

Poster 20

EXPLORING ADVENTITIAL COLLAGEN ENGAGEMENT IN HUMAN CEREBRAL ARTERIES UNDER SUPRAPHYSIOLOGICAL LOADS: A COMBINED BIOMECHANICS AND MICROSCOPY STUDY

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The nonlinear mechanical response of arteries under physiological loading is largely governed by the gradual recruitment of tortuous medial collagen fibers. In contrast, adventitial collagen is believed to primarily provide a protective reserve during supraphysiological loading scenarios, such as those encountered in balloon angioplasty or traumatic brain injuries. In this study, we examined this hypothesis in human cerebral arteries by employing multiphoton microscopy (MPM) to visualize adventitial collagen while concurrently subjecting the arteries to progressive uniaxial tension beyond the elastic limit, up to failure. Middle cerebral arteries were obtained from five patients (two males, three females; mean age: 73.4 ± 7.9 years) and were mechanically tested within 24 hours post-mortem in their fresh state.

Our findings demonstrate that adventitial fibers initially lose their tortuosity, followed by gradual alignment in the loading direction. Statistical analyses further revealed that fibers situated closer to the media begin with a more aligned orientation (i.e., lower angular dispersion) and retain a higher degree of alignment under loading compared to those located near the outer adventitia. We observed that at a critical stretch ratio, $\lambda^* = 1.25 \pm 0.09$, the internal elastic lamina (IEL) fails, resulting in rupture of medial fibers adjacent to the IEL. Beyond this threshold, only a subset of medial fibers continue to contribute to load bearing, with a significant shift of load transferred to the adventitial collagen, which serves to protect the remaining media by preventing additional tearing and helping preserve tissue integrity. However, in this elderly cohort, adventitial engagement occurs too late during the loading process to effectively prevent multiple transverse IEL tears and associated medial fiber rupture. This study highlights the critical role of collagen recruitment properties in determining subfailure damage in arterial tissues. Future work will examine how the coupling between collagen recruitment and damage varies with age and disease.

Poster 21

REFRAMING PRION DISEASE THROUGH MECHANOBIOLOGY: FOCAL ADHESION INSTABILITY AND CEREBRAL MICROVASCULAR DYSFUNCTION

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Prion disease is a rare, universally fatal neurodegenerative disease marked by rapid clinical decline and currently lacks effective treatment. Despite extensive research, its underlying pathobiology remains incompletely understood. Prion disease is driven by a self-perpetuating cascade of protein misfolding, intracellular aggregation, and accumulation, ultimately leading to widespread neuronal dysfunction and cell death. Transcriptomic analysis by Polick et al. (2021) using a zebrafish model demonstrated that knockdown of normal cellular prior protein (PrPc) led to a significant downregulation of genes across several KEGG pathways, particularly those associated with focal adhesion kinase (FAK), actin cytoskeleton regulation, and cell adhesion. Near-normal or elevated levels of PrPc are essential for the propagation of its pathogenic isoform, PrPsc, which is also responsible for driving prion disease. Among the downregulated genes, a significant reduction in p130Cas was observed, underscoring the importance of matrix signaling and focal adhesion pathways in both cellular mechanotransduction and biochemical intracellular environment essential for prion pathogenesis. Proper matrix-mediated adhesion and prion protein expression are critical for communication between neurons, ECM, and the cerebral microvasculature. Disruption of this communication, especially within the cerebral microvasculature, may impair focal adhesions dynamics and intracellular mechanical homeostasis, thereby facilitating disease progression. To explore this link, we cultured mouse embryonic fibroblasts (MEFs) on fibronectin-coated, stiffness-tunable polyacrylamide hydrogels and analyzed prion expression using RNAseq following targeted manipulation of key mechanotransduction proteins. On stiff substrates, FAK and p130cas knockdown reduced prion levels by approximately 6% and 12%, respectively, while mechanosensitive lamellipodin knockdown increased prion expression by 34%. Conversely, overexpression of lamellipodin on soft substrates led to a 66% decrease in prion expression. These findings suggest that ECM stiffness and mechanotransduction pathways, particularly lamellipodin, regulate prion protein expression and may represent novel therapeutic targets in prion disease.

Poster 22

DEEP LEARNING-BASED SEGMENTATION OF BIOLOGIC COMPONENTS FROM MARTIUS SCARLET BLUE-STAINED STROKE CLOT HISTOLOGY

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Ischemic stroke (IS) remains a leading cause of morbidity and mortality, affecting approximately 795,000 people annually in the US, with 87% of these strokes being ischemic. Mechanical thrombectomy, a transformative treatment for IS, also provides an invaluable source of clot tissue for research, enabling insights into clot composition, structure, and potential recurrence risk. Histological analysis, particularly with Martius Scarlet Blue (MSB) staining, is the current standard for analyzing these tissues, allowing for clear differentiation of platelets and fibrin. However, the lack of fully automated analysis tools for MSB-stained slides limits consistency and scalability in clot analysis. In this study, we propose an automated approach to segment clot components on MSB-stained slides using Deeplabv3+ with a ResNet18 backbone. Using a heterogeneous dataset from STRIP AI, containing samples from stroke patients across 11 centers, we trained and evaluated the model on patch-and image-level segmentations, achieving a mean intersection-over-union (IoU) of 0.352±0.140 and 0.450±0.114, respectively, in the validation set. Misclassification trends included RBCs and platelets frequently identified as fibrin (31.4% and 16.2%) and platelets or blue artifacts labeled as background or WBCs. Qualitative analysis corroborated these patterns, revealing misclassifications at RBC-fibrin interfaces and misinterpretations of artifacts as WBCs, often due to staining inconsistencies. These findings emphasize the need for improved stain homogenization and expanded datasets to enhance model performance. This automated method offers a consistent, rapid, and scalable approach for analyzing clot tissue, potentially advancing biological insights and improving IS treatment strategies.

Poster 23

EFFECT OF REGIONAL HEMODYNAMICS TO WALL THICKNESS HETEROGENEITY AND ADVENTITIAL COLLAGEN ARCHITECTURE IN HUMAN INTRACRANIAL ANEURYSMS

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Intracranial aneurysm rupture has been linked to patchy wall-thickness variation, but the hemodynamic forces that generate this heterogeneity and their impact on matrix integrity remain unproven. The investigation focuses on two hypotheses: (i) regional extremes of wall-shear stress (WSS), flow patterns such as impingement and vortex affect three-dimensional patterns of wall-thickness heterogeneity, and (ii) the same regions exhibit altered abluminal collagen architecture. This abluminal focus is motivated by evidence that the adventitial layer harbors the principal load-bearing type I collagen network and offers the clearest fiber resolution in multiphoton imaging (Robertson et al., 2015). Resected aneurysm domes undergo ex-vivo micro-CT scanning (voxel ≈ 3 μm), yielding high-fidelity maps of wall thickness analogous to the approach of Tobe et al. (2024). Each surface mesh is mapped onto the patient-specific computational fluid dynamic (CFD) fields derived from pre-operative angiography, enabling point-wise sampling of time-averaged WSS, impingement magnitude and vortex. Collagen integrity and fiber orientation are quantified en face on the abluminal surface by scanning multiphoton microscopy (SMPM), preserving native geometry. Statistical analysis is used to evaluate the strength and independence of the relationship between blood flow metrics and both local thickness variation and collagen architecture. By integrating micro-CT, CFD and SMPM collagen imaging in a unified framework, this study investigates whether extreme, spatially heterogeneous hemodynamics create focal wall thinning or thickening and concurrent matrix adaptation. These results subsequently improve our understanding of the coupling between altered flow and wall changes, ultimately enhancing our ability to use personalized CFD results to improve rupture risk prediction.

Poster 24

A VERY DELAYED CASE OF TRAUMATIC CEREBRAL VASOSPASM

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Traumatic brain injury (TBI) is known to be a major cause of morbidity and mortality in the United States, resulting from a wide range of traumatic injuries. According to the American College of Surgeons Best Practices Guidelines for the Management of Traumatic Brain Injury, treatment is general targeted towards maintaining normothermia, adequate oxygenation, normocapnia, normotension, normoglycemia, as well as avoiding hyponatremia, anemia, or coagulopathy. These measures are to prevent further secondary injury, after the primary injury has occurred. However, TBI is often associated with another cause of secondary injury, known as traumatic cerebral vasospasm (TCV). The largest available study (299 patients) demonstrated the incidence of TCV in the first 2 weeks following TBI to be as high as 45.2%. TCV typically occurs in association with traumatic subarachnoid hemorrhage and often occurs 2-3 days after TBI, but can occur as late as 6 days post-trauma. TCV can result in delayed cerebral ischemia (DCI), which is a significant cause of further morbidity and mortality in this population. This study explores the unique case of a patient who experienced clinically significant traumatic cerebral vasospasm identified 18 days after the initial trauma. The patient had multiple risk factors for TCV, including diffuse injury (>3 lobes of the brain injured), severe SDH, severe SAH, cerebral contusions, female sex, history of substance use, smoking history, low GCS score on arrival (<8), and high Injury Severity Score. She was identified to have vasospasm of the R MCA with CTA and cerebral angiography, then was subsequently treated with intra-arterial verapamil targeting the bilateral ICAs.