South Asian American Vascular Society

SAAVS

14TH ANNUAL MEETING

THURSDAY, JUNE 11, 2026

BOSTON, MASSACHUSETTS

CONNECTING MINDS.

ADVANCING CARE.

IMPROVING OUTCOMES.

WELCOME LETTER FROM THE PRESIDENT

Dear Colleagues and Friends,

On behalf of the South Asian American Vascular Society (SAAVS), it is my great pleasure to welcome you to the 14th Annual Meeting of SAAVS, taking place on Thursday, June 11, 2026, in Boston, Massachusetts.

Our Annual Meeting continues to serve as an important platform for education, collaboration, mentorship, and innovation within the vascular surgery community. Each year, we are inspired by the growth of our organization and by the dedication of our members to advancing excellence in patient care, research, leadership, and diversity within our specialty.

This year's program has been thoughtfully designed to provide meaningful scientific discussions, engaging educational sessions, and valuable networking opportunities for trainees, early-career surgeons, and established leaders alike. We are excited to gather in the historic city of Boston as we continue strengthening the connections that unite our community and inspire future generations of vascular specialists.

We are honored to welcome our Keynote Speaker, Brajesh K. Lal, MD, a nationally recognized leader in vascular surgery whose contributions to clinical innovation, academic medicine, and leadership have had a tremendous impact on the field.
I would like to extend my sincere gratitude to our Board of Directors, speakers, sponsors, and members whose dedication, hard work, and continued support made this meeting possible.

Most importantly, thank you for your continued support of SAAVS and for your dedication to improving the lives of patients through excellence in vascular care.

We look forward to welcoming you to Boston for what promises to be an outstanding and memorable meeting.

Warm regards,
Neelima Katragunta, MD
President, South Asian American Vascular Society

2025 – 2026
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SPEAKER BIOS

KEYNOTE SPEAKER

BRAJESH K LAL, MD

"The Future of Carotid Interventions After CREST-2"

Dr. Brajesh K. Lal is Professor of Vascular Surgery at the University of Maryland School of Medicine and Professor of Neurology at Mayo Clinic. He also serves as Vice Chair of Surgery and Chief of Vascular Surgery at the Baltimore VA Medical Center. He directs the Center for Vascular Research (www.thecvr.org) and the NIH Vascular Imaging Core Facility (www.thevic.org) at the University of Maryland.

A nationally and internationally recognized leader in carotid and vascular disease, Dr. Lal's research and clinical trials have shaped current practice and standards of care. He was one of the early champions and proud co-founders of the South Asian American Vascular Society.

SWECHHA BHATT, MD

Dr. Swechha Bhatt is a Postdoctoral Research Fellow at Dua's Coagulation Lab at Massachusetts General Hospital. A medical graduate from Nepal, she brings firsthand insight into the critical shortage of vascular surgical care in South Asia, having developed simulation-based training programs and advocated for vascular surgery workforce development in low-resource settings. Her work bridges bench science and global surgical equity, with a career focus on becoming a clinician-scientist in vascular surgery.

Dr. Bhatt is among a new generation of South Asian physician-scientists committed to advancing vascular care both in academic medicine and underserved communities worldwide.

RADHA BANSAL, MBBS

Dr. Radha Bansal is a Postdoctoral Research Fellow in the Division of Vascular and Endovascular Surgery at Massachusetts General Hospital, where she conducts research in Dr. Anahita Dua's Coagulation Laboratory.

Her work uses serial thromboelastography with platelet mapping to study antiplatelet response and clinical outcomes after peripheral revascularization. She is currently investigating how serotonergic antidepressant therapy interacts with antiplatelet treatment in patients with peripheral artery disease—at the intersection of psychiatry, pharmacology, and vascular medicine.

Grounded in surgical anatomy and education, Dr. Bansal is building a career in academic surgery driven by clinically meaningful translational research.

AHSAN ZIL-E-ALI, MD, MPH

Dr. Ahsan Zil-E-Ali is a Research Fellow at Penn State University. Trained as a physician and epidemiologist, his research focuses on clinical outcomes, health disparities, and care delivery in vascular surgery, with particular emphasis on peripheral arterial and aneurysmal disease.

He is a senior contributor and co-lead of the Penn State Vascular Surgery Outcomes Research Team (VSORT), with over 100 peer-reviewed publications and presentations at national and regional conferences.

Dr. Zil-E-Ali mentors medical students and residents in vascular research and is committed to advancing evidence-based care and surgical quality.

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6:45 PM	**REGISTRATION**
7:00 PM – 8:00 PM	**WELCOME RECEPTION**
8:00 PM	**PRESIDENTIAL WELCOME AND INTRODUCTIONS** *Neelima Katragunda, MD; University of Tennessee, Chattanooga*
8:00 PM – 8:15 PM	**STUDENT AND RESIDENT PRESENTATIONS**
8:00 PM	**Temporal Patterns and Predictors of Endoleak after Elective Endovascular Aortic Repair.** Ahsan Zil-E-Ali, Research Fellow, Jacob W. Soucy, Medical Student, Faisal Aziz, Chief of Vascular Surgery, *Penn State University*
8:05 PM	**Serotonergic Antidepressants Increase Temporal Variability of Platelet Inhibition After Peripheral Revascularization.** Radha Bansal, MBBS, Shezan Fouzdar, MBBS, MS, Tiffany R. Bellomo, MD, Swechha Bhatt, MBBS, Adriana A. Rodriguez Alvarez, MD, Aseman Bagheri Sheshdeh, BS, Prajakta Lokhande, MD, Anahita Dua, MD, MS, MBA. *Massachusetts General Hospital*
8:10 PM	**Poor Glycemic Control Drives Fibrinolysis Shutdown in Peripheral Artery Disease** Swechha Bhatt, Postdoctoral Research Fellow; *Massachusetts General Hospital*
8:15 PM – 8:45 PM	**KEYNOTE ADDRESS** **"The future of carotid interventions after CREST-2"** *Brajesh K. Lal, MD; University of Maryland School of Medicine*
8:45 PM	**RESIDENT PRESENTATION COMPETITION AWARDS**
8:50 PM	**INTRODUCTION OF SAAVS NEW PRESIDENT AND EXECUTIVE COUNCIL**
9:00 PM	**BOLLYWOOD DANCING**

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SCIENTIFIC ABSTRACT

South Asian American Vascular Society

TEMPORAL PATTERNS AND PREDICTORS OF ENDOLEAK AFTER ELECTIVE ENDOVASCULAR AORTIC REPAIR

Ahsan Zil-E-Ali, Research Fellow, Penn State University, Jacob W. Soucy, Medical Student, Penn State College of Medicine, Faisal Aziz, Chief of Vascular Surgery, Penn State University

OBJECTIVE: Endoleak remains the most common complication after elective EVAR and may impact long-term repair durability. While endoleaks are routinely documented, their temporal behavior has not been well defined. We sought to characterize distinct timing patterns of endoleak and identify factors associated with each presentation.

METHODS: Retrospective analysis was performed using the Society for Vascular Surgery Vascular Quality Initiative (SVS-VQI) database. All elective EVAR procedures from 2003-2024 were included. Patients were categorized into four groups based on endoleak timing: no endoleak, completion-only, follow-up-only, and persistent endoleak present at both intervals. Patient characteristics, anatomic variables, and procedural details were compared. Multivariable logistic regression was used to identify independent predictors of each phenotype.

RESULTS: Among 90,546 patients undergoing elective EVAR, 25.4% demonstrated an endoleak at either completion or follow-up. Completion-only endoleaks were associated with advancing age, larger aneurysm diameter, iliac occlusion, and markers of procedural complexity. Persistent endoleaks were primarily associated with advancing age. Follow-up-only endoleaks were associated with advancing age, longer aortic neck length, higher BMI categories, and increased procedural complexity. Protective factors varied between groups with the follow-up-only group having the greatest number of individual protective factors. Endoleak subtype distribution shifted over time, with Type II endoleaks predominating at follow-up visits regardless of initial timing.

CONCLUSIONS: Endoleaks following elective EVAR demonstrate distinct temporal patterns with differing associated factors. These findings suggest that early seal-related leaks and delayed sac perfusion represent separate processes. Recognition of timing phenotypes may help refine intraoperative decision-making and postoperative surveillance strategies.

SCIENTIFIC ABSTRACT

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SEROTONERGIC ANTIDEPRESSANTS INCREASE TEMPORAL VARIABILITY OF PLATELET INHIBITION AFTER PERIPHERAL REVASCULARIZATION

Radha Bansal, MBBS, Shezan Fouzdar, MBBS, MS, Tiffany R. Bellomo, MD, Swechha Bhatt, MBBS, Adriana A. Rodriguez Alvarez, MD, Aseman Bagheri Sheshdeh, BS, Prajakta Lokhande, MD, Anahita Dua, MD, MS, MBA

Massachusetts General Hospital, Boston, MA, United States.

BACKGROUND: Platelet inhibition after peripheral artery disease (PAD) revascularization is increasingly recognized to be dynamic. Serotonin reuptake inhibitors potentially alter platelet activation and response to P2Y12 inhibition by inhibiting platelet serotonin uptake but its impact on the temporal stability of platelet inhibition remains unclear. We assessed whether serotonergic modulation alters platelet inhibition dynamics and affect the response to P2Y12 inhibition after revascularization.

METHODS: We analyzed 317 patients undergoing lower extremity revascularization with serial thromboelastography platelet mapping (TEG-PM), yielding 888 paired observations (median 6 visits/patient). Visit-to-visit variability in ADP-mediated platelet inhibition was quantified using log-transformed absolute differences between consecutive measurements. Serotonergic exposure (SSRI, SNRI, or trazodone) was modeled as a time-varying covariate using linear mixed-effects models adjusting for time between measurements and concurrent antithrombotic therapies. Transition analyses evaluated variability during changes in exposure status.

RESULTS: Serotonergic exposure (32% observations) was associated with increased variability in platelet inhibition ($\beta=0.22$, $p=0.03$). Variability peaked during initiation of serotonergic therapy ($\beta=0.71$, $p=0.01$), representing the primary driver of longitudinal variability, while sustained exposure showed attenuated effects. Ticagrelor use was independently associated with reduced variability ($\beta\approx-0.60$, $p<0.001$). Clopidogrel showed a trend toward increased variability ($\beta\approx0.22$, $p=0.09$).

CONCLUSIONS: Serotonergic antidepressants are associated with an underrecognized, initiation-driven instability in platelet inhibition following PAD revascularization. Dynamic platelet monitoring after revascularization suggests P2Y12 inhibitor selection may influence the stability of platelet inhibition in the serotonergic exposure.

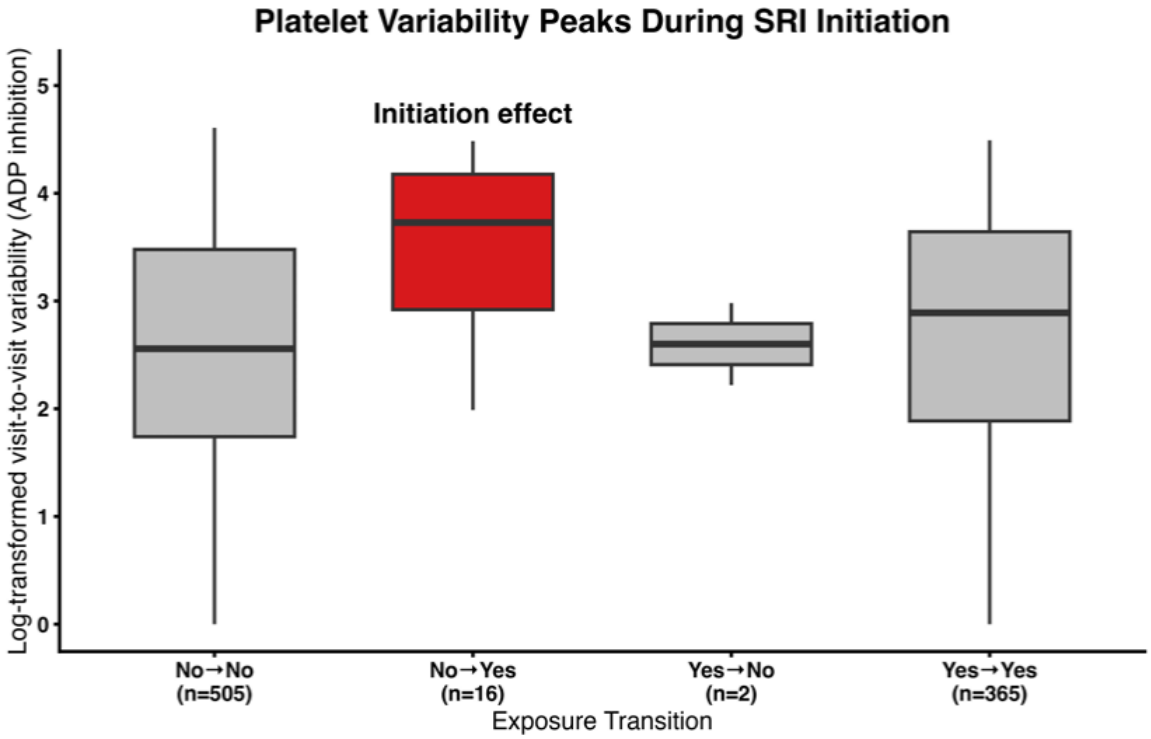

Figure 1. Variability peaked during initiation of serotonergic therapy (No→Yes), with lower variability during sustained exposure (Yes→Yes) and non-exposure (No→No). The small discontinuation group (Yes→No) is shown for completeness.

SCIENTIFIC ABSTRACT

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POOR GLYCEMIC CONTROL DRIVES FIBRINOLYSIS SHUTDOWN IN PERIPHERAL ARTERY DISEASE AND IDENTIFIES A PROTHROMBOTIC PHENOTYPE THROUGH VISCOELASTIC TESTING

Swechha Bhatt, Postdoctoral Research Fellow
Massachusetts General Hospital, Boston, MA, United States.

BACKGROUND: Fibrinolysis shutdown is an underrecognized contributor to thrombotic risk in peripheral artery disease (PAD). While thromboelastography (TEG) is used to assess platelet function, it also captures fibrin-driven clot dynamics relevant to post-revascularization outcomes. This study evaluated the association between glycemic control and fibrinolysis shutdown and its relevance for vascular surgeons.

METHODS: A retrospective cohort study was conducted at a tertiary care center from 2022 to 2025, including 192 patients with PAD undergoing revascularization with preoperative viscoelastic testing. Patients were stratified by hemoglobin A1c (HbA1c) <7% versus ≥7%. Fibrinolysis was assessed using TEG with LY30 as the primary parameter and categorized into predefined phenotypes. Multivariable logistic regression evaluated the association between HbA1c and severe fibrinolysis shutdown (LY30 ≤0.3%), adjusting for covariates. Statistical significance was defined as $p<0.05$.

RESULTS: Patients with HbA1c ≥7% demonstrated markedly impaired fibrinolysis, with near-complete suppression of clot breakdown (median LY30: 0.00 vs 0.30; p=0.015; Table 1) and a higher prevalence of severe fibrinolysis shutdown (74.7% vs 51.3%; p=0.009). After adjustment, poor glycemic control was associated with more than threefold higher odds of severe shutdown (adjusted OR 3.18, 95% CI 1.64–6.43; p<0.001; Figure 1), with higher predicted probability (68.8% vs 41.0%). Each 1% increase in HbA1c increased odds of shutdown by 50% (p=0.001), demonstrating a dose–response relationship.

CONCLUSIONS: Poor glycemic control is independently associated with fibrinolysis shutdown in PAD, identifying a prothrombotic phenotype not captured by platelet metrics alone. For vascular surgeons, incorporating fibrinolysis assessment with clot strength and fibrin contribution may improve perioperative risk stratification and guide targeted antithrombotic strategies.

Table 1. Association of glycemic control with fibrinolysis parameters and phenotype assessed by viscoelastic testing.

Variable	HbA1c <7%[1]	HbA1c ≥7%[1]	p-value[2]
LY30 (%)	0.30 [0.00–0.90]	0.00 [0.00–0.35]	**0.015**
Fibrinolysis phenotype, n (%)[3]			**0.009**
Severe shutdown (≤0.3%)	58 (51.3%)	59 (74.7%)	
Partial shutdown (0.3–0.8%)	25 (22.1%)	11 (13.9%)	
Physiologic (0.8–3.0%)	25 (22.1%)	7 (8.9%)	
Hyperfibrinolysis (>3.0%)	5 (4.4%)	2 (2.5%)	

[1] Median [interquartile range]; n (%).

[2] Continuous variables were compared using the Wilcoxon rank-sum test; categorical variables were compared using Pearson's Chi-squared test or Fisher's exact test, as appropriate.

[3] Fibrinolysis phenotypes were defined as follows: severe shutdown (LY30 ≤0.3%), partial shutdown (0.3–0.8%), physiologic fibrinolysis (0.8–3.0%), and hyperfibrinolysis (>3.0%).

Legend: This table presents the distribution of fibrinolysis activity, measured using LY30, stratified by glycemic control. Continuous LY30 values are summarized using median and interquartile range, and fibrinolysis phenotypes are categorized based on predefined LY30 thresholds. Comparisons between HbA1c groups evaluate differences in both continuous fibrinolysis measures and categorical fibrinolysis phenotypes.

South Asian American Vascular Society

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