

ICAD Reloaded

Symptomatic intracranial atherosclerotic disease remains a leading cause of recurrent ischemic stroke. Aggressive medical therapy is first - line —yet selected patients still fail. The question is not whether to intervene, but **who, when, and how.**

Presented By: Srihari Sundararajan, MD ('Sri')

University Radiology Group & Rutgers Robert Wood Johnson Medical School

Chief of Interventional Neuroradiology | Assistant Professor of Radiology

Attending in Neurointerventional Radiology, Interventional Radiology, and Neuroradiology

September 18, 2026

DISCLOSURES

Disclosures

None

The presenter has no relevant financial relationships, conflicts of interest, or commercial support to disclose in relation to this presentation.

All clinical case examples presented are illustrative and educational in nature. Off-label device use is discussed for educational purposes only and does not constitute a clinical recommendation.

Why ICAD Still Matters

Prevalence

Causes ~8–10 % of ischemic strokes in the US; disproportionately affects Asian, Black, and Hispanic populations —a true health equity issue.

Early Recurrence Risk

Symptomatic severe stenosis (70–99%) carries meaningfully early recurrent stroke and TIA risk, particularly within the first 30–90 days after an index event.

Stroke Mechanisms

Artery-to-artery embolism, perforator compromise, and hemodynamic failure —each demands a distinct therapeutic approach.

The Endovascular Dilemma

Recurrence despite optimized medical therapy creates the critical clinical question: who benefits from intervention, and when is the risk acceptable?

Not Every Stenosis Is the Same Disease

Percent stenosis alone is an insufficient surrogate for stroke risk or procedural hazard. The biology beneath the lumen matters as much as the lumen itself.

Plaque Morphology & Lesion Length

Eccentric versus concentric plaque, lesion length, vessel diameter, and local perforator density each independently alter the risk-benefit calculus of intervention.

Embolic vs. Hemodynamic Phenotype

Artery-to-artery thrombus differs fundamentally from flow-limiting hypoperfusion. Misidentifying the mechanism leads to mismatched therapy.

Perforator Density & Collaterals

Perforator-rich segments (basilar, M1) carry higher snowplow risk. Robust collateral status modifies hemodynamic vulnerability and may reduce urgency for intervention.

Advanced Imaging

Vessel-wall MRI identifying plaque enhancement and perfusion imaging demonstrating impaired cerebrovascular reserve refine mechanism far beyond simple percent stenosis on luminal angiography.

LANDMARK EVIDENCE

The Natural History That Started the Debate: WASID

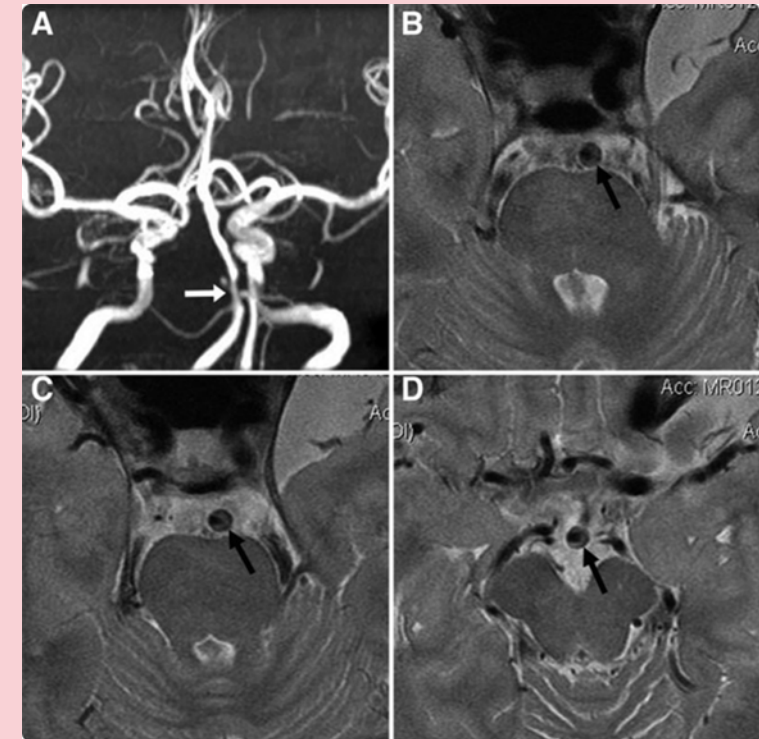
The **Warfarin -Aspirin Symptomatic Intracranial Disease (WASID)** trial established the epidemiologic foundation for modern ICAD management. Patients with symptomatic 70–99% intracranial stenosis carried a 2-year stroke risk approaching 20–25% in the territory of the stenotic artery.

Key findings that shaped subsequent trials:

- Recent symptoms, severe stenosis $\geq 70\%$, and poor collateral status identified highest-risk subgroups
- Warfarin offered **no advantage** over aspirin and caused significantly more hemorrhagic harm
- Medical therapy (aspirin) became the foundation—but recurrence rates remained clinically unacceptable in high-risk patients
- This residual risk created the intellectual rationale for endovascular investigation

Chimowitz et al., NEJM 2005; Kasner et al., Circulation 2006

WASID Era Risk at a Glance



Stroke —Evaluating Intracranial Atherosclerosis (2014)

~23% 2-year ipsilateral stroke risk with 70–99% stenosis on aspirin alone

Warfarin arm stopped early due to excess major hemorrhage and death

Conclusion: Aspirin = standard; endovascular research mandated for high-risk patients.

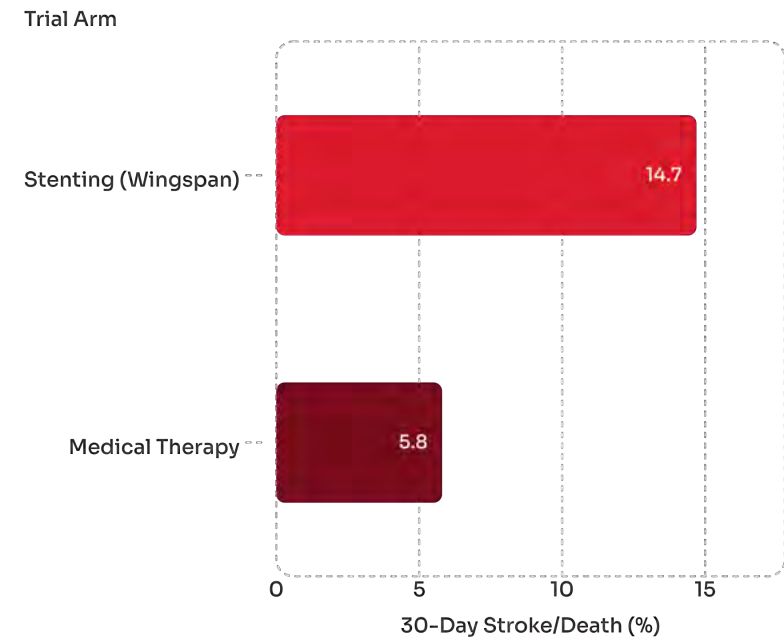
SAMMPRIS Changed Everything

The **Stenting and Aggressive Medical Management for Preventing Recurrent Stroke in Intracranial Stenosis (SAMMPRIS)** trial was the pivotal inflection point. It compared aggressive medical therapy (AMT) alone versus AMT plus Wingspan PTAS in patients with recent TIA or stroke and 70–99% stenosis.

- **30 - day stroke/death rate:** 14.7% in the stenting arm vs. 5.8% in the medical arm — trial stopped early
- Aggressive medical management included aspirin + clopidogrel for 90 days plus rigorous risk factor control
- Intensive medical therapy significantly outperformed early Wingspan PTAS
- DAPT plus high intensity statins and strict BP targets became the **new standard of care**

Chimowitz et al., NEJM 2011; Lancet 2014

JCM — PTAS for Intracranial Stenosis Post - SAMMPRIS (2025)



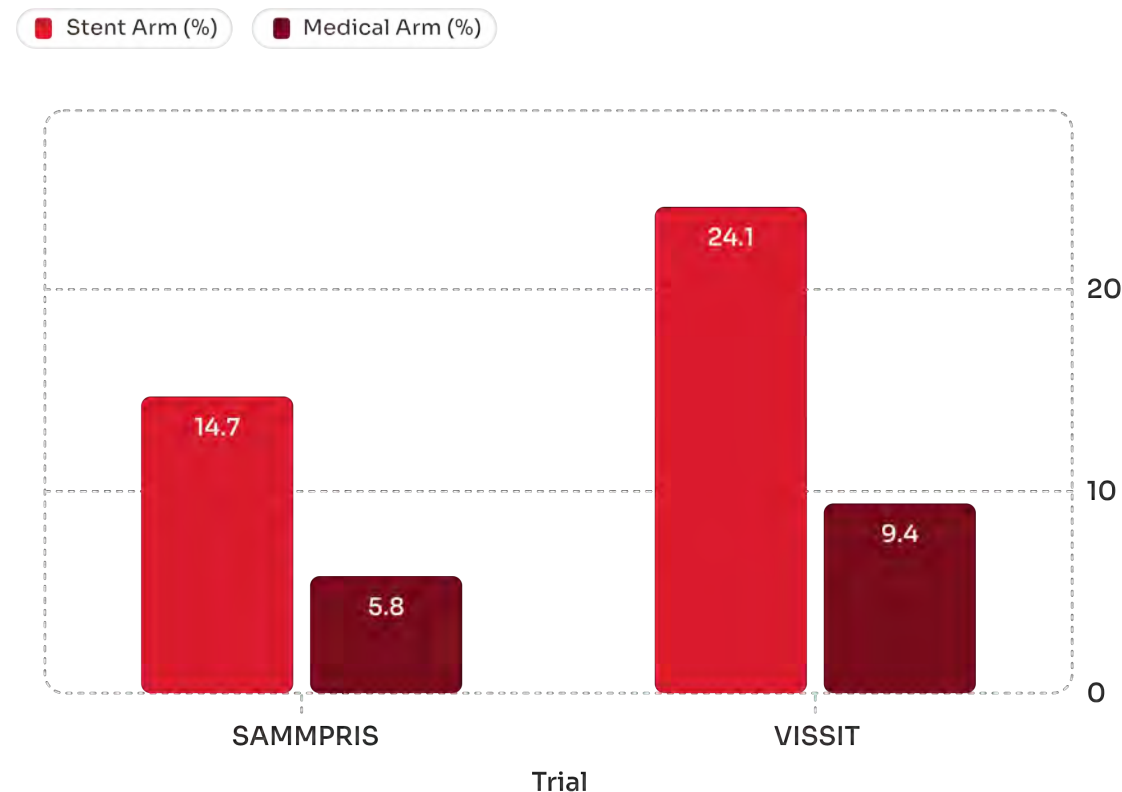
30 - day stroke/death rates, SAMMPRIS (Chimowitz et al., NEJM 2011)

VISSIT Reinforced the Warning

The **Vitesse Intracranial Stent Study for Ischemic Therapy (VISSIT)** evaluated balloon -expandable intracranial stenting for symptomatic high -grade ICAD. Where SAMMPRIS used a self -expanding device, VISSIT tested a different stent platform —yet arrived at the same sobering conclusion.

- Balloon -expandable stenting produced excess early stroke and death events compared to medical therapy
- The 30 - day rate of stroke or TIA was significantly higher in the stent arm
- Routine intervention for symptomatic ICAD could not be justified on any platform
- These two landmark trials collectively and appropriately reset the field toward medical therapy as the unambiguous first - line standard

Zaidat et al., JAMA 2015



30 - day primary endpoint rates. SAMMPRIS: Chimowitz NEJM 2011; VISSIT: Zaidat JAMA 2015.

Aggressive Medical Therapy Is the Baseline, Not the Backup

SAMMPRIS and VISSIT established that maximally optimized medical therapy is the non-negotiable foundation. No endovascular discussion is legitimate until the following pillars are firmly in place —and confirmed adherent.



Dual Antiplatelet Therapy

Aspirin + clopidogrel for approximately 90 days following a recent severe symptomatic ICAD event, transitioning to aspirin monotherapy thereafter.



High-Intensity Statin

Rosuvastatin or atorvastatin targeting aggressive LDL reduction. Plaque stabilization and anti-inflammatory effects are as important as lipid lowering.



Vascular Risk Control

Strict blood pressure management, diabetes optimization, smoking cessation, supervised exercise, and weight management —all simultaneously.



Confirm True Adherence

Verify medication adherence objectively before declaring medical failure. Apparent failure on therapy is meaningless if the patient is not actually taking the medications.

But Some Patients Still Fail

A clinically meaningful subset of patients experience recurrent territorial events despite **confirmed adherence** to optimized dual antiplatelet, statin, and risk factor management. Identifying this subset —and distinguishing true medical failure from other causes —is the decisive clinical step.

Recurrent Territorial Events

Recurrent TIA or ischemic stroke clearly in the territory of the stenotic artery, occurring despite confirmed DAPT and statin adherence and optimized vascular risk control.

Severe Stenosis + Compromised Perfusion

Documented 70 –99% stenosis combined with objective evidence of impaired distal perfusion on perfusion imaging or poor angiographic collateral status.

Unstable Embolic Lesion

Repeated embolic events from an angiographically or vessel wall - MRI—confirmed unstable lesion despite full dual antiplatelet and statin therapy.

Exclude Mimics First

Careful exclusion of non -ICAD stroke mechanisms — cardioembolic sources, small vessel disease, hyperperfusion — before any declaration of medical failure and referral for intervention.

The Problem With Asking Only "Does Stenting Work?"

The binary framing of early randomized trials obscured critical heterogeneity. Early trials enrolled phenotypically diverse patients into a single treatment arm and intervened during biologically unstable acute periods—a recipe for periprocedural harm regardless of device quality.

1 Mixed Lesion Phenotypes

Perforator-bearing basilar and M1 lesions behave fundamentally differently from purely flow-limiting MCA or ICA stenoses. Lumping them together dilutes signal.

2 Unstable Intervention Timing

Early trials frequently intervened within days of an acute stroke, during a period of plaque instability, systemic inflammation, and disrupted autoregulation—amplifying procedural risk substantially.

3 Technical Evolution Since Early Trials

Operator experience, access system profiles, microwire technology, balloon sizing principles, and antiplatelet management have all evolved dramatically since 2011. Early trial results do not reflect modern technique.

WEAVE: What Better Selection Looked Like

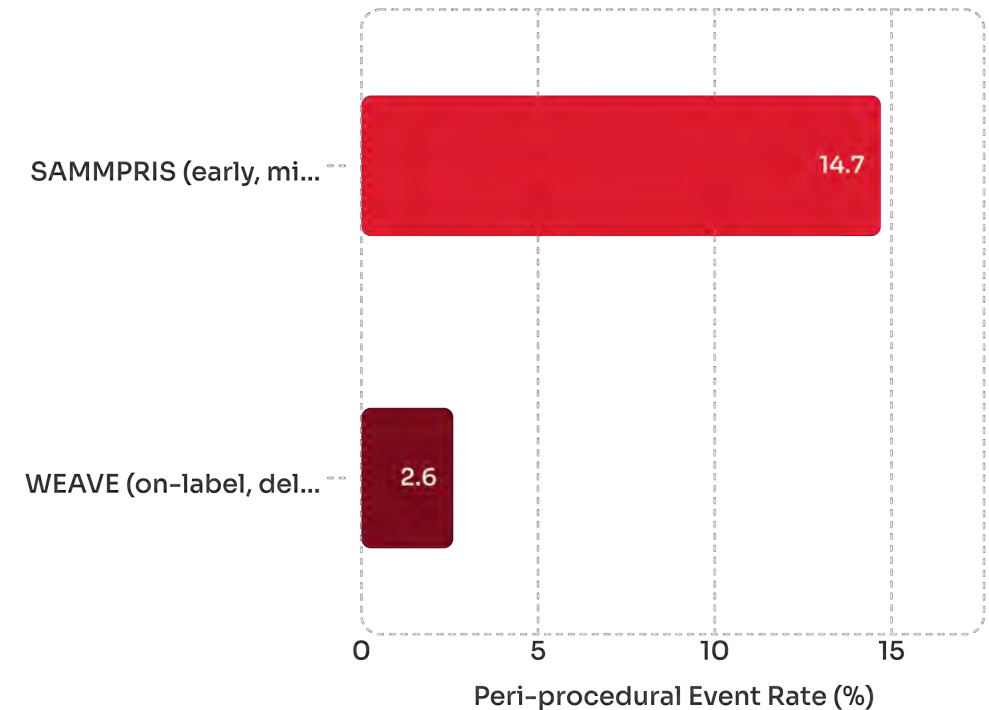
The Wingspan One - Year Vascular Events and Neurologic Outcomes

(WEAVE) trial was a post-market surveillance safety study designed to evaluate Wingspan PTAS under strict on-label conditions. It was not a randomized trial against medical therapy—but its findings were instructive.

- **Strict on-label criteria:** 70–99% stenosis, failure of ≥ 2 antithrombotics, ≥ 8 days from qualifying event
- Procedures performed only by experienced, credentialed operators at high-volume centers
- 72-hour periprocedural stroke/death/ICH rate: **2.6%**—dramatically lower than SAMMPRIS's 14.7%
- Demonstrated that **technique and selection materially affect procedural risk**, independent of device

Alexander et al., Stroke 2019

Study / Condition



72-hour periprocedural stroke/death/ICH. WEAVE: Alexander et al., Stroke 2019.

WOVEN: Safety Is Not the Same as Efficacy

The **Wingspan One - Year Vascular Events and Neurological Outcomes (WOVEN)** registry extended follow - up from WEAVE to 12 months in patients who underwent on - label Wingspan stenting. The one - year outcomes were compared against historical early - stenting experience.

- One - year stroke or death: **8.5%** —improved versus historical early - stenting registries but not trivial
- Recurrent events continued to occur beyond the periprocedural window, highlighting residual longitudinal risk
- Registry follow - up provides important feasibility and durability data in selected patients
- **Critical distinction:** Demonstrating improved procedural safety does not establish superiority over optimized medical therapy — only a randomized trial can do that

Alexander et al., J Neurointerv Surg 2021

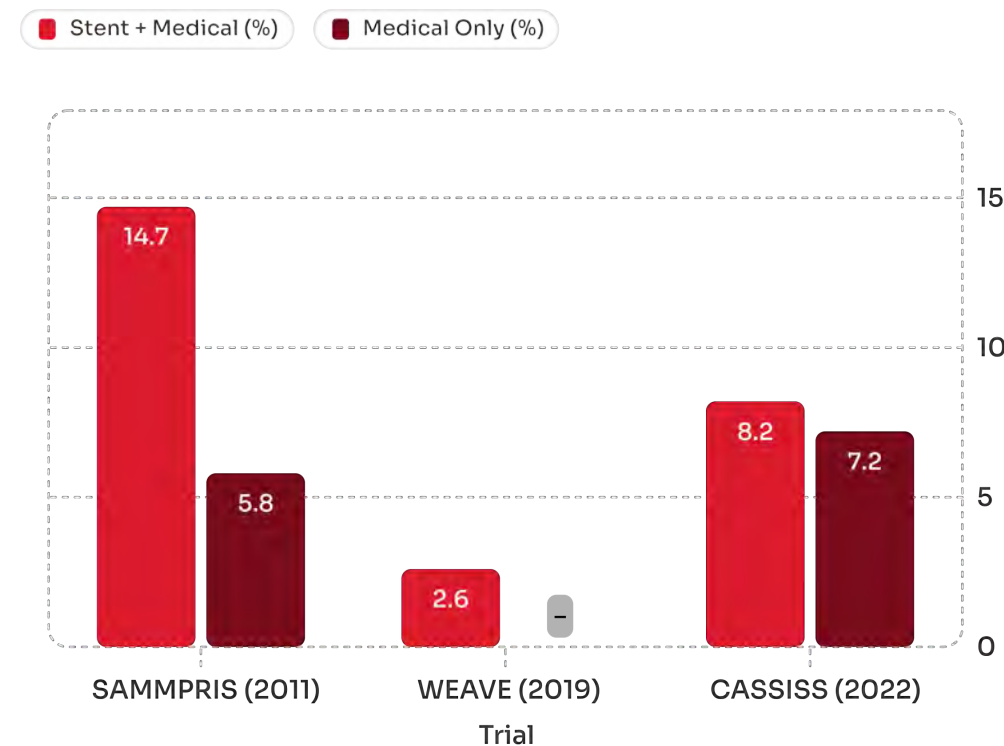
The Key Message

WEAVE + WOVEN together demonstrate that **skilled, delayed, on - label Wingspan stenting is far safer** than early trials suggested —but neither study replaces randomized comparative evidence against modern medical therapy.

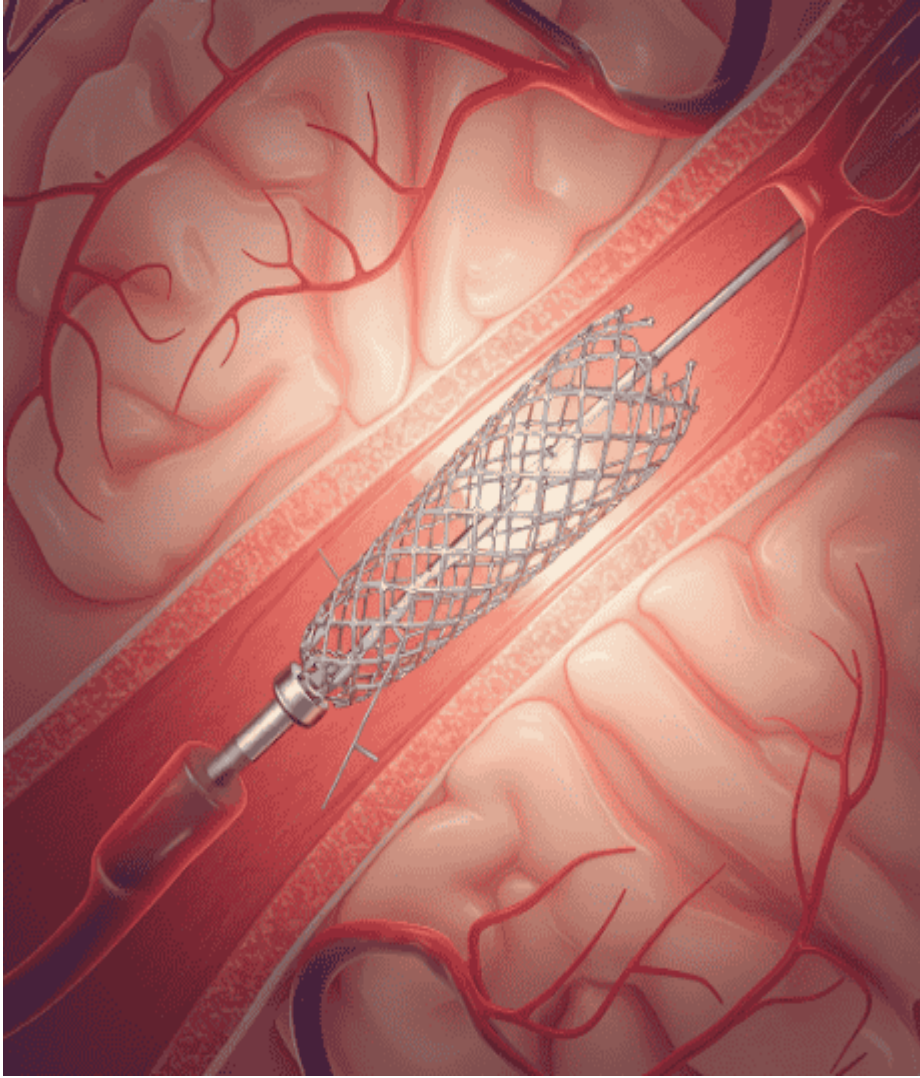
CASSISS: Selection Improved, Superiority Did Not Appear

The **Chinese Angioplasty & Stenting for Symptomatic Intracranial Severe Stenosis (CASSISS)** trial was the most methodologically rigorous attempt to revisit endovascular therapy under modern conditions. Patients were randomized at least 3 weeks after the qualifying event, and procedures were performed by experienced operators at high - volume centers.

- Delayed stenting significantly reduced the periprocedural hazard compared to SAMMPRIS -era experience
- Adding stenting to medical therapy did **not significantly improve** the primary endpoint (stroke, TIA, or death within 30 days or ischemic stroke beyond 30 days in the territory) vs. medical therapy alone
- Modern procedural safety does not automatically establish routine clinical efficacy
- The question of **who specifically benefits** remains unanswered and drives ongoing research



Primary event rates across key ICAD endovascular trials. CASSISS: Gao et al., JAMA 2022.



So Who Might Still Be an Endovascular Candidate?

The evidence does not support routine stenting. It does support patients meeting all of the following criteria:

carefully selected, experienced - center intervention in

1

Severe Focal Stenosis 70 –99%

Refractory to optimized, confirmed - adherent medical therapy. Imaging - confirmed ICAD etiology with appropriate anatomical target.

2

Recurrent Territorial Events

Recurrent TIA or stroke despite DAPT + high - intensity statin, with events clearly attributable to the stenotic lesion territory.

3

Favorable Lesion Phenotype

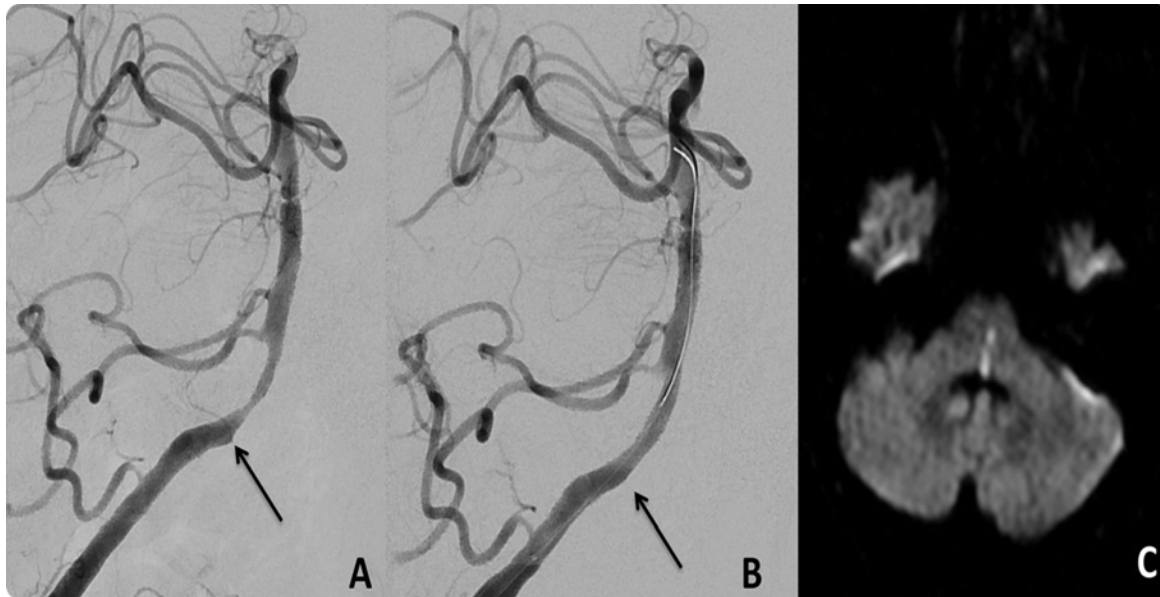
Non - perforator dominant mechanism favored; objective hemodynamic compromise on perfusion imaging; poor collateral status amplifying ischemic vulnerability.

4

Delayed Timing & Amenable Anatomy

Intervention planned in the delayed, stable phase —not acutely. Anatomy amenable to controlled submaximal angioplasty or stenting without excessive tortuosity or vessel angulation.

Angioplasty Alone: Less Metal, Different Trade offs



Gentle, **undersized (submaximal) balloon angioplasty** can meaningfully improve luminal caliber and restore flow without placing a permanent intracranial implant. This approach has specific advantages and well-defined limitations:

- **Advantages:** No permanent metal scaffold; may reduce perforator snowplow effect; eliminates chronic stent thrombosis and in-stent restenosis risk; reversible if complications occur
- **Limitations:** Elastic recoil (immediate loss of gain); risk of dissection requiring bailout stenting; long-term restenosis rates higher than stenting in some series
- **Technique is everything:** Slow, controlled inflation (often 1–2 minutes); diameter target 80–90% of reference vessel; never 1:1 sizing
- A flow-limiting dissection after angioplasty mandates stenting as a bailout —always have a stent available on the table

ICAD Stent Options: Matching the Tool to the Lesion

When stenting is required —either as planned primary therapy or as bailout after angioplasty—device selection profoundly affects both technical success and downstream risk. The goal is **improved perfusion, not anatomic perfection**. Targeting ~50% residual stenosis is often safer than forcing a 0% result.

Stroke —Future Endovascular Treatments for Intracranial Stenosis

Wingspan (Self - Expanding)

Nitinol self - expanding stent; the only FDA - approved intracranial device for ICAD. Delivered over a Gateway balloon. Gentle, gradual radial force. Used in SAMMPRIS, WEAVE, WOVEN. Preferred for tortuous anatomy and smaller vessels (2.5 –4.5 mm).

Atlas (Self - Expanding)

Lower - profile nitinol stent system. Gaining traction in neurointerventional practice for ICAD due to improved trackability, flexibility, and compatibility with standard microcatheters. Frequently used at centers including our own for elective ICAD stenting cases.

Onyx Resolute / Balloon - Expandable (Off - Label)

Balloon - mounted coronary drug - eluting stents offer precise placement and high radial force. However, their rapid full expansion can cause exaggerated intimal hyperplasia and over - distension of intracranial vessels —a significant concern. Best reserved for very specific anatomies with operator expertise.

Coronary Bare - Metal & DES (Off - Label)

Used in literature for ICAD at select high - volume Asian centers, particularly for posterior circulation lesions in larger - caliber vessels. Risk of accelerated intimal hyperplasia from oversizing remains the critical limiting factor. Not routine in US practice.

⚠ The goal of intracranial stenting is to improve perfusion —not to achieve 100% luminal reconstruction. Oversizing stents risks perforator compromise, vessel dissection, and exaggerated neointimal hyperplasia.

The Tigertriever: An Adjustable Stent-Retriever as an ICAD Tool

The **Tigertriever** (Rapid Medical) is a fully adjustable, size -tunable stent -retriever that has found a role beyond its primary indication of acute large -vessel occlusion thrombectomy. Its unique radially adjustable design allows operators to precisely control expansion diameter —a highly desirable property when treating the delicate, perforator -adjacent anatomy of ICAD lesions.

Available Sizes

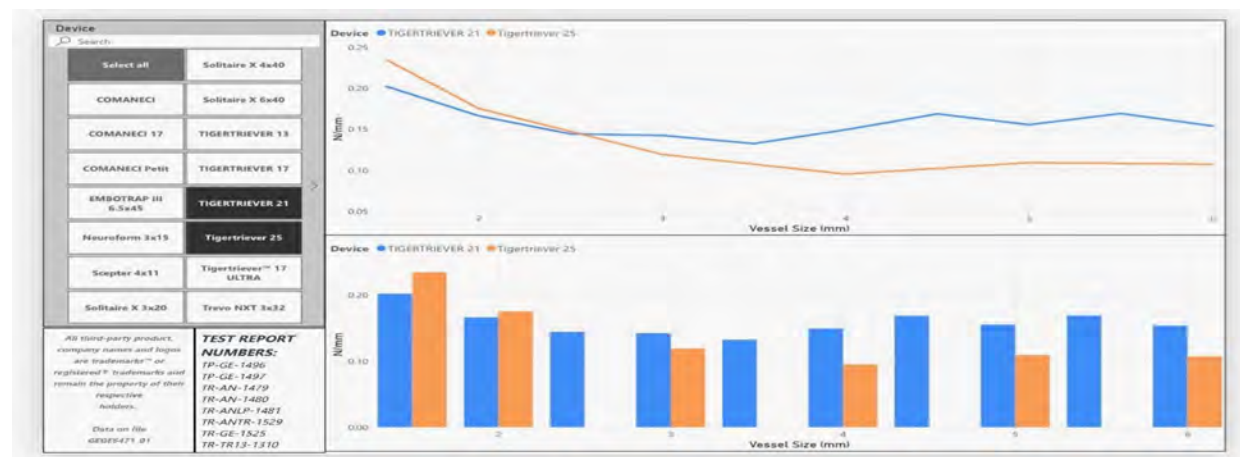
- **Tigertriever 13:** For the smallest intracranial vessels —distal M2, A2, P2 segments and other small -caliber perforating territory vessels
- **Tigertriever 17:** For smaller intracranial vessels —covers distal MCA, smaller basilar perforating territory vessels
- **Tigertriever 21:** Mid -range vessel sizes —applicable to M1, mid -basilar, and comparable intracranial segments
- **Tigertriever 25:** The newest, largest size —optimized for proximal M1, basilar trunk, and supraclinoid ICA lesions
- Adjustability allows the operator to match device diameter precisely to vessel caliber rather than accepting a fixed footprint

Potential Role in ICAD

Used as a temporary scaffolding tool during angioplasty, or deployed briefly to assess vessel response and lesion stability b committing to a permanent implant. Emerging case series suggest feasibility as a flow -restoration bridge in medically refractory ICAD with thrombotic occlusion.

Radial Force Profile: Tigertriever 21 vs. 25

The graph below illustrates how radial force (N/mm) varies with vessel diameter for the Tigertriever 21 and 25. The Tigertriever 21 delivers relatively stable, higher radial force across smaller vessel sizes (2 –5 mm), while the Tigertriever 25 starts lower at smaller diameters and becomes increasingly competitive at larger vessel sizes (~5 –6 mm). This complementary radial force profile allows size-appropriate device selection based on target vessel caliber.



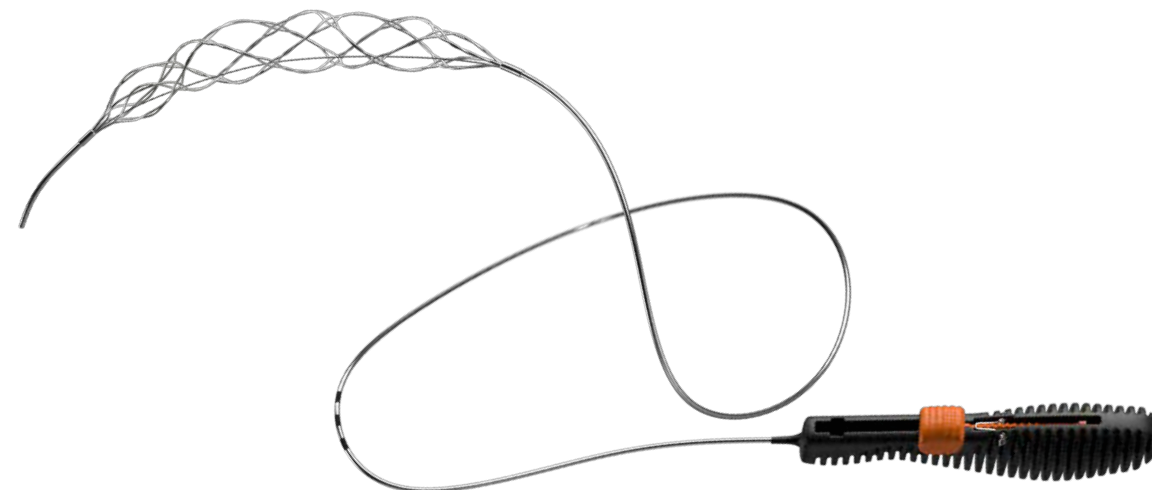
Why Adjustability Matters in ICAD

Standard stents commit to a fixed radial force at full expansion. In tortuous intracranial arteries with perforator -bearing stenoses, the ability to **fine -tune expansion diameter in real time** provides a critical margin of safety unavailable with conventional devices.

The Tigertriever's controlled deployment allows stepwise expansion —the operator can assess vessel and perforator response before full diameter is reached, enabling a "test -and -adjust" approach not possible with balloon -mounted or fixed self -expanding systems.

The complementary radial force profiles of the Tigertriever 21 and Tigertriever 25 further enable size -matched device selection: use the 21 for smaller -to -mid caliber vessels where higher radial force at smaller diameters is advantageous, and the 25 for proximal, larger -caliber targets where its profile becomes optimal.

- ❑ Not FDA -approved for ICAD stenting; use is off -label and should occur within experienced neurointerventional teams with appropriate informed consent.



Technical Principles

01

Proximal Support

Establish stable, coaxial guide catheter support without inducing catheter-mediated vasospasm or vessel injury. Inadequate support leads to device instability and loss of wire position.

03

Submaximal Balloon Sizing

Target balloon diameter at 80–90 % of reference vessel. Avoid 1:1 sizing. Slow, controlled inflation over 60–120 seconds minimizes acute plaque disruption and perforator displacement.

02

Atraumatic Lesion Crossing

Cross the stenosis using a soft, compliant microwire with a gentle J-curve. Avoid stiff wires that straighten tortuous anatomy and produce sub-intimal passage or dissection.

04

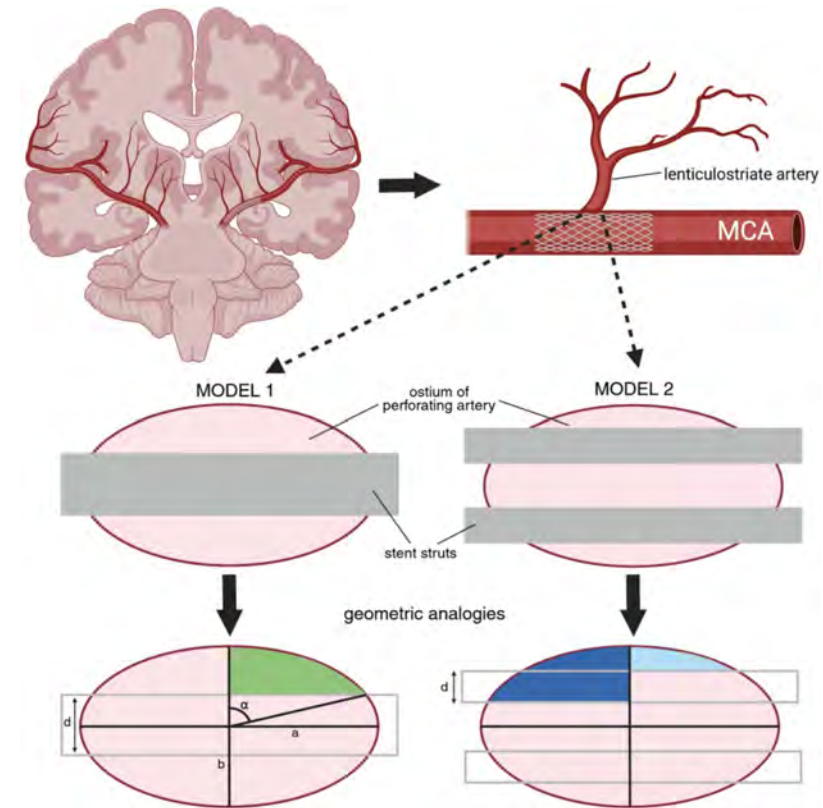
Meticulous Post-Treatment Assessment

Verify distal runoff, assess perforator patency, exclude dissection, quantify residual stenosis —before removing access. A satisfactory angiographic result is confirmed, not assumed.

The Achilles Heel: Perforators

The most feared periprocedural complication of intracranial angioplasty and stenting is not dissection or vessel perforation—it is **perforator occlusion**, mediated by the "snowplow effect." Balloon or stent expansion redistributes atherosclerotic plaque laterally into the ostia of en - passage perforating arteries, producing immediate or delayed occlusion.

- **Highest - risk segments:** Basilar artery (bilateral pontine perforators), M1 segment (lenticulostriate arteries), and ACA A1 segment
- Presenting infarcts in perforator territory on pre - procedure MRI may signal an **unfavorable phenotype** — suggesting the mechanism is already perforator compromise rather than hemodynamic failure
- Minimizing radial expansion force, avoiding oversizing, and using the slowest possible inflation technique are the primary mitigation strategies
- No device or technique eliminates the snowplow risk —it must be factored into patient selection from the outset



Neurosurgical Review —Lenticulostriate & Pontine Artery Patency After Intracranial Stenting (2025)

Clinical Pearl

When pre - procedure DWI shows lacunar or perforator - territory infarcts *ipsilateral to the stenosis*, this strongly suggests the dominant stroke mechanism is perforator occlusion — not hemodynamic failure. **Angioplasty or stenting will not address —and may worsen —this mechanism.**

Antiplatelets Are Part of the Procedure

Intracranial stenting creates an acutely thrombogenic metallic surface in a high low-flow, high-shear cerebral arterial environment. Oral P2Y12 loading may be delayed, unreliable, or impossible in emergent or acute-on-chronic scenarios. Intravenous antiplatelet agents provide **rapid, titratable, and reversible platelet inhibition** precisely precisely when minutes matter most.

Elective ICAD Stenting Pre-load with aspirin + clopidogrel or ticagrelor $\geq$57 days prior. Platelet-function testing (VerifyNow P2Y12) to confirm adequate inhibition before proceeding is strongly recommended at experienced centers.	Subacute / Urgent Stenting When oral loading is unreliable or timing is compressed, IV cangrelor provides immediate, predictable P2Y12 inhibition. Bridging to oral P2Y12 therapy follows after the procedure is complete.	Rescue / Thrombotic Complication In-stent thrombosis or platelet-rich clot during the procedure requires prompt GP IIb/IIIa blockade with tirofiban or eptifibatide. These agents target the final common pathway of platelet aggregation for maximal, immediate effect.
---	--	--

Cangrelor: A P2Y₁₂ Inhibitor With an On/Off Switch

Cangrelor is a direct-acting, reversible, IV P2Y₁₂ receptor antagonist that requires **no hepatic conversion** —a critical advantage over clopidogrel in the periprocedural setting where time and reliability are paramount.



Rapid Onset

Near-complete platelet inhibition within 2 minutes of IV bolus.
No loading latency, no clopidogrel resistance variability, no CYP2C19 pharmacogenomic uncertainty.



Ultra-Short Half-Life

Plasma half-life of approximately 3–6 minutes. Platelet function recovers to near-baseline within 30–60 minutes of infusion cessation —critical when hemorrhagic risk remains dynamically evolving.



Predictable Reversibility

The "on/off switch" pharmacology allows real-time titration of platelet inhibition —an unmatched property among antiplatelet agents for high-stakes intracranial procedures.



Transition to Oral Therapy

Ticagrelor can overlap with cangrelor infusion. Clopidogrel or prasugrel should be given only *after* the cangrelor infusion stops to avoid competitive receptor binding and pharmacological antagonism.

Tirofiban: Fast GP IIb/IIIa Rescue

Tirofiban is a small - molecule, reversible glycoprotein IIb/IIIa antagonist that blocks fibrinogen binding —the **final common pathway of platelet aggregation** , regardless of upstream activation trigger. It is the workhorse rescue agent during intracranial intervention when thrombotic complications emerge.

Primary Indications in ICAD Intervention

- Acute in - stent thrombosis recognized on peri - procedural angiography
- Platelet - rich thrombus complicating angioplasty or guidewire passage
- High - risk emergent stenting where immediate, maximal platelet inhibition is required

Key Pharmacological Considerations

- Rapid IV onset; offset slower than cangrelor (~4 –8 hours) but faster than oral agents
- Renal clearance —dose - adjust in CKD
- Potent final - pathway blockade mandates **individualized hemorrhagic risk assessment** before use

Cangrelor vs. Tirofiban: Quick Reference

Property	Cangrelor	Tirofiban
Target	P2Y12	GP IIb/IIIa
Half - life	3–6 min	~2 hrs
Offset	~60 min	4–8 hrs
Primary use	Planned stenting	Rescue thrombosis
Renal adj.	No	Yes

Neither agent replaces meticulous technique or definitive oral antiplatelet planning.

Case in Focus: Medically Refractory M1 ICAD

Clinical Scenario: A 67-year-old man with hypertension, hyperlipidemia, and type 2 diabetes presents with his third left hemispheric TIA in 6 weeks. MRI demonstrates scattered acute DWI lesions in the left MCA territory with no perforator - territory infarcts. CTA confirms 82% left M1 stenosis. He is confirmed adherent to aspirin + clopidogrel, rosuvastatin 40 mg, and has achieved target BP control.

Decision Framework Applied

- **Mechanism:** Artery - to - artery embolic pattern —favorable phenotype for intervention
- **Timing:** Intervention deferred to 3 weeks after last TIA to allow lesion stabilization
- **Approach:** Submaximal balloon angioplasty targeting ~75% of reference M1 diameter; Atlas stent on standby for bailout
- **Antiplatelet:** Cangrelor infusion initiated at sheath insertion; transitioned to ticagrelor post - procedure
- **Result:** Residual ~35% stenosis accepted —perfusion target achieved. No perforator signal loss. Patient event - free at 12 months



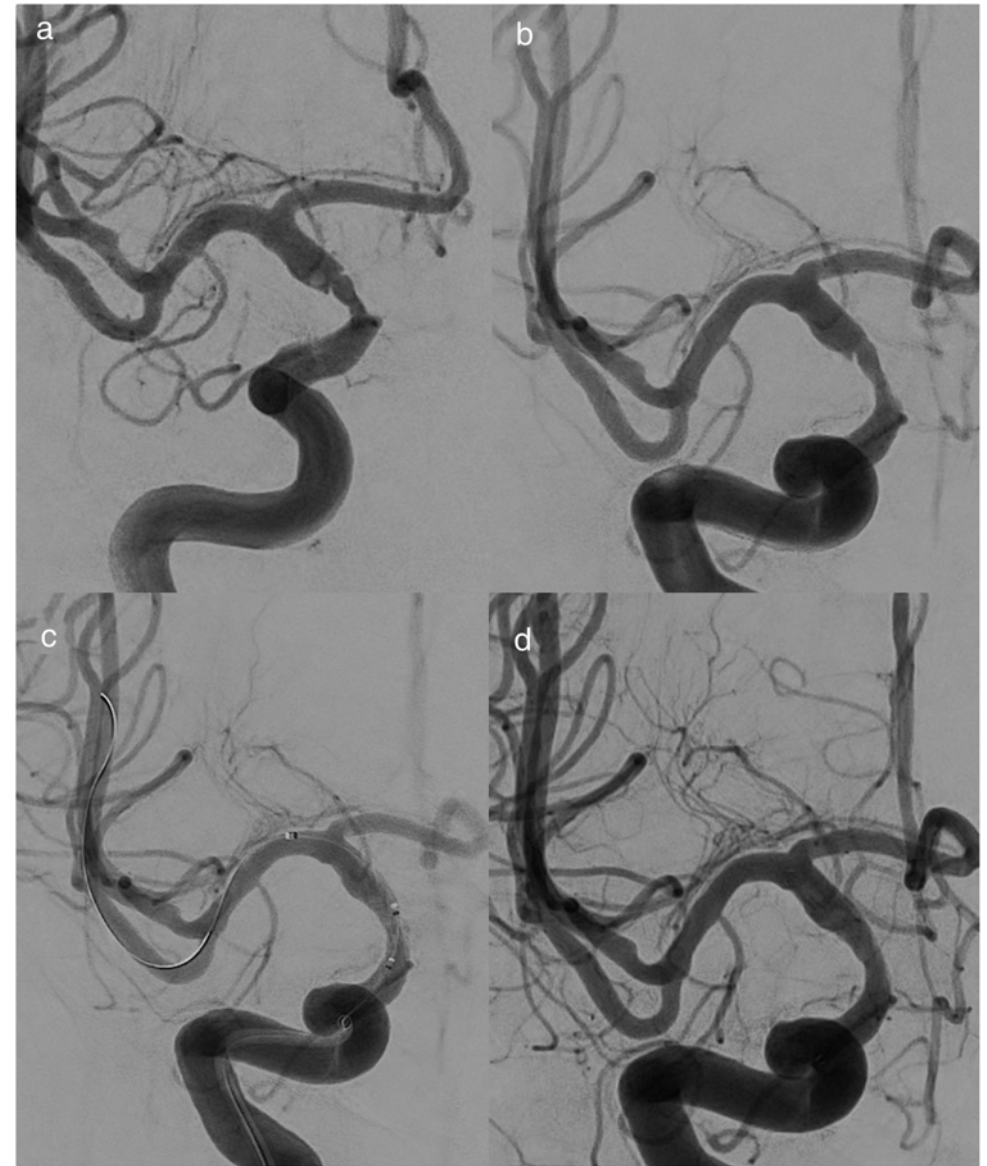
Residual stenosis of 30 –50% is often the appropriate endpoint —the goal is perfusion restoration, not anatomic perfection.

Case in Focus: Symptomatic Supraclinoid ICA Stenosis

Clinical Scenario: A 58-year-old woman with poorly controlled hypertension and a 40 pack-year smoking history presents with two episodes of right-sided amaurosis fugax and one right hemispheric TIA over 3 weeks. MRA demonstrates high-grade stenosis of the supraclinoid left ICA. DSA confirms 78% stenosis at the supraclinoid segment with sluggish distal filling. She has been on aspirin + clopidogrel and rosuvastatin 40 mg for 6 weeks with confirmed adherence and no further events—until a fourth TIA prompts re-evaluation.

Decision Framework Applied

- **Mechanism:** Hemodynamic + embolic mixed phenotype—perfusion imaging confirms impaired cerebrovascular reserve in the left MCA territory
- **Lesion anatomy:** Supraclinoid ICA—relatively low perforator density compared to M1 or basilar; favorable anatomy for intervention
- **Timing:** Intervention planned at 6 weeks post-index event; lesion biologically stable
- **Approach:** Submaximal angioplasty with Gateway balloon (2.5 × 15 mm) followed by Wingspan stent deployment; target ~50% residual stenosis
- **Antiplatelet:** Cangrelor bridge initiated at sheath insertion; transitioned to ticagrelor 90 mg BID post-procedure
- **Result:** Residual ~40% stenosis; robust distal ICA and MCA filling restored. No new DWI lesions on 24-hour post-procedure MRI. Event-free at 18 months.



The supraclinoid ICA is a favorable ICAD target—lower perforator density than M1 or basilar, accessible anatomy, and robust collateral reserve via the ophthalmic and posterior communicating arteries.

CASE EXAMPLE

Case in Focus: Symptomatic Basilar Artery Stenosis

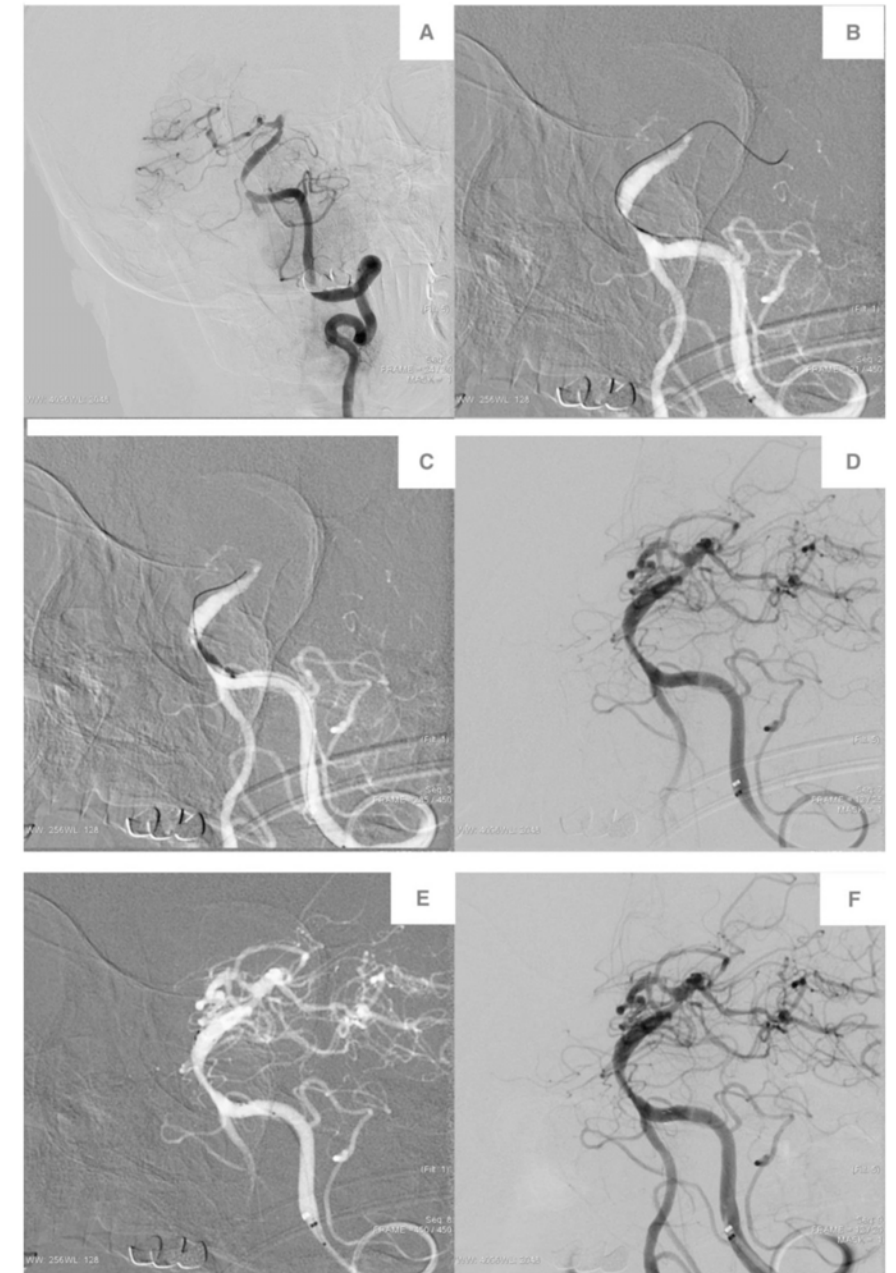
Clinical Scenario: A 72-year-old man with hypertension, diabetes, and hyperlipidemia presents with two episodes of bilateral limb weakness, dysarthria, and vertigo over 10 days — classic posterior circulation TIAs. MRI DWI demonstrates bilateral pontine and cerebellar acute infarcts. DSA confirms 85% mid-basilar stenosis. He has been on aspirin + clopidogrel and rosuvastatin 40 mg for 8 weeks with confirmed adherence. A third event — transient quadriplegia — occurs at week 9, prompting urgent reassessment.

Decision Framework Applied

- **Mechanism:** Hemodynamic failure — bilateral posterior circulation infarcts, impaired basilar flow on perfusion imaging, poor posterior communicating artery collaterals bilaterally
- **Lesion anatomy:** Mid-basilar — highest-risk ICAD location; dense bilateral pontine perforators; snowplow risk is maximal here
- **Risk-benefit deliberation:** Bilateral pontine perforator territory at stake; natural history of recurrence on medical therapy in this phenotype is catastrophic; shared decision-making with patient and family documented
- **Timing:** Intervention at 10 weeks post-index event; lesion stable on repeat imaging
- **Approach:** Submaximal angioplasty with Gateway balloon (2.0 × 9 mm); deliberate undersizing to minimize perforator displacement; no stent deployed — angioplasty alone accepted with ~45% residual stenosis and restored basilar flow
- **Antiplatelet:** Cangrelor infusion throughout; transitioned to ticagrelor post procedure
- **Result:** Robust basilar and bilateral PCA filling restored. No new DWI lesions at 24-hour MRI. Event-free at 24 months on dual antiplatelet therapy.



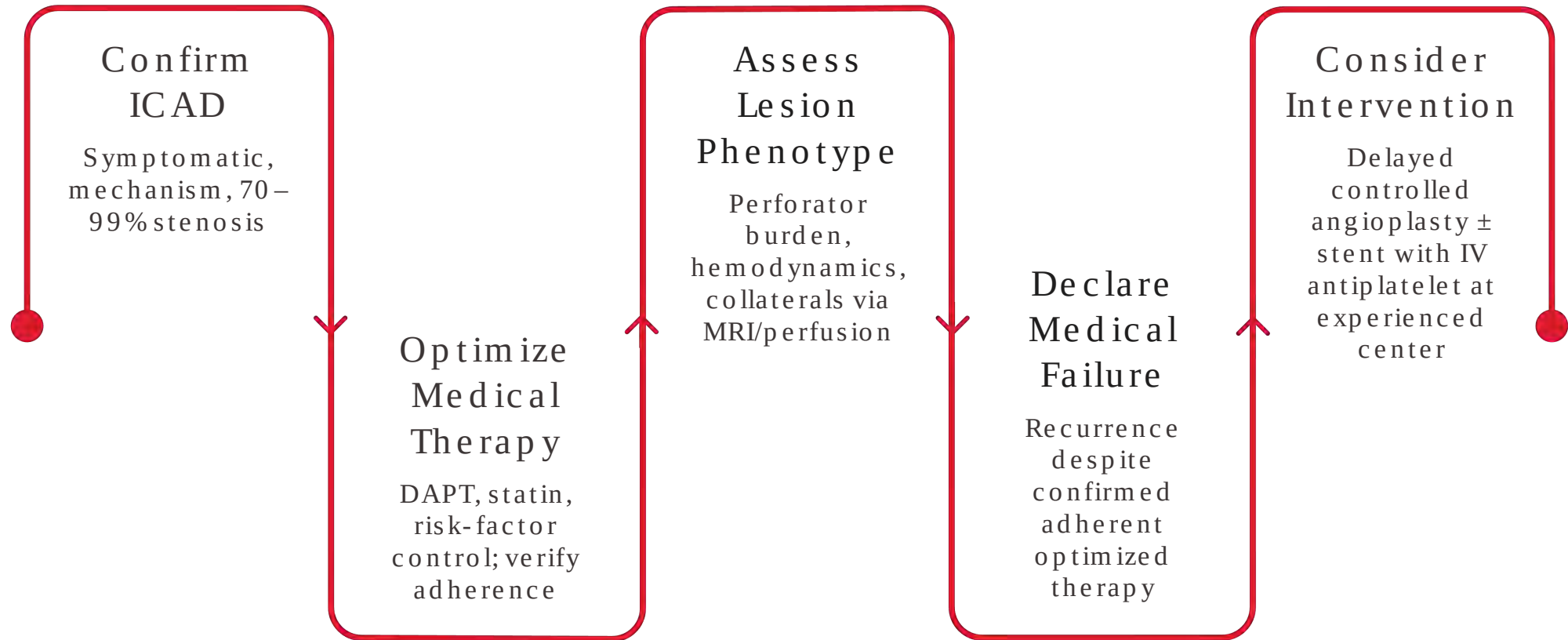
The basilar artery is the highest-risk ICAD target. Bilateral pontine perforators are unforgiving — undersizing is not a compromise, it is the strategy. Angioplasty alone is often the correct endpoint.



A Modern ICAD Algorithm

A structured, sequential decision framework
refractory, high-risk ICAD.

—applied consistently—reduces inappropriate intervention and optimizes outcomes for patients with truly



At every decision point, the burden of proof for proceeding to intervention rests with the clinical team. Shared decision-making with the patient — including a frank discussion of procedural risk and the absence of level-1 efficacy data — is mandatory.

ICAD Reloaded: What Should We Remember?

Medical Therapy Remains First-Line

DAPT + high-intensity statin + aggressive risk-factor control is the non-negotiable foundation. No endovascular discussion is valid until true adherence is confirmed and sustained.

Selection, Timing & Technique Define Success

The catastrophic outcomes of early trials reflected wrong patients, wrong timing, and early-era technique—not an indictment of the concept. WEAVE and WOVEN proved that better selection dramatically changes the risk profile.

Phenotype-Driven Decision-Making

Embolic lesions, flow-limiting lesions, and perforator-dominant lesions each demand different strategies. Vessel-wall MRI and perfusion imaging are not optional—they define the mechanism that guides therapy.

Cangrelor & Tirofiban Expand Control

IV antiplatelet pharmacology has transformed periprocedural platelet management. These are not interchangeable—cangrelor for planned stenting, tirofiban for thrombotic rescue. Neither replaces meticulous technique.

Goal: Perfusion, Not Perfection

Residual stenosis of 30–50% is often the safest, most appropriate endpoint. Forcing anatomic perfection in a perforator-adjacent stenosis trades a radiologic result for a clinical catastrophe.

"ICAD Reloaded is not about abandoning the lessons of SAMMPRIS—it is about applying them precisely enough to know which patients deserve a second chance at intervention, and then executing that intervention with every advantage modern technique provides."

Thank You

Questions & Discussion

Srihari Sundararajan, MD

University Radiology Group & Rutgers Robert Wood Johnson
Medical School

Chief of Interventional Neuroradiology | Assistant Professor of
Radiology

srihari.sundararajan@univrad.com

ICAD Reloaded — Rutgers RWJMS Stroke/NCC Symposium

Key References

1. Chimowitz MI et al. Warfarin - Aspirin for Symptomatic Intracranial Disease (WASID). NEJM 2005;352:1305 -16.
2. Chimowitz MI et al. Stenting vs. Aggressive Medical Therapy for Intracranial Stenosis (SAMMPRIS). NEJM 2011;365:993 -1003.
3. Derdeyn CP et al. SAMMPRIS Final Results. Lancet 2014;383:333 -41.
4. Zaidat OO et al. VISSIT Trial. JAMA 2015;313:1240 -8.
5. Alexander MJ et al. WEAVE Trial. Stroke 2019;50:889 -94.
6. Liebeskind DS et al. WOVEN Registry. Stroke 2020;51:1546 -51.
7. Gao P et al. CASSISS Trial. JAMA 2022;328:534 -42.
8. Mossa - Basha M et al. ASNR Expert Consensus: Intracranial Vessel Wall MRI. AJNR 2017;38:218 -29.
9. Sanchez S et al. Comprehensive Imaging Analysis of Intracranial Atherosclerosis. JNIS 2024.
10. Yang X et al. HR - MR Follow - Up of Intracranial Steno - Occlusive Disease. Front Neurol 2021;12:706645.
11. Capirossi C et al. Supracaloid ICA Stenosis: Self - Expanding Stent. Interv Neuroradiol 2022.
12. Xiao J et al. Gateway Balloon + Neuroform Atlas Stenting for Symptomatic ICAS. Front Neurol 2025;17:1854000.
13. Rapid Medical. Tigertriever 13/17/21/25 — Radial Force Data. Test Report Numbers: TF - GE- 1436, TF- GE- 1493, TF- IKh- 1479, TF- IKh- 1400, TF- IKh- 1481, TF- ANTR- 1529, TF- GE- 1529, TF- YR13- 1210.
14. Jiang S et al. Paclitaxel DCB Angioplasty for ICAD via HR - VWI. Interv Neuroradiol 2024.
15. Holmstedt CA, Turan TN, Chimowitz MI. Atherosclerotic Intracranial Arterial Stenosis. Lancet Neurol 2013;12:1106-14.

RUTGERS

Robert Wood Johnson
Medical School

RWJBarnabas
HEALTH

UNIVERSITY
RADIOLOGY 