# APPLIED

# NEUROVASCULAR

# ANATOMY

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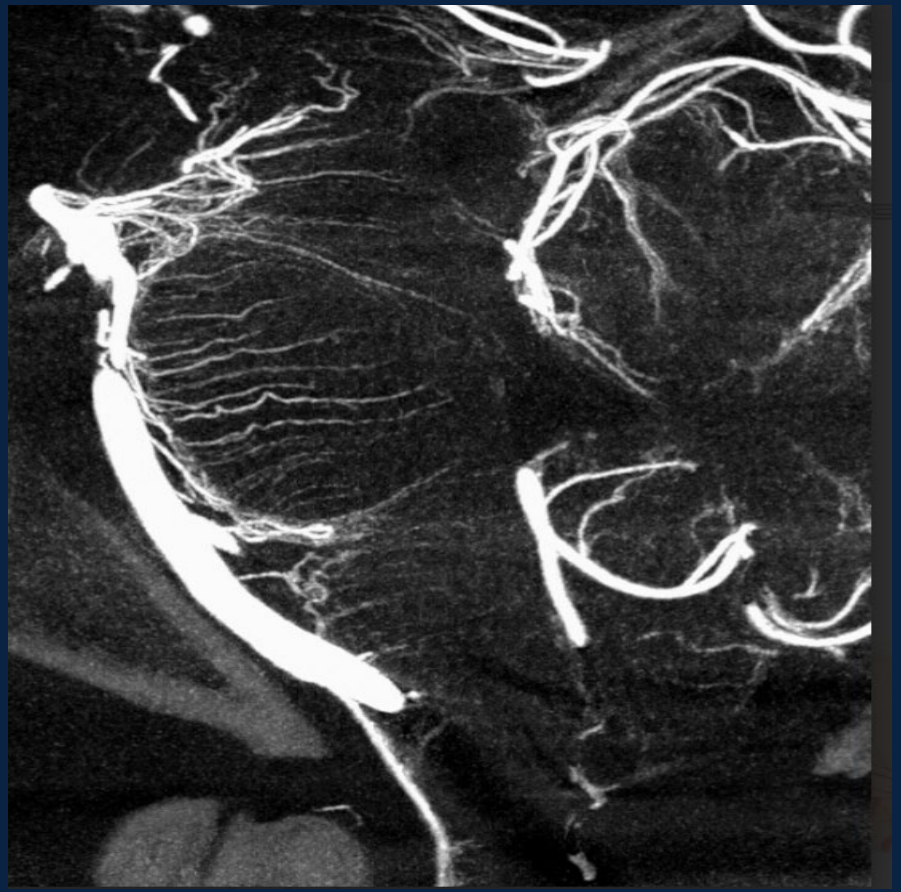

# Disclosures

No financial disclosures. No conflicts of interest.

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# Learning Objectives

01 Apply neurovascular anatomy to clinical interventional situations

02 Understand the "interconnected network" model of cerebrovascular anatomy

03 Identify and manage EC-IC dangerous anastomoses — especially meningo-ophthalmic spectrum

04 Utilize collateral pathways in stroke assessment and treatment planning

# The Cervical Scaffold & Dangerous Anastomoses

## Supra-Aortic Trunks

### Bovine Arch

LCA shares trunk with Innominate — most common aortic arch variant (~20%)

### Direct Vertebral Origin

VA arising directly from aorta — rare but highly significant for catheterization planning

### Subclavian Steal

Proximal subclavian stenosis → retrograde vertebral flow on exertion

## Network Principle

Vessels must be seen as part of a network, not isolated entities. The cervical scaffold is the foundation — variant anatomy here propagates through every downstream territory.

## Key Catheterization Pitfalls

- Bovine arch: single sheath controls both CCA & subclavian
- Vertebral from arch: requires different approach angle
- Always map arch type before selective catheterization

# The Carotid Siphon

| Segment | Key Features | Clinical Implication |
|---|---|---|
| Cervical | Begins at bifurcation; atherosclerosis common | Plaque characterization by CTA/MRI before stenting |
| Petrous | Through carotid canal; tortuous; sympathetics | Pseudoaneurysm from temporal bone trauma |
| Cavernous | S-shaped; venous plexus surrounds; CN III-VI nearby | Rupture → CCF (no SAH); aneurysms treated with flow diversion |
| Clinoid | Extradural transitional zone; distal dural ring | Critical: below ring = no SAH risk; above = SAH risk |
| Supraclinoid | Ophthalmic, PCOM, AChA origins; subarachnoid | Aneurysm rupture here causes SAH — verify ring position on DSA |

💡 **Clinical Pearl:** The distal dural ring is the critical landmark — rupture above = SAH; below = no SAH. Always verify on DSA/3D angio before treating cavernous vs. supraclinoid aneurysms.

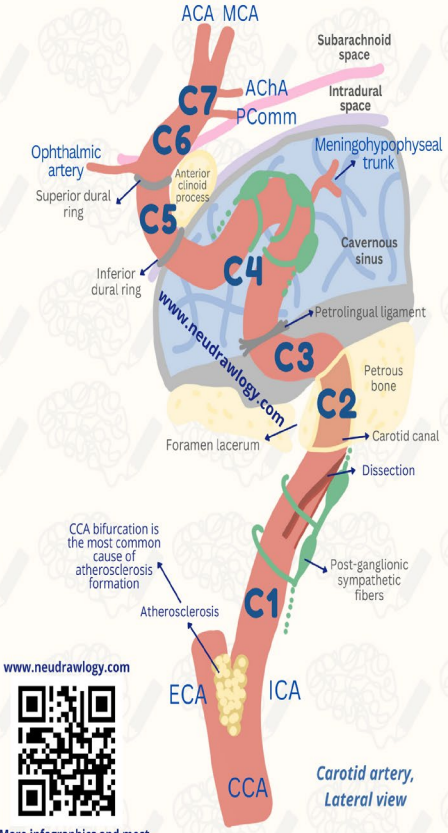
Created by: Gabriela Figueiredo Pucci, MD and Lucas Rios Rocha, MD

# Internal Carotid Artery Segments

@gabifpucci / @neudrawlogy and @LucasRochaMD

## Anatomical and Clinical correlations according to the Bouthellier Classification

www.neudrawlogy.com

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More infographics and most updated version of this one here!

| Segment | Anatomy | Clinical correlation |
|---|---|---|
| C1: Cervical | 2-3cm distal to the bifurcation: the most frequent site of dissection | Horner's syndrome (unilateral miosis, ptosis) - damage of post-ganglionic sympathetic pupillary fibers *See Horner's syndrome infographic |
| C2: Petrous | Carotid enters the petrous bone through the carotid canal, becoming intracranial | Atherosclerotic disease can present with auditive manifestations due to proximity to middle year structures |
| C3: Lacerum | Runs close to the lacerum foramen, not passing through it | Skull base fractures can injury this segment resulting in life-threatening hemorrhage |
| C4: Cavernous | Close relationship to CN III, IV, V1, V2 and VI *See Tolosa-Hunt Syndrome infographic | Aneurysms arising here can extend superiorly and enter the subarachnoid space (cave aneurysms) |
| C5: Clinoid | Intradural portion | Clinoidal meningiomas are in close relationship to this segment, can cause vision loss due to CN II compression |
| C6: Ophthalmic | Gives origin to the first branch after entering the skull, the ophthalmic artery | Ophthalmic artery's first branch (retinal artery) can occlude causing amaurosis fugax |
| C7: Communicating | Gives origin to the PComm segment | The junction between the Pcomm and the C7/Communicating segment : the most frequent site for saccular cerebral aneurysms |

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## ICA Segment Guide

- C1 Cervical
- C2 Petrous
- C3 Lacerum
- C4 Cavernous
- C5 Clinoid
- C6 Ophthalmic
- C7 Communicating

# Anterior Cerebral Artery (ACA)

## Segmental Anatomy

**A1** — Recurrent Artery of Heubner → caudate head, anterior internal capsule

**A2** — Medial frontal/parietal (leg area). Occlusion → contralateral leg weakness, abulia, incontinence

**A3+** — Callosomarginal and pericallosal — cortical supply to parasagittal cortex

## Circle of Willis Variants

### Fetal PCA (20–25%)

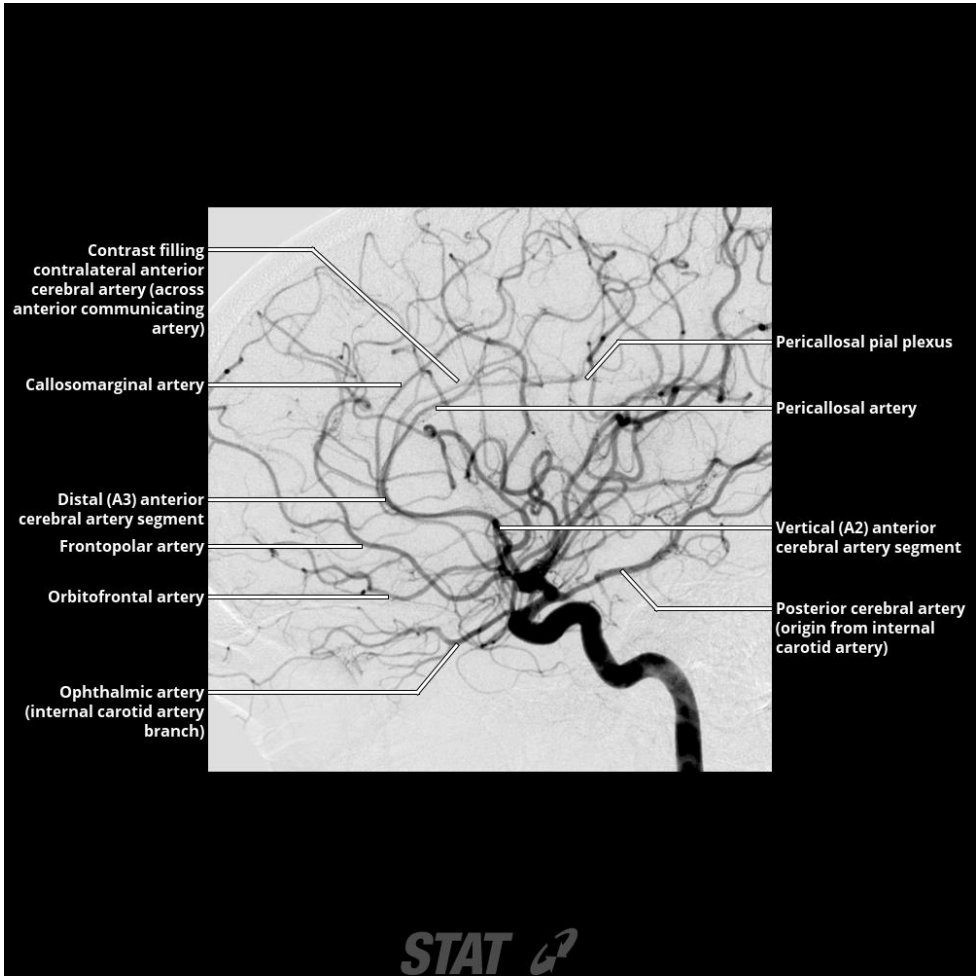
PCA arises from ICA not basilar. Leptomeningeal collaterals between carotid and VB systems fail to develop → increased risk of posterior territory ischemia with ICA occlusion.

### Azygos ACA

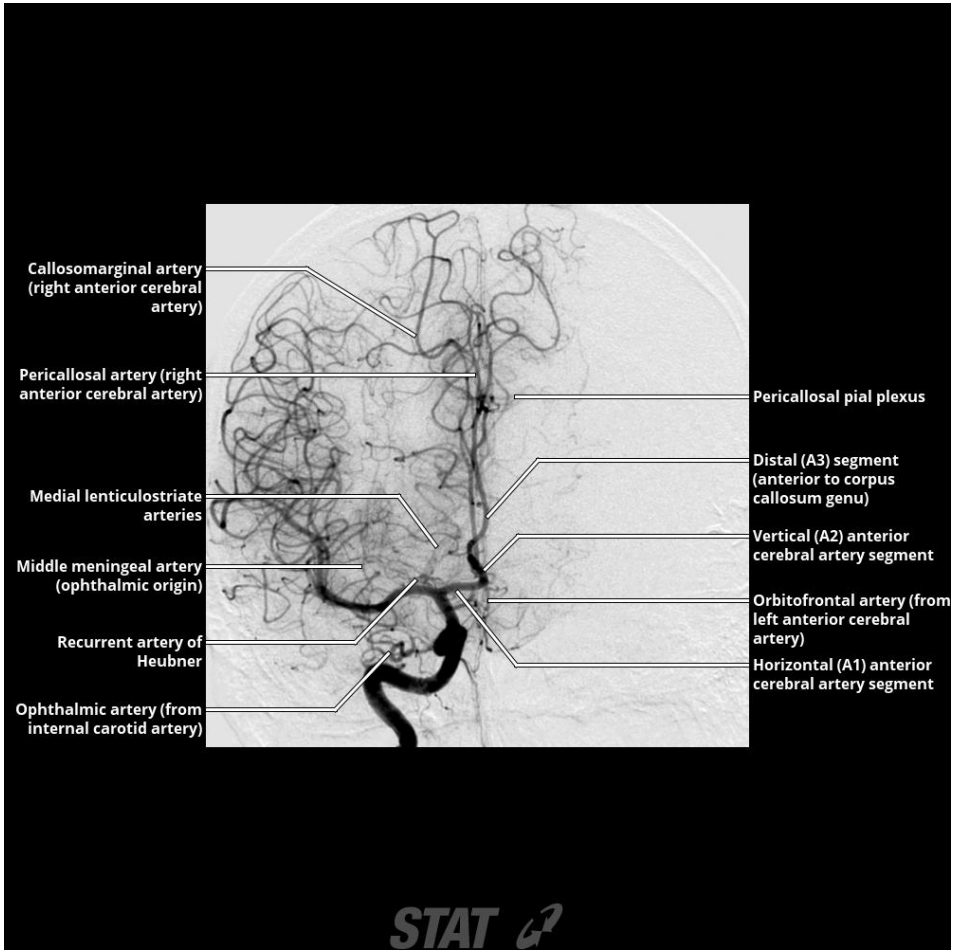
Single trunk supplies both medial hemispheres. Occlusion → bilateral paramedian deficits. Critical to identify before ACA aneurysm treatment.

### Hypoplastic A1

Common variant. Angiography: will not see venous drainage of contralateral ACA territory. Affects collateral modeling in stroke.




Digital subtraction internal carotid angiogram, lateral view, midarterial phase, shows the ACA and its major cortical branches.




Digital subtraction right internal carotid angiogram, AP view, midarterial phase, shows the ACA and its branches. Both distal ACAs fill from this injection because contrast has refluxed across the anterior communicating artery (which is not well seen on this projection). Note the ACAs are generally positioned in the midline,

# Angiographic Anatomy — ICA / ACA / MCA

# Middle Cerebral Artery (MCA)

*Phylogeny: The MCA is a "new" vessel, born from the anterior division of the primitive ICA to supply the expanding frontoparietal cortex. (neuroangio.org)*

## M1 Sphenoidal

Lenticulostriate arteries (basal ganglia & internal capsule). Occlusion → dense contralateral hemiplegia (face/arm > leg). Perforators are end-arteries.

## M3 Opercular

Opercular segments over Sylvian fissure. Superior/inferior divisions supply motor/language cortex.

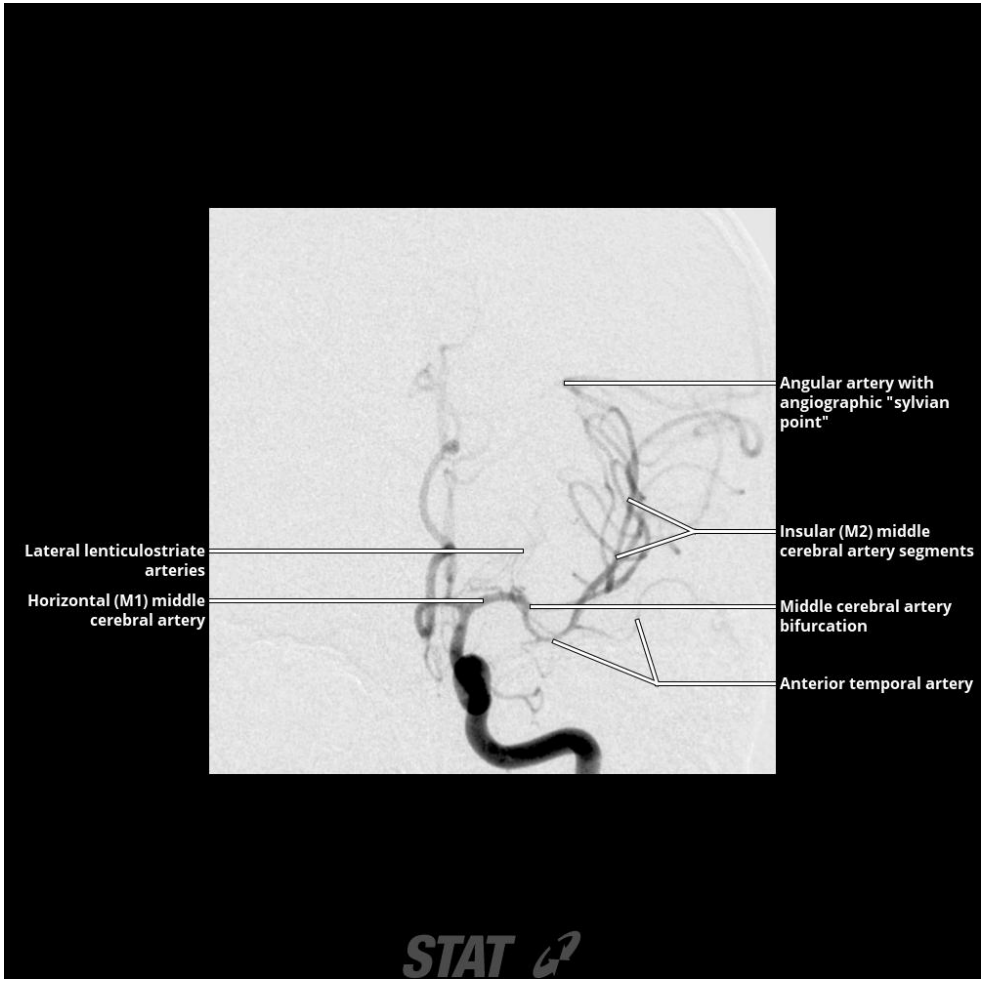
## M2 Insular

Common site for embolic occlusion. Branches fan onto insular cortex. Key landmark for thrombectomy catheter positioning.

## M4 Cortical

Terminal cortical branches. Leptomeningeal anastomoses here with ACA (superior) and PCA (posterior).

💡 **Clinical Pearl:** Good leptomeningeal collaterals from ACA/PCA can retrograde fill MCA branches, preserving tissue until recanalization. Collateral quality determines infarct volume.




Three AP views of left internal carotid angiogram illustrate normal MCA angiographic anatomy. Only the horizontal (M1) and insular (M2) segments are filled out on this early arterial phase image. The MCA bifurcates within 1 cm of its origin, a so-called "early bifurcating" MCA. The angiographic "sylvian point" is the highest,


Midarterial phase demonstrates the insular (M2) and opercular (M3) MCA segments as well as early filling of some cortical (M4) MCA branches.


Late arterial phase shows contrast has been washed out of the more proximal (M1, M2) MCA segments. The distal cortical (M4) MCA branches are now completely opacified. Note the "brain stain" caused by opacification of small branches within the basal ganglia as well as the cortex.

# Posterior Cerebral Artery (PCA)

*Embryology : The PCA originally belongs to the anterior carotid circulation. Transfer to vertebrobasilar system is necessitated by the large frontoparietal territory the carotid system must support in humans. The "Fetal PCA" variant (20–25%) simply reflects persistence of this early arrangement.*

### P1 (Pre-communicating)

From basilar bifurcation to PCOM. Perforators to thalamus, midbrain. In Fetal PCA: this segment is hypoplastic/absent.

### P2 (Ambient)

Ambient cistern around brainstem. Posterior choroidal arteries arise here (thalamic supply — critical for consciousness).

### P3/P4 (Calcarine)

Primary visual cortex supply. Occlusion → homonymous hemianopia (macular sparing from MCA collaterals possible).

💡 **Clinical Pearl:** In Fetal PCA: ICA occlusion leads to PCA territory infarction (posterior territory). Leptomeningeal collaterals between carotid and VB systems fail to develop — uniquely vulnerable.

# Posterior Fossa — The Vertebrobasilar System

## PICA

**Territory: Lateral medulla + Inferior cerebellum**

Wallenberg Syndrome: ipsilateral face + contralateral body sensory loss, dysphagia, Horner's. Extent varies by perforator territory.

## AICA

**Territory: Pons + Middle cerebellar peduncle**

Intimately related to CN VII/VIII; vascular loop → hemifacial spasm. AICA-PICA balance: continuous Gaussian distribution.

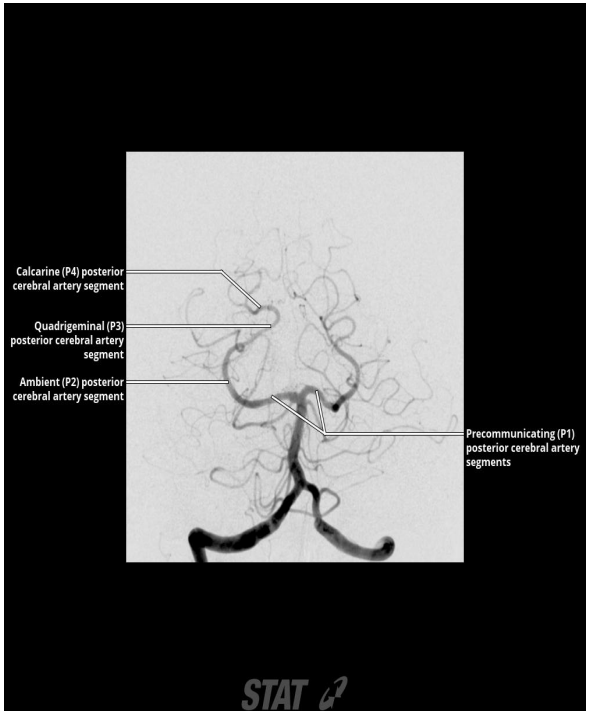
## SCA

**Territory: Superior cerebellum + Tectum**

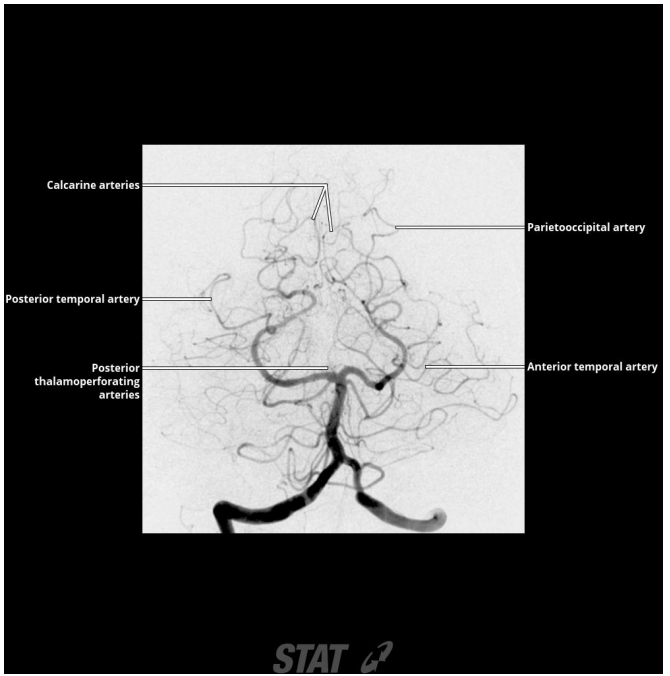
Duplicated SCAs common — simply persistence of adjacent transverse mesencephalic arteries (neuroangio.org embryology).

*Embryology: Unlike PICA (cervical origin), AICA is a true cerebellar/brainstem artery from the longitudinal neural system.*

# Posterior Circulation Angiogram — Vertebrobasilar

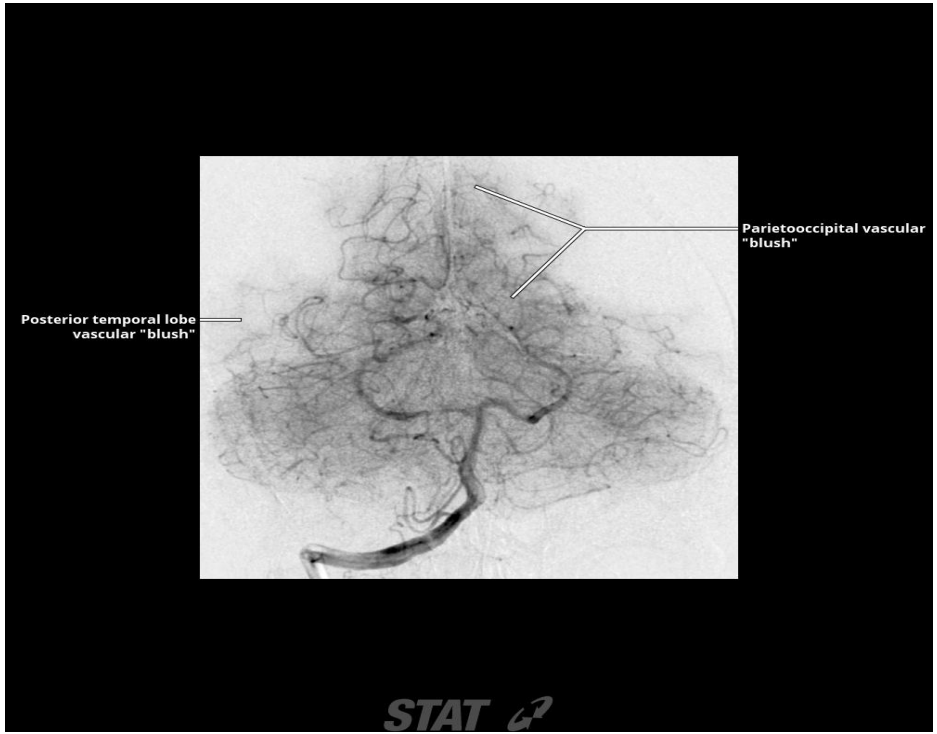



Series of 3 AP views of a vertebrobasilar angiogram depict the PCA segments and their branches. The precommunicating (P1) segment is best seen in this projection. The PCAs sweep laterally and then posterosuperiorly around the midbrain.



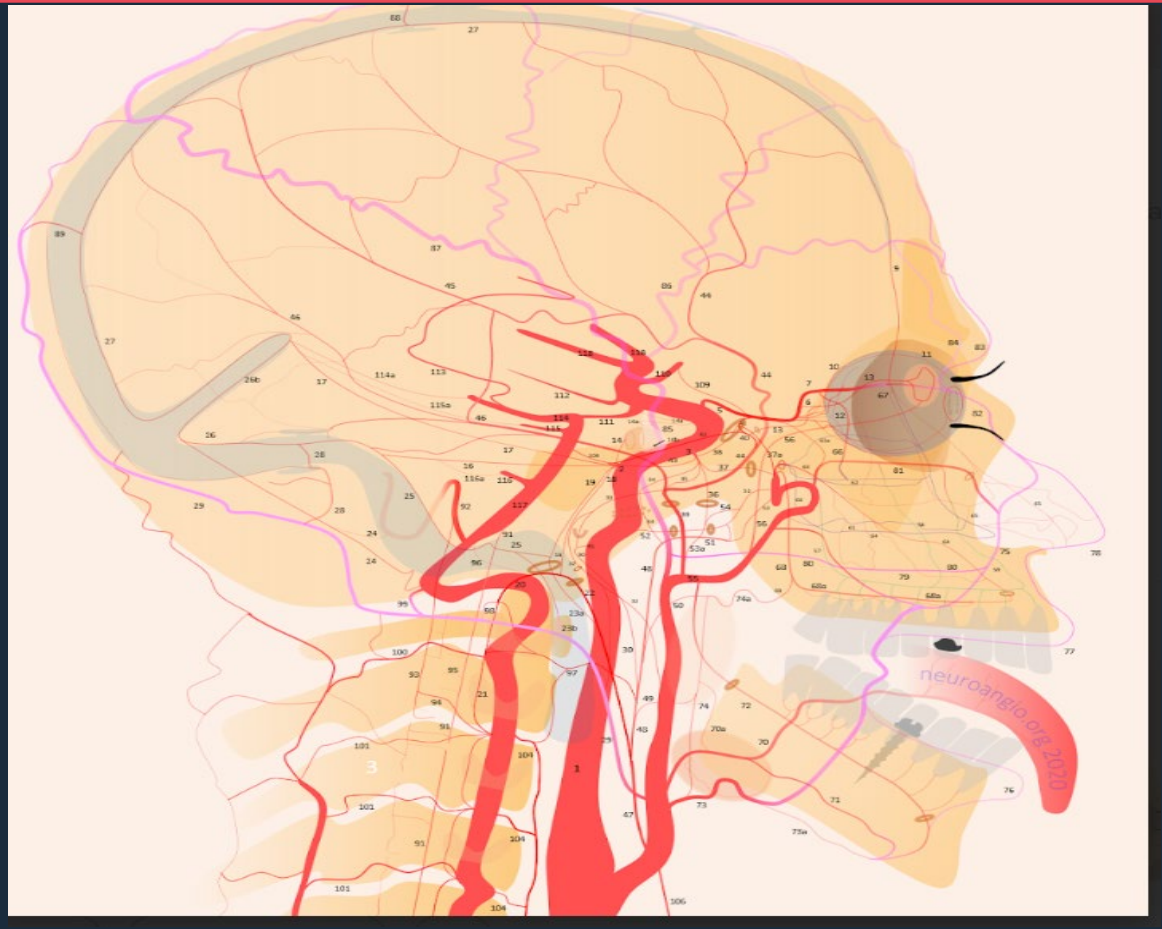

Midarterial phase shows several of the cortical PCA branches especially well. In this view, anterior and posterior temporal arteries often overlap somewhat. In this projection, the posterior thalamoperforating arteries are seen as a faint vascular blush lying just above the terminal basilar artery bifurcation.

# Vertebrobasilar — Late Phase




Late arterial phase shows the vascular blush of the PCA supply to the medial parietal and occipital lobes as well as the temporal lobes. The unopacified vertical "filling defect" is the dura of the falx cerebri that separates the 2 cerebral hemispheres.

# EC-IC Collateral Circulation — Network Model




## Key Collateral Routes

- Ophthalmic (ECA→ICA)
- ILT — Inferolateral Trunk
- PCOM (ICA↔VB)
- ACOM (bilateral ICA)
- Leptomeningeal (MCA↔ACA)
- Transosseous / emissary

*"Anatomic presence ≠ functional flow" — neuroangio.org*

# The "Dangerous Anastomoses" — Meningo-ophthalmic Spectrum

*These EC-IC connections are beautiful but treacherous during endovascular treatment. ( "What you don't see leads to when patient can't see.")*

1

## Meningo-ophthalmic Variant

Ophthalmic artery arises partially/fully from the MMA. Proximal MMA particle embolization → particles into retinal artery → BLINDNESS. The central retinal artery is ~160µm — a true end-artery with no collaterals.

*Most famous dangerous anastomosis*

2

## Deep Recurrent Meningeal Artery

Connects ophthalmic artery (orbit) to MMA (intracranial) via the sphenoid ridge. Think: railroad with stops at lacrimal, then ophthalmic, then ILT-ICA. Both dangerous AND useful for reconstitution.

*neuroangio.org: Sphenoid ridge artery is the key connection*

3

## Foramen Rotundum Artery

Connection between internal maxillary artery and cavernous ICA. Embolization endangers CN V2 (maxillary nerve). ILT also has a proximal branch toward carotid canal — always watch it.

💡 **Clinical Pearl:** Always identify the orbital blush before MMA embolization. Good angiographic technique + knowing anatomy = how you stay safe.

# The Four Layers of the Neurovascular System

*A Key to Embolization*

**1**

**Extracranial → Cranial Base**

e.g. MMA, IMAX, STA (ECA territory)

**2**

**Dural / Calvarial Network**

e.g. Meningeal arteries, diploic veins

**3**

**Leptomeningeal Arterial Net**

e.g. MCA ↔ ACA pial collaterals

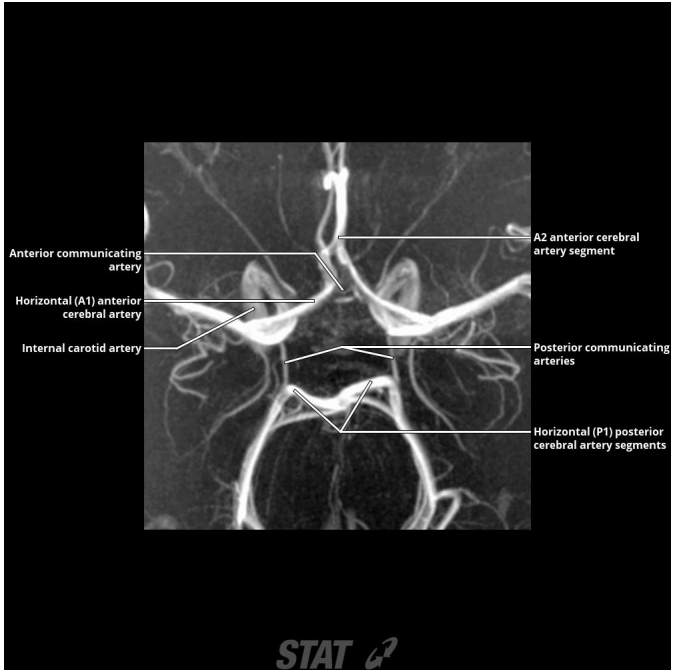
**4**

**Extracranial Venous / Emissary**

e.g. Condylar, mastoid, ophthalmic veins

💡 **Clinical Pearl:** Embolization for chronic SDH (MMA) must consider ALL four layers to prevent non-target embolization through dangerous anastomoses — especially the meningo-ophthalmic spectrum.

# COW




Submentovertex view from a high-resolution MR angiogram obtained at 7T is depicted for comparison with the previous graphic and 3D CTA. In this case, all segments of the COW are present, a so-called "balanced" COW in which no segment is absent or hypoplastic.

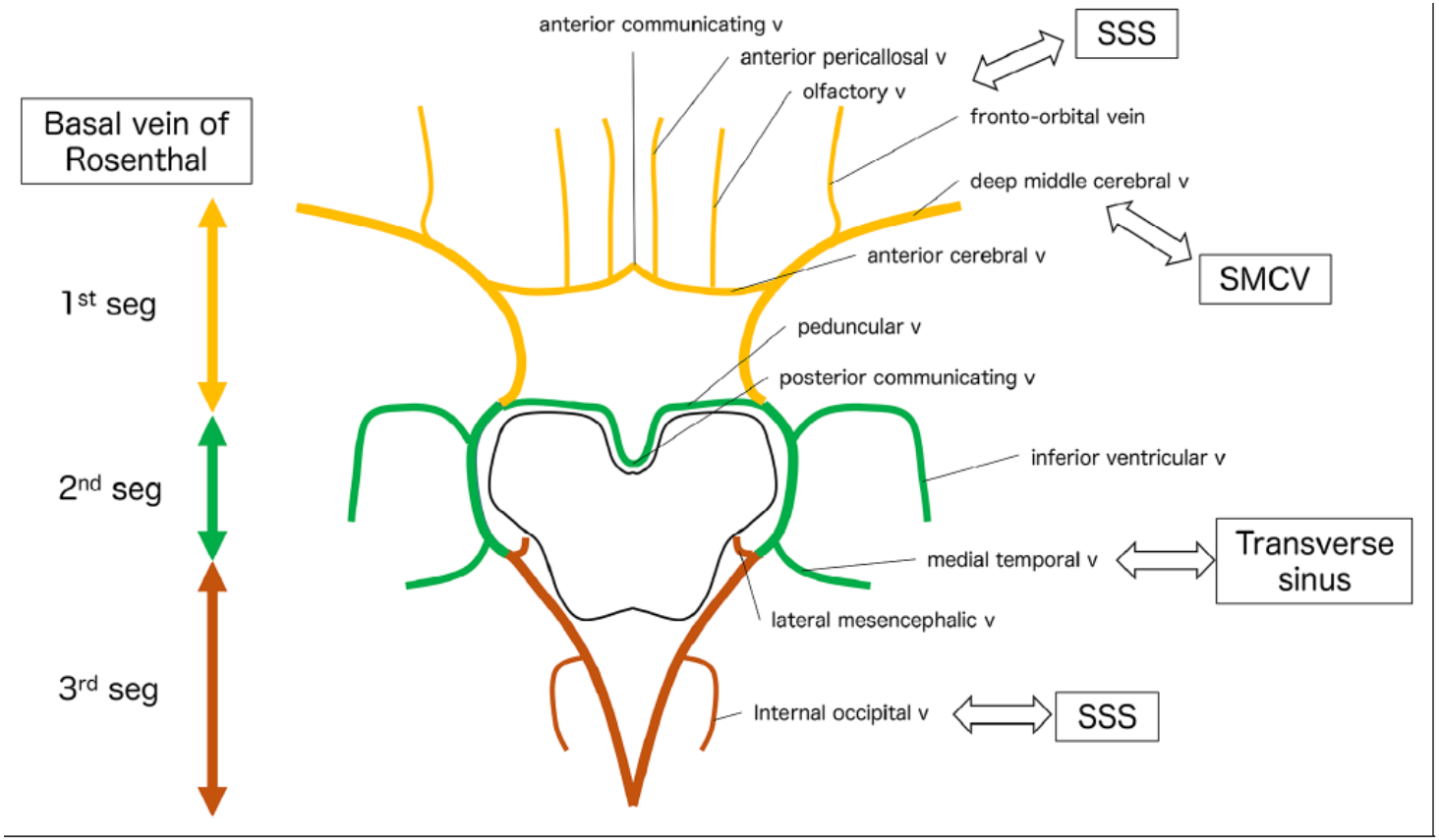
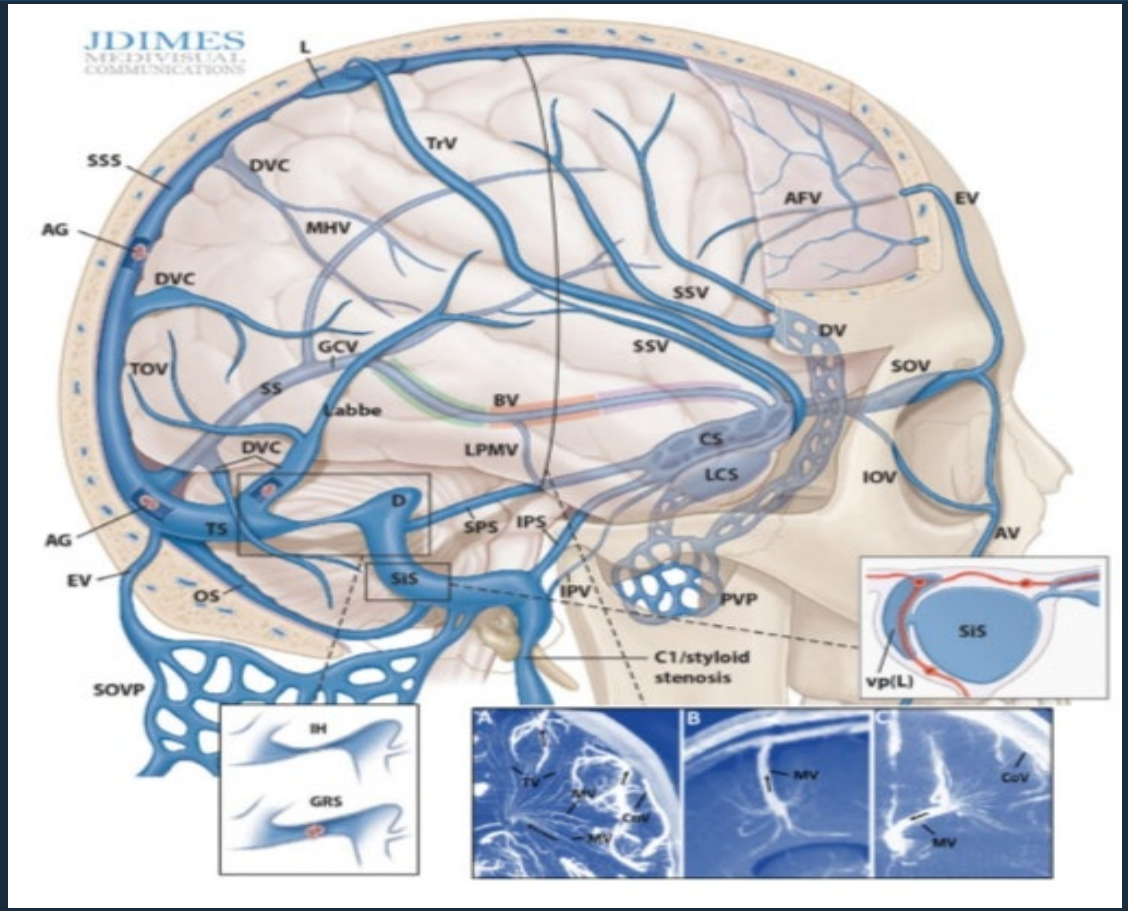



**Figure 3. Venous circle of Trolard and the 3 segments of the basal vein of Rosenthal.** There is a venous circle in the Trolard at the base of the brain. Three segments and their tributaries in the basal vein are shown on the left side using a color code. Possible collateral connections are shown on the right with 2-headed arrows. SMCV indicates superficial middle cerebral vein; and SSS, superior sagittal sinus.

# Venous Anatomy — Dural Sinus Overview




## River Analogy

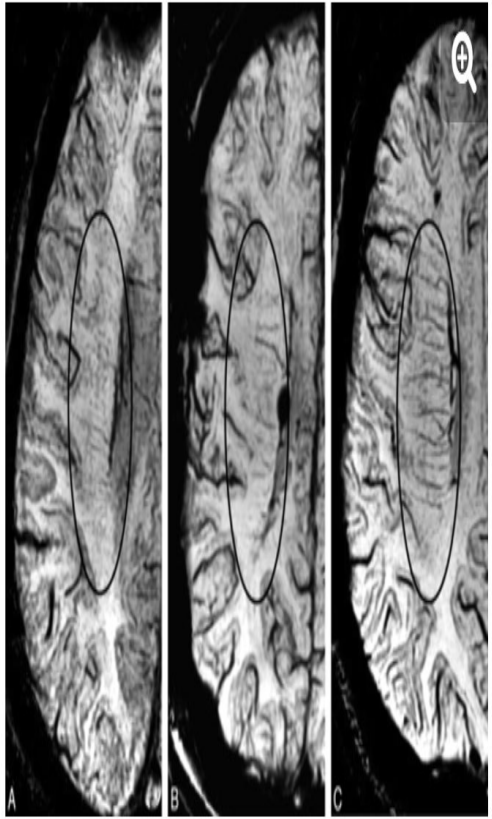
- No valves — bidirectional flow possible
- Develop AFTER arteries — more variable
- Dura = tabula rasa for venous drainage
- Dominant transverse sinus critical for surgical planning
- SSS hypoplasia anteriorly: common variant, not thrombosis

# Venous Anatomy — The "River" Metaphor

*Core Rule: Veins are like rivers. Do not read the book on where classic sinuses "should" be.*

| | | |
|---|---|---|
| **Superior Sagittal Sinus** | Highly variable anterior extent. Often replaced by cavernous sinus drainage of frontal lobes. | Venous thrombosis: CT bone for hypoplastic foramen vs. pathologic clot |
| **Transverse/Sigmoid** | Asymmetric — left typically hypoplastic. Size depends on tributaries (e.g., Labbé). | Dominant side absence = stroke risk during sacrifice |
| **Vein of Labbé** | Drains temporal lobe to transverse/sigmoid. Highly variable. | SACRIFICE → hemorrhagic temporal infarct — avoid! |
| **Cavernous Sinus** | Multiple compartments; may be bypassed by laterocavernous sinus. | Access route for CCF treatment; multiple cranial nerves at risk |

# Why Functional Venous Anatomy & Collaterals Matter




## Unique Features

No valves, develop later, variable flow direction — fundamentally different from arteries

## Collateral Networks

Venous collaterals actively participate in normal drainage and compensate for compromised routes

## Interventional Importance

Understanding venous anatomy prevents complications during neurointerventional procedures

## Systems Approach

View veins as interconnected adaptive networks — predict flow redistribution and dangerous anastomoses

# Superficial, Deep, & Basal Venous Organization

## Superficial System

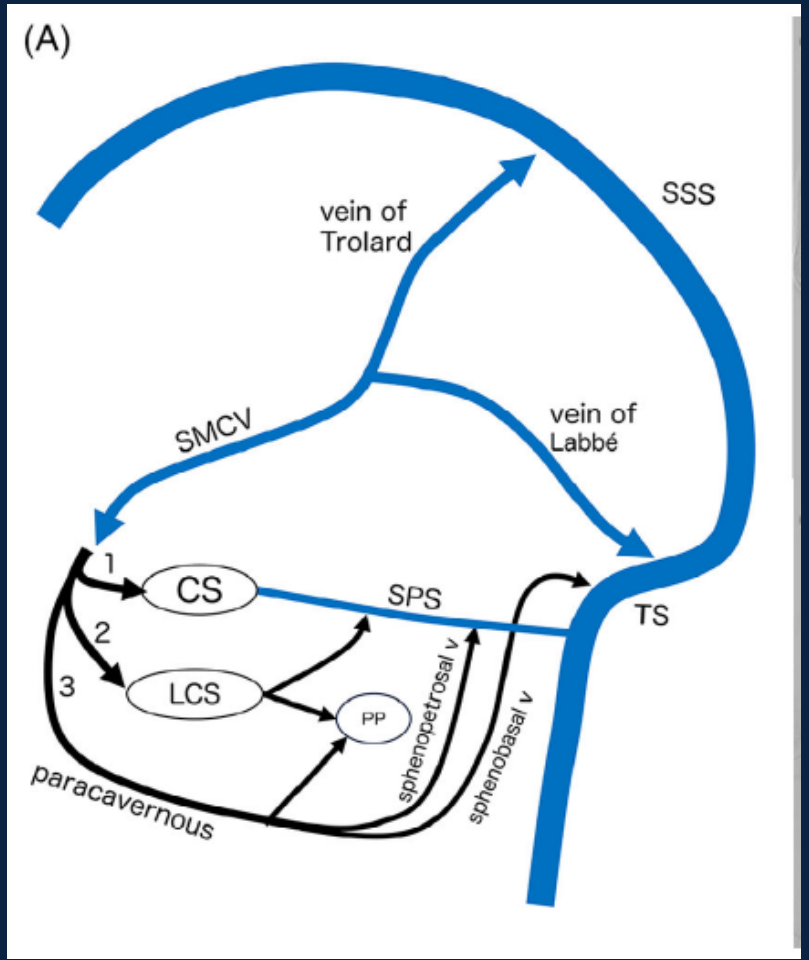
Cortex + subcortical WM → dural sinuses via anastomotic veins (Trolard, Labbé). Highly variable.

## Deep System

Basal ganglia + thalami → Internal Cerebral Veins → Vein of Galen → Straight Sinus.

## Basal Vein of Rosenthal

Links superficial and deep systems. Major collateral channel. Variable anatomy — key "river" junction.



# Major Collateral Routes: Deep & Superficial Systems

Striate Route

**Striate Route**

Subependymal veins → deep middle cerebral & basal veins

Terminal Vein Route

**Terminal Vein Route**

Choroid plexus → internal cerebral & basal veins (Galenic outflow bypass)

Hippocampal/Thalamic

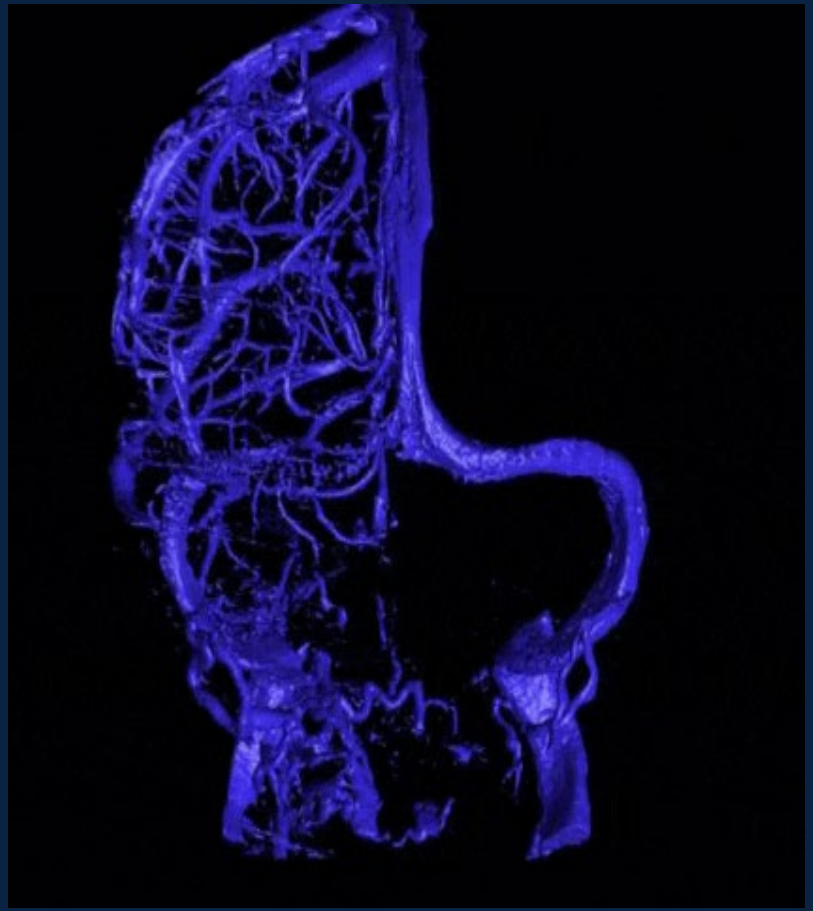
**Hippocampal/Thalamic**

Alternate drainage in chronic outflow compromise

Transcerebral Veins

**Transcerebral Veins**

Subependymal → cortical veins. Enlarge over time (chronic adaptation)



# Brainstem & Cerebellar Drainage Systems

## Infratentorial Venous System

Dense veins in a confined space with extensive anastomoses and high anatomical variation.

### Superior (Galenic)

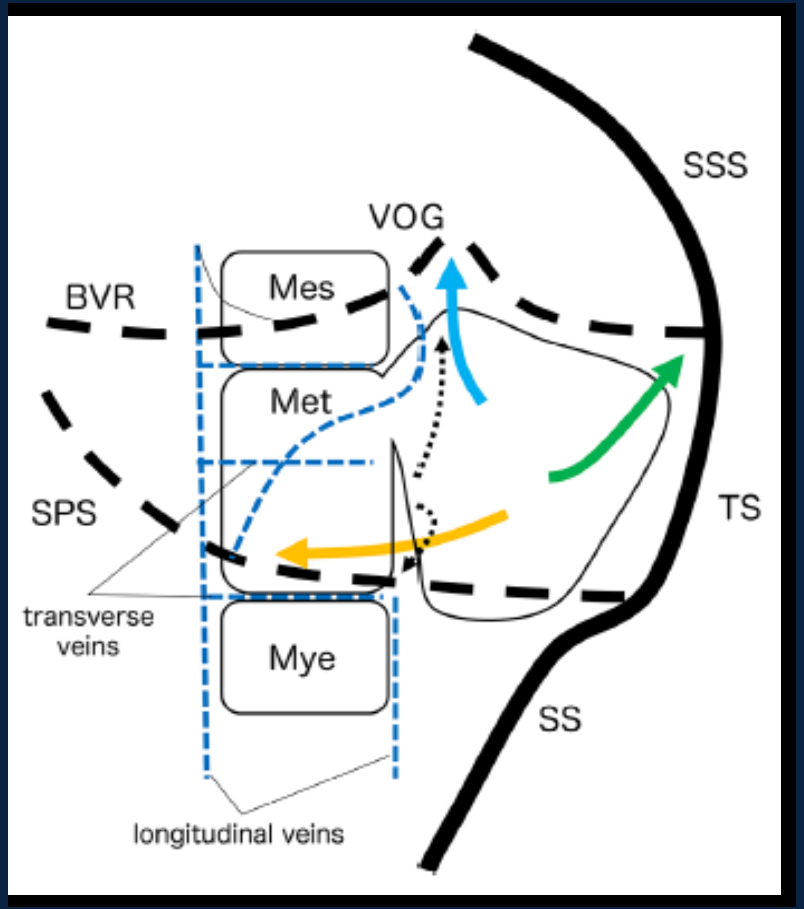
Via vein of Galen — primary outflow for brainstem and cerebellum

### Anterior (Petrosal)

Petrosal vein: major hub; drains to superior petrosal sinus

### Posterior (Tentorial)

Tentorial sinus route; variable; anastomoses with occipital sinus



# Applying Functional Venous Anatomy in Practice

### Diagnosis & Risk

Functional venous anatomy directly affects diagnosis, risk assessment, treatment planning in neurointervention

### Venous Collaterals

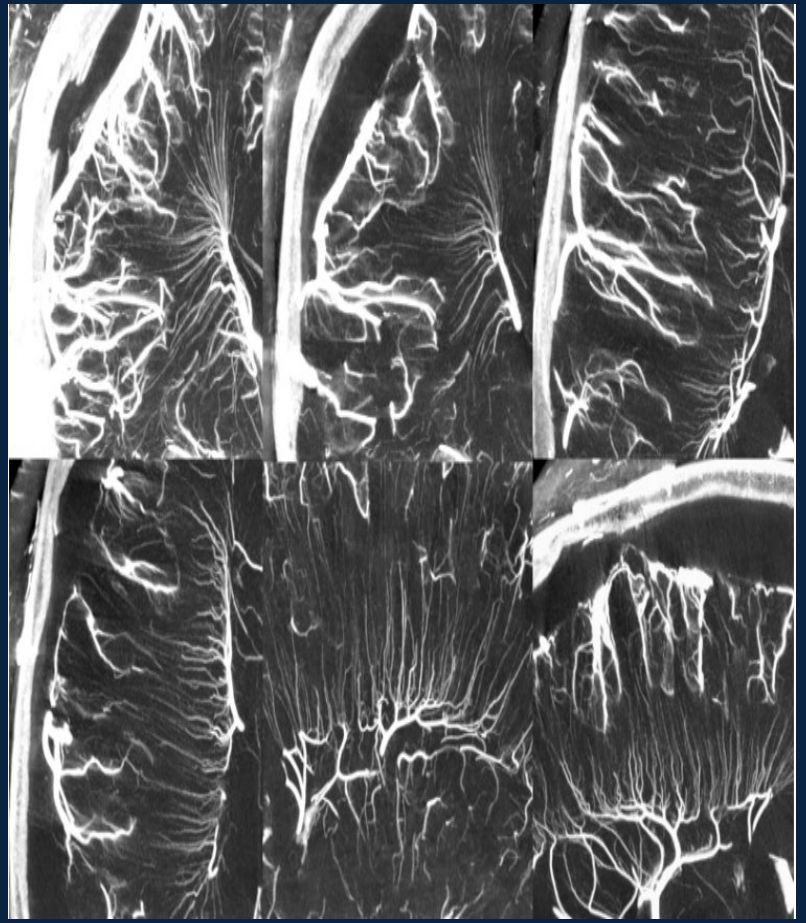
Influence symptom patterns and treatment in AVFs, venous sinus thrombosis, dural AVMs

### Adaptive Networks

Assess venous systems as adaptive networks → predicts tolerance to venous sacrifice

### 3D Imaging

3D angiography + Cone-Beam CT (CBCT) essential for functional venous anatomy interpretation



# Collateral Circulation — Pathophysiology over Anatomy

$$I = V / R$$

Flow = Perfusion Pressure ÷ Vascular Resistance

**Key Insight**

Anatomic presence of a collateral pathway does NOT guarantee functional flow.

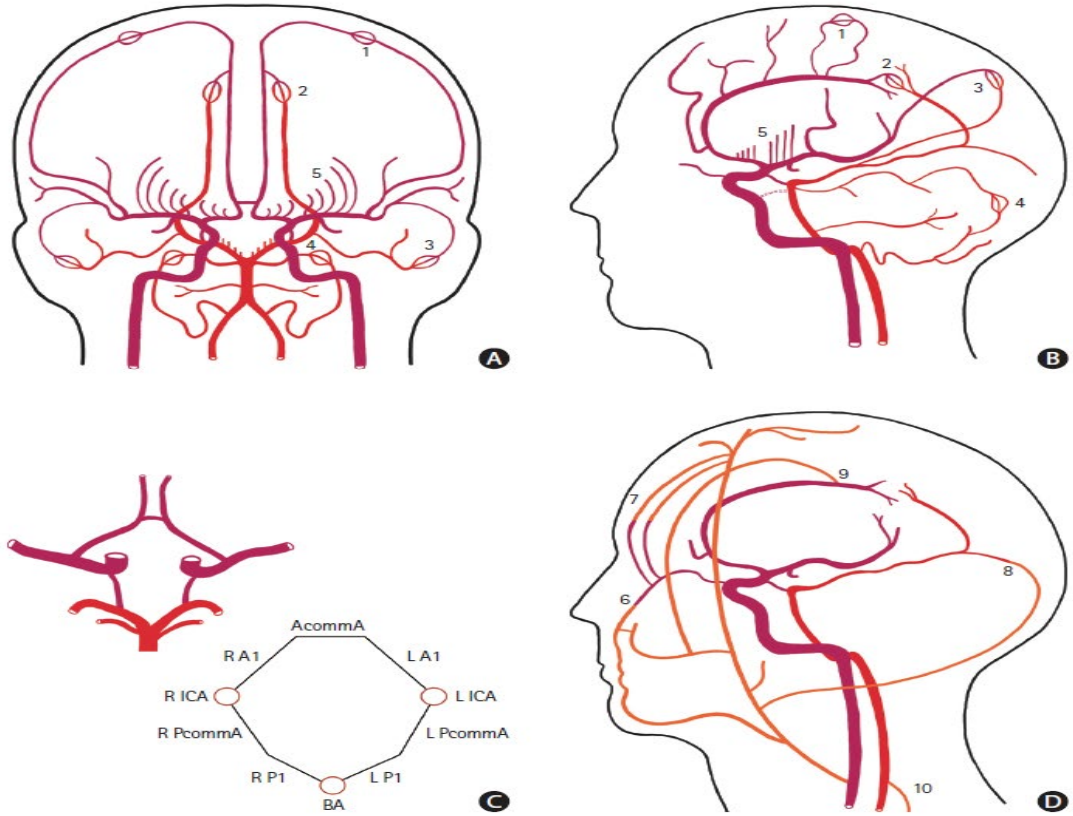
**Perfusion Pressure (V)**

Determined by systemic BP and ICP. Low BP → reduced collateral driving pressure, even with patent anatomy.

**Autoregulatory Reserve (R)**

Young, healthy vessels dilate well (low resistance). Atherosclerotic vessels are non-compliant → resistance stays high despite occlusion.

- Pial Leptomeningeal (MCA ↔ ACA): crucial for limiting infarct size
- Ophthalmic Collateral: reversed flow in ophthalmic artery = severe proximal ICA stenosis
- Intracranial ICA reconstitution via ILT: ECA → ICA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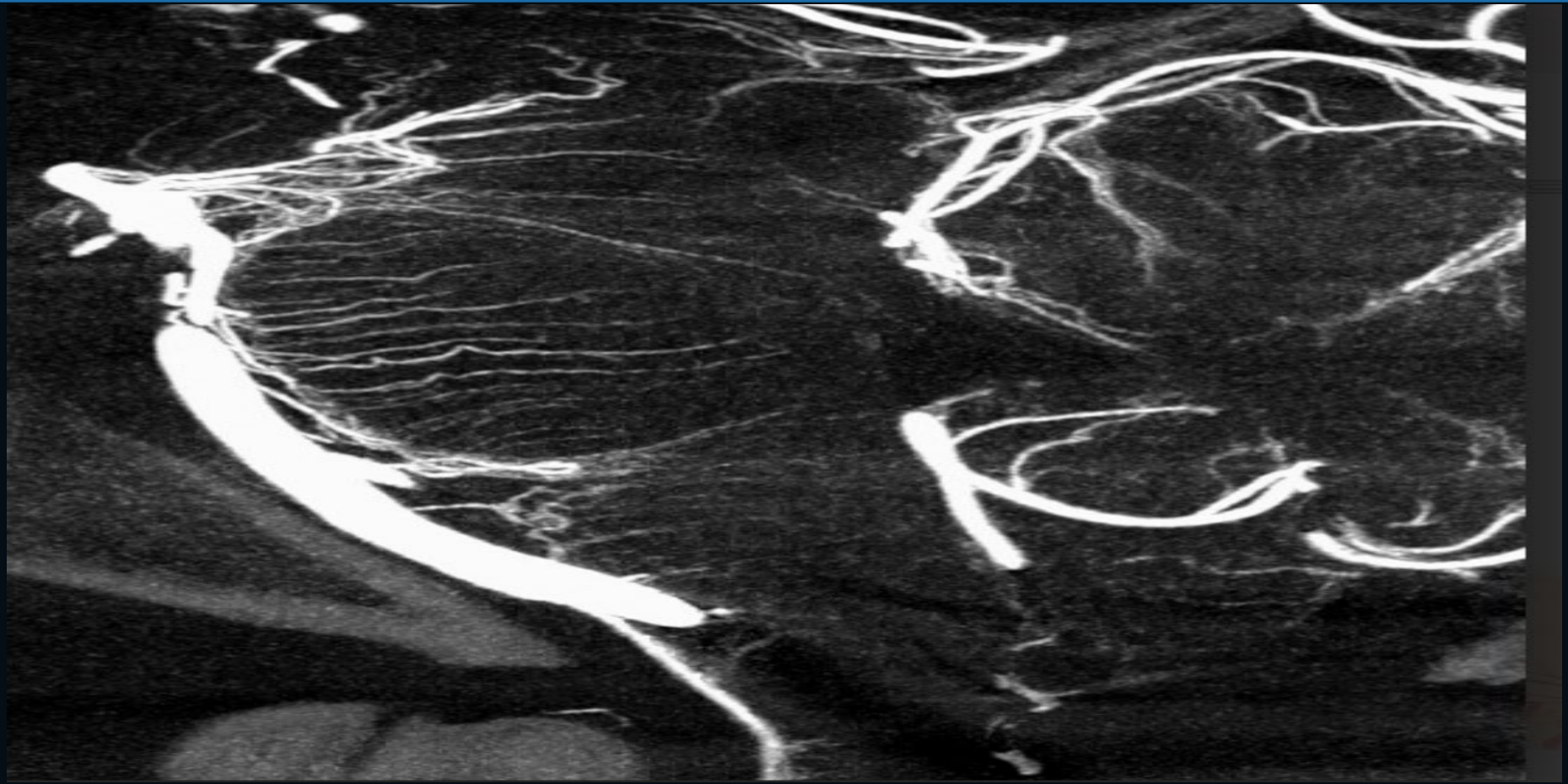

# Take-Home Points

1

### Anatomy is Dynamic

Think lattice, not just circle; veins are rivers. See vessels as interconnected systems, not isolated entities.

2

### Respect Dangerous Anastomoses

Pre-embolization CBCT/DSA must check for meningo-ophthalmic connections. What you don't see leads to when patient can't see.

3

### Stop Memorizing Stroke Syndromes

Understand the lateral medullary perforator territory to predict Wallenberg severity. Know the AICA-PICA balance.

4

### Collaterals are Pathophysiologic

Anatomic presence ≠ functional flow (I = V/R). Young compliant vessels ≠ atherosclerotic non-compliant vessels.

5

### CBCT & Perfusion = Future

Use them to look for dangerous anastomoses and to assess collateral quality — not just core/penumbra selection.