DURAL AV FISTULAE

NEUROSURGERY LECTURE

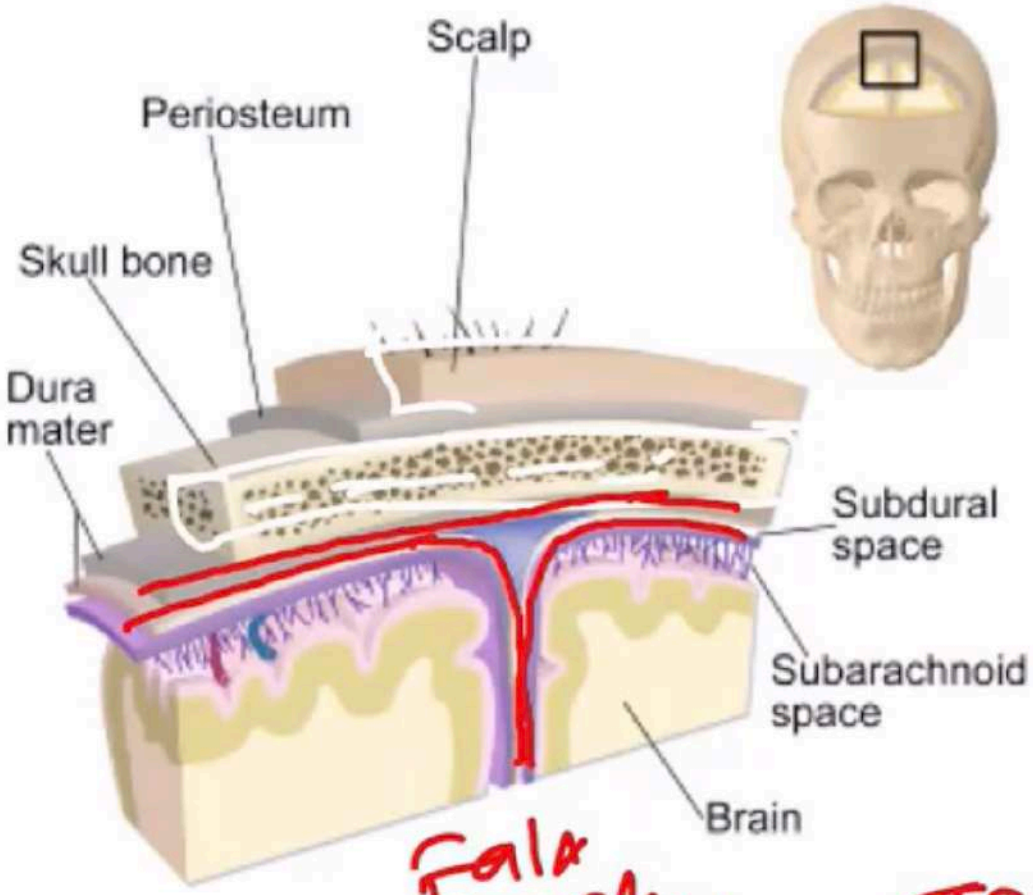
DR HIMANSHU CHAMPANERI

CONSULTANT NEUROSURGEON

MEDANTA HOSPITAL, GURUGRAM

ANATOMY OF DURA MATER

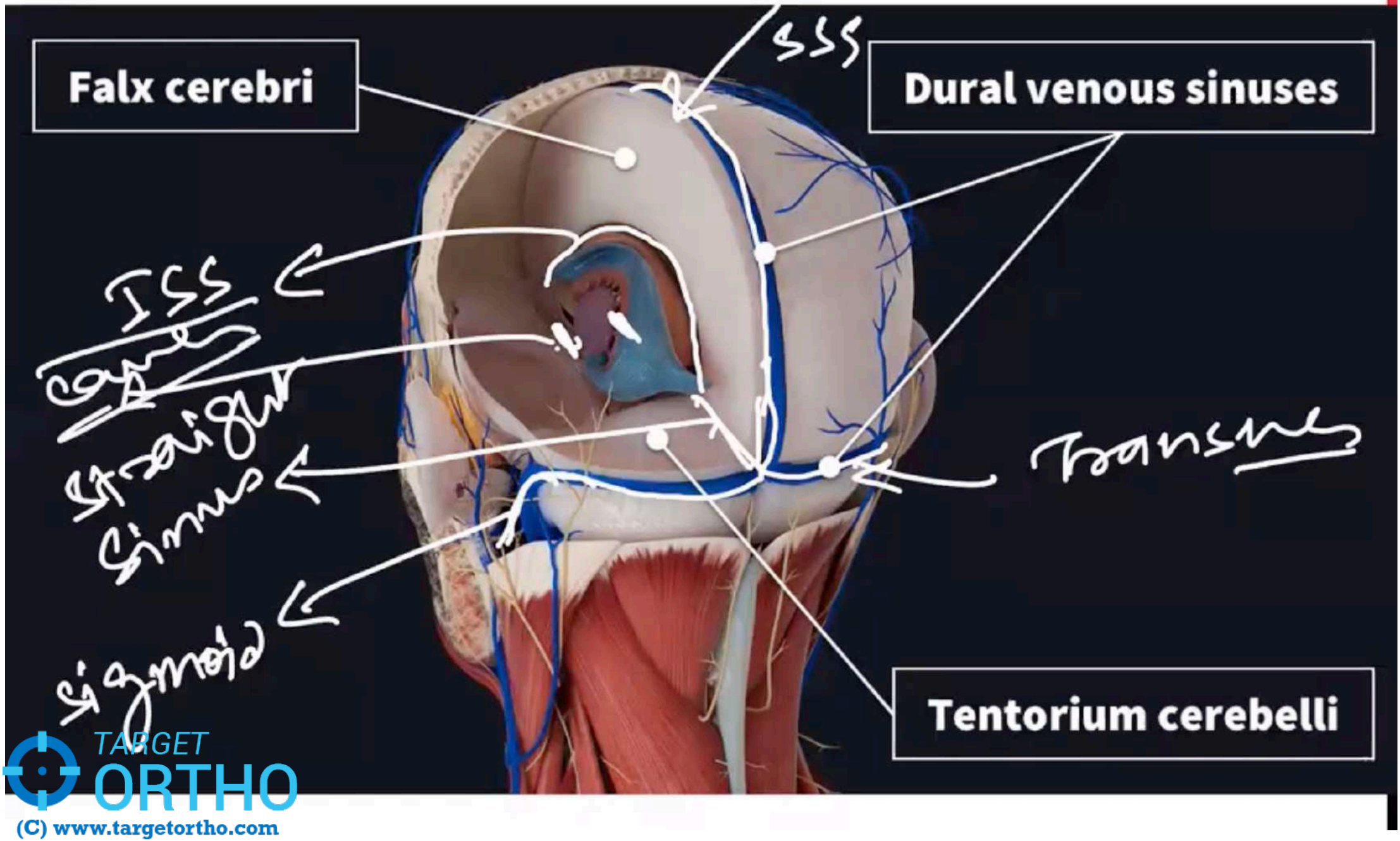
Dura
Arachnoid
Pia

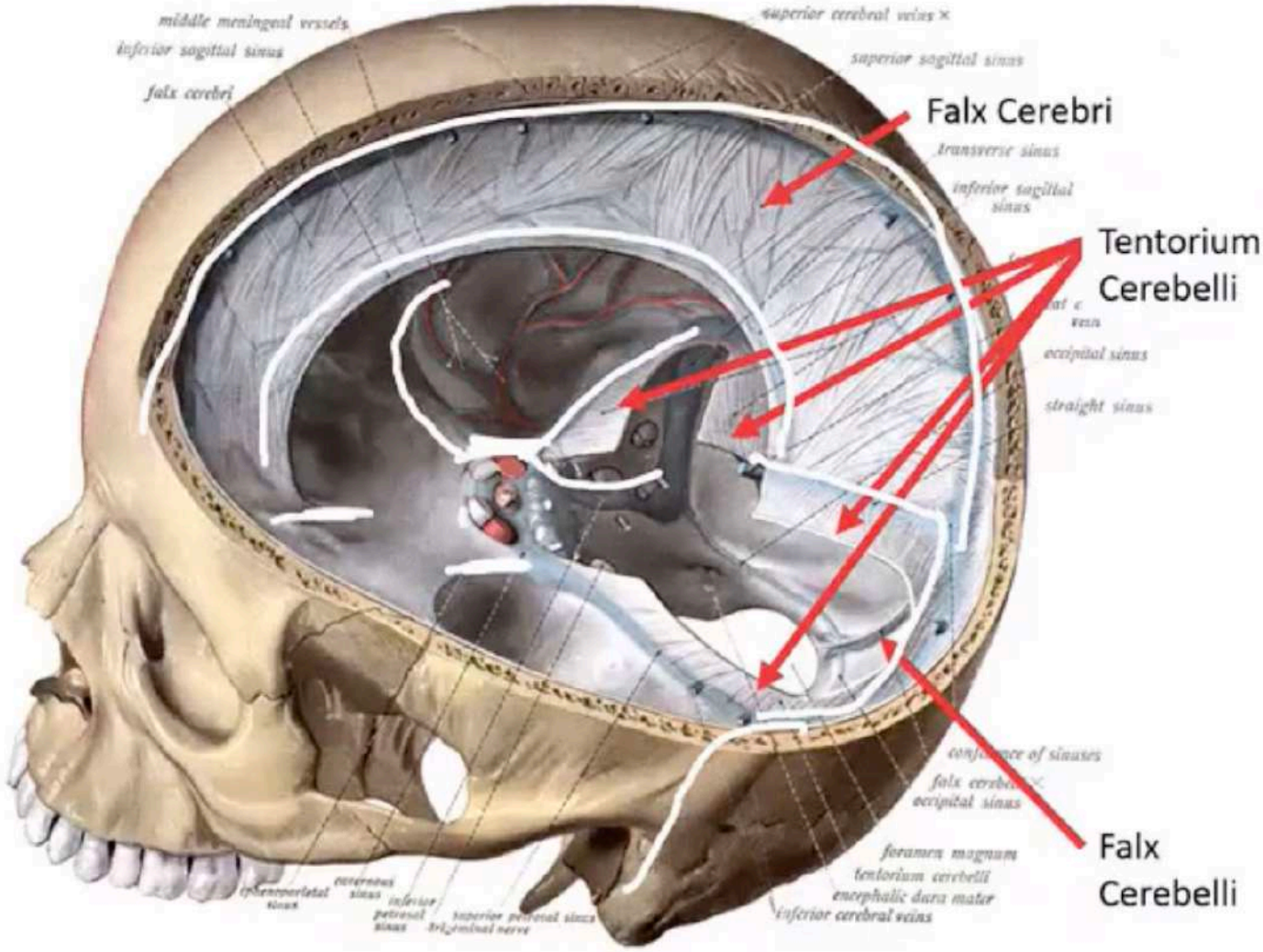


Falx cerebri

Tentorium cerebelli

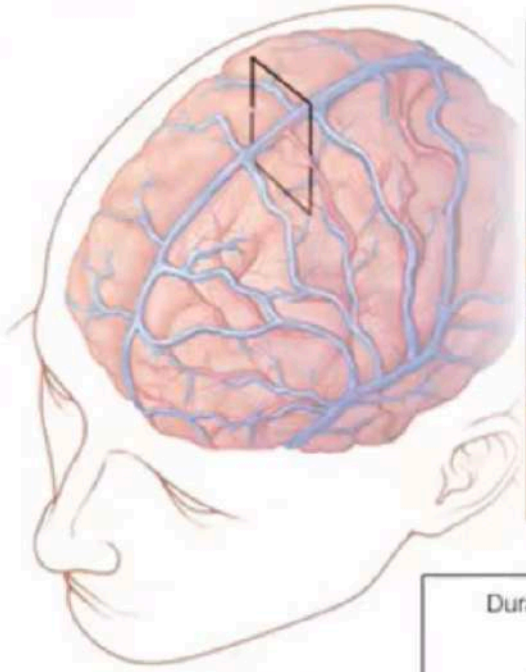
Layers covering the Brain

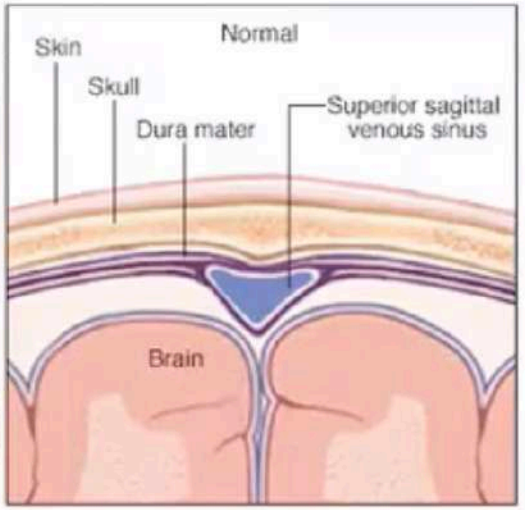


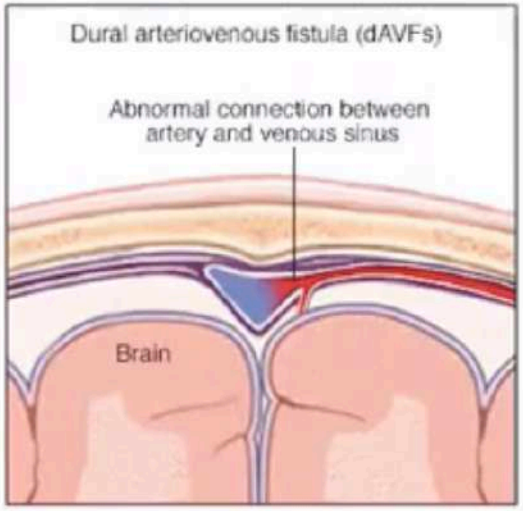


INTRODUCTION – DURAL AVF

- Aka Dural AVMs
- The arteriovenous shunt is contained within the leaflets of the dura mater
- Supplied by branches of the ICA/ECA/Vertebral A.
- They are considered acquired rather than congenital
- Hence preferred to be ~~called fistulae~~ over malformation

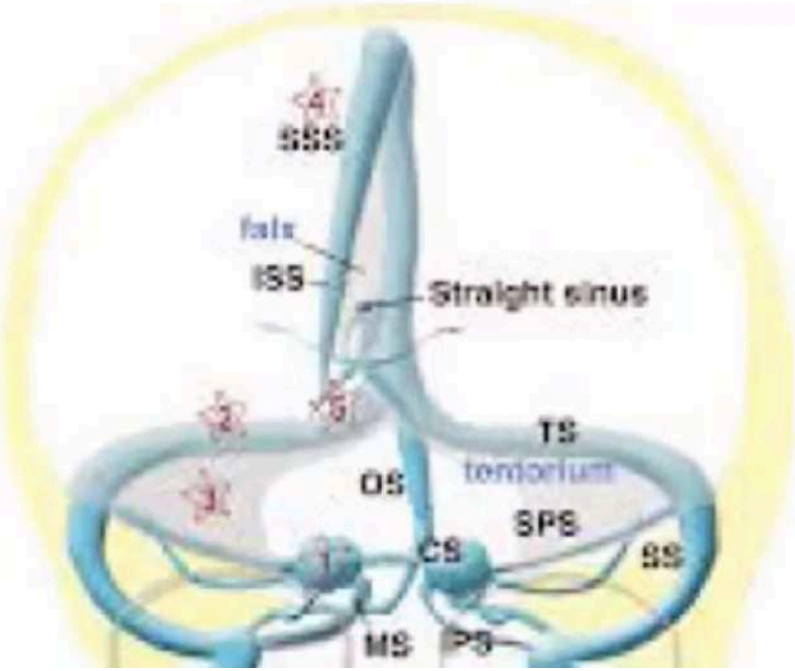




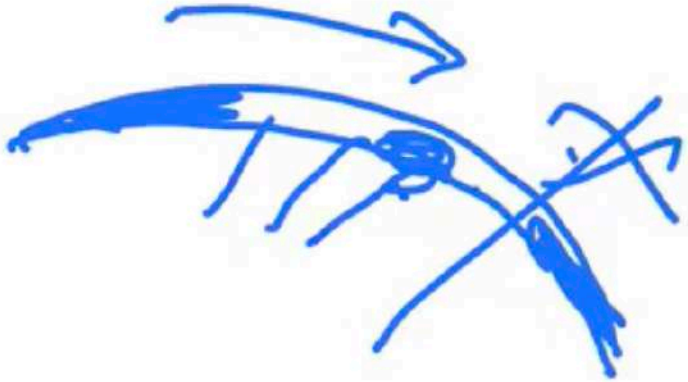


COMMON LOCATIONS

1. **Transverse / sigmoid: 63%**
   - Slight left predominence
   - Epicenter at the junction of sinuses
2. **Tentorial / petrosal**
3. **Anterior fossa / ethmoidal**
4. **[illegible]**
5. **Cavernous sinus (carotid-cavernous fistula: CCF)**
6. **Superior sagittal sinus**
7. **Foramen magnum**



ETIOLOGY



- **acquired, idiopathic lesions**
- **Associations with**
  - Venous sinus thrombosis
  - Theories:
    - Dormant embryonic dural arteriovenous channels
    - Angiogenesis and de novo formation of DAVF

EPIDEMIOLOGY

- 10 to 15% of all intracranial AVMs
- 61-66% in females
- Usual age 40-59 years
- Rarely in children

PRESENTATION

Symptoms / signs

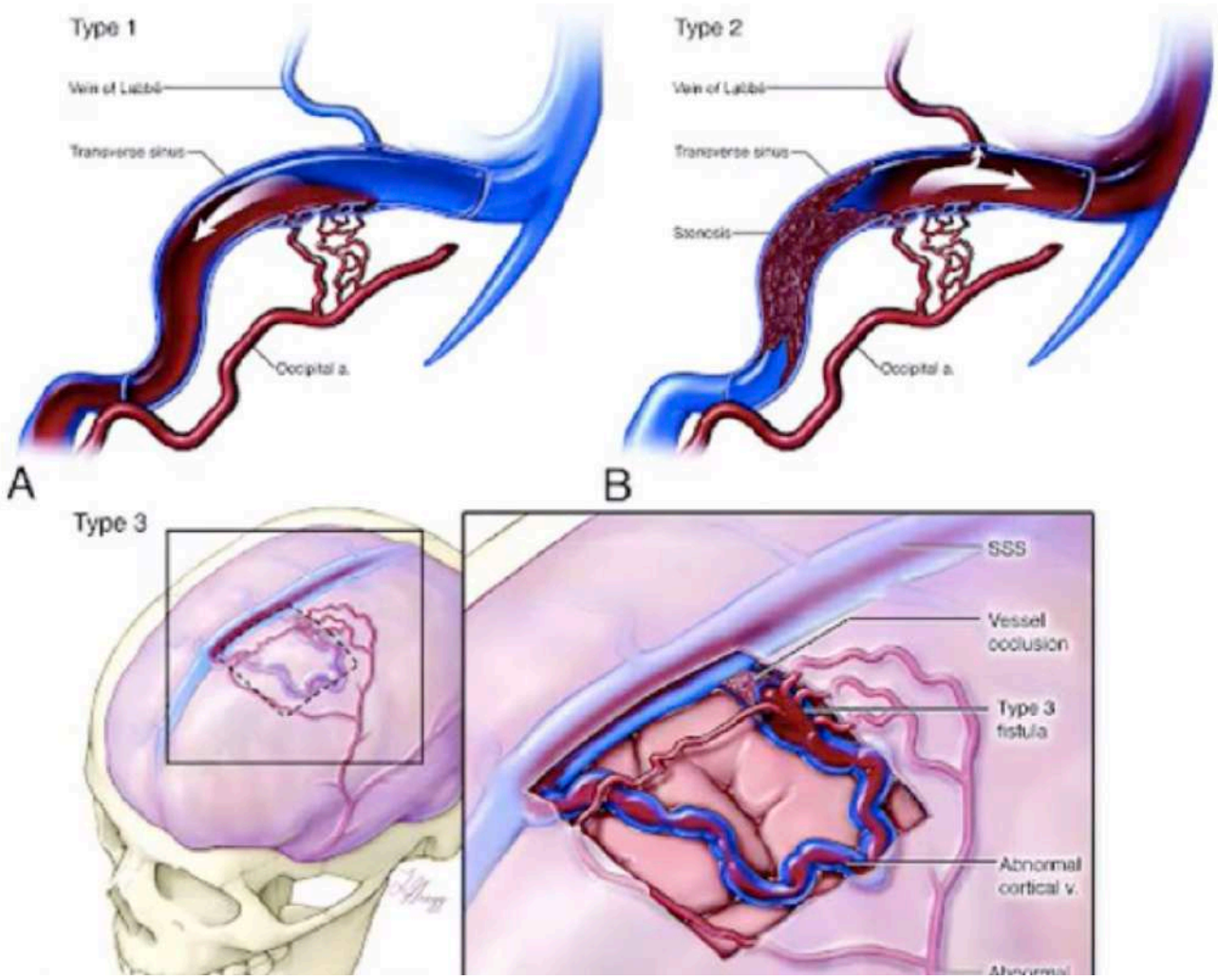
- Pulsatile tinnitus : 92%
- Occipital bruit : 89%
- Headache : 41%
- Visual impairment : 33%
- Papilloedema : 26%
- Seizures, CN palsies, orbital venous congestion

PATHOGENESIS

DAVF → cortical venous hypertension

→ Raised ICP → indication for treatment

→ Global cerebral edema / hydrocephalus



PATHOGENESIS

SDH

DAVF → cortical venous hypertension

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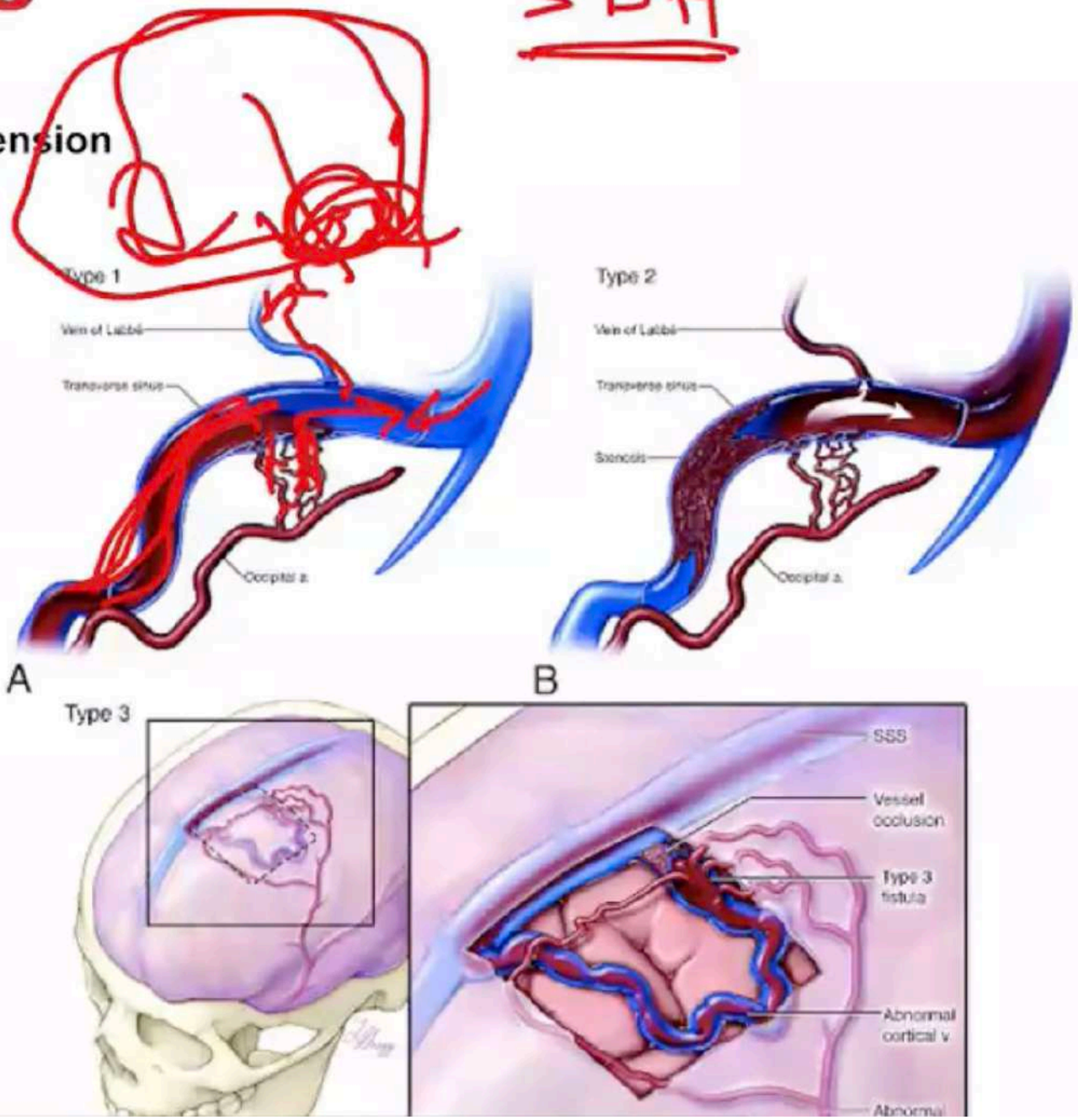
Obs

Non-obs / comm

comm.



EVALUATION

CT / MRI without contrast

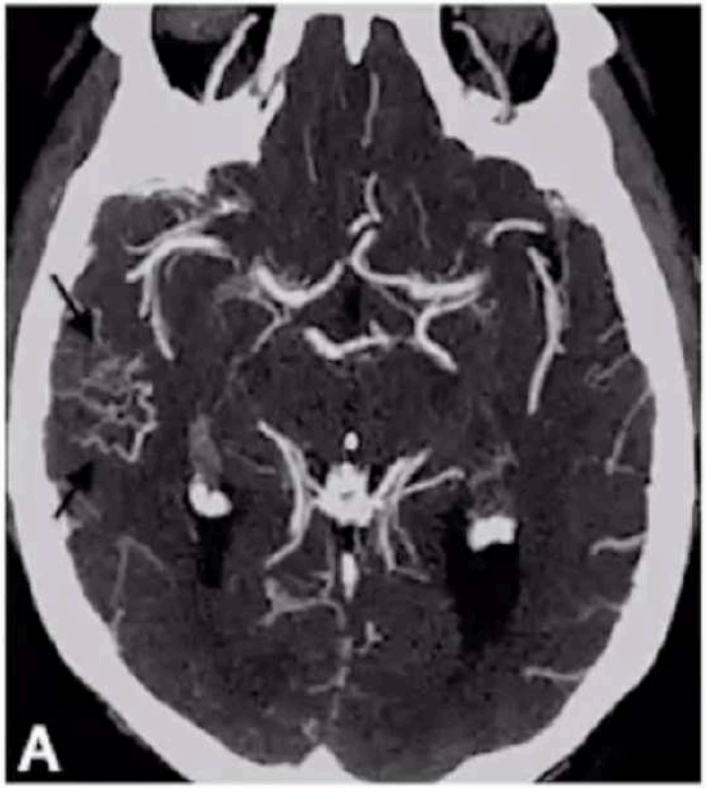
- Often normal

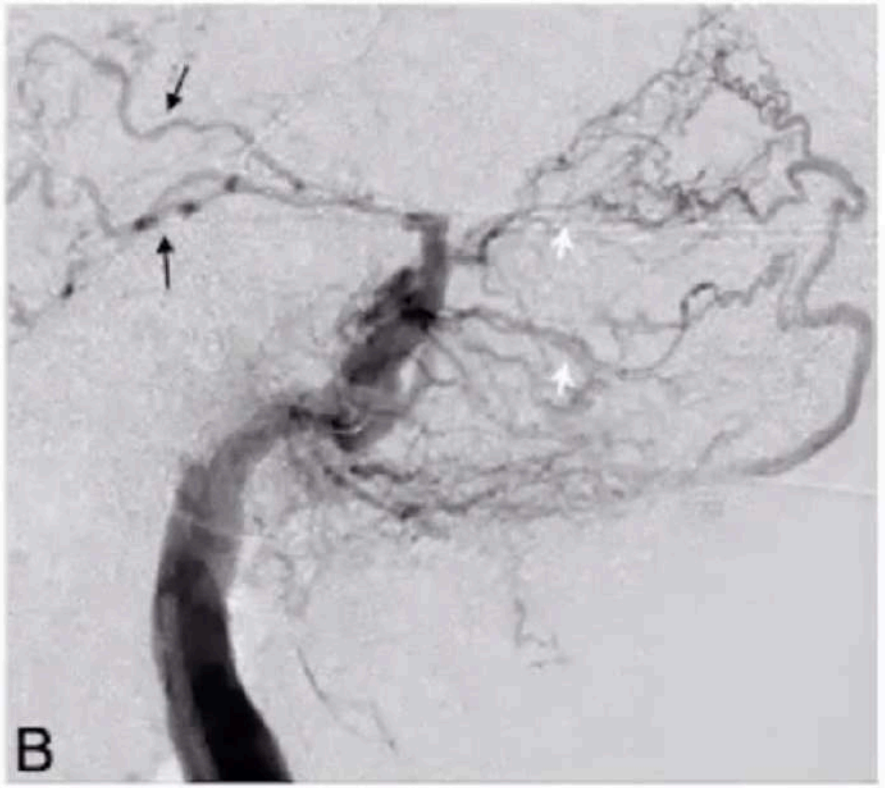
CTA

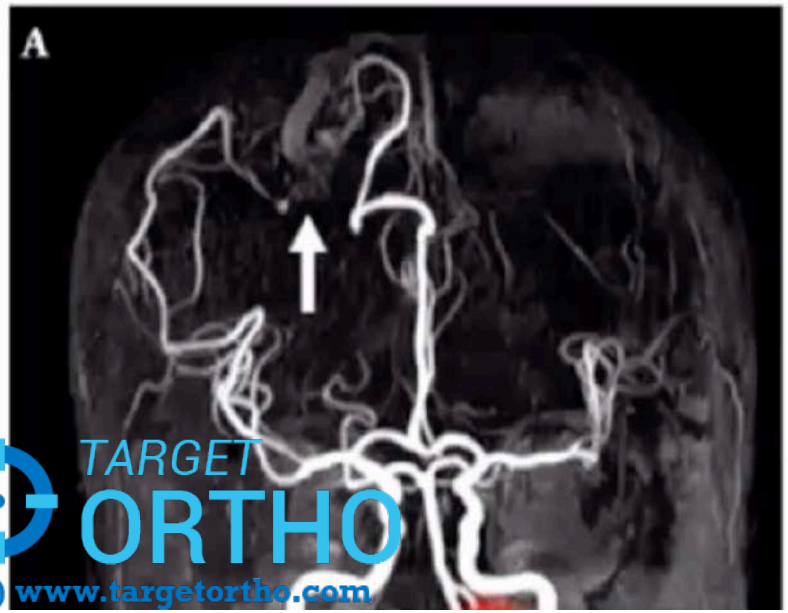
- Dilated tortuous vessels – feeders / drainers

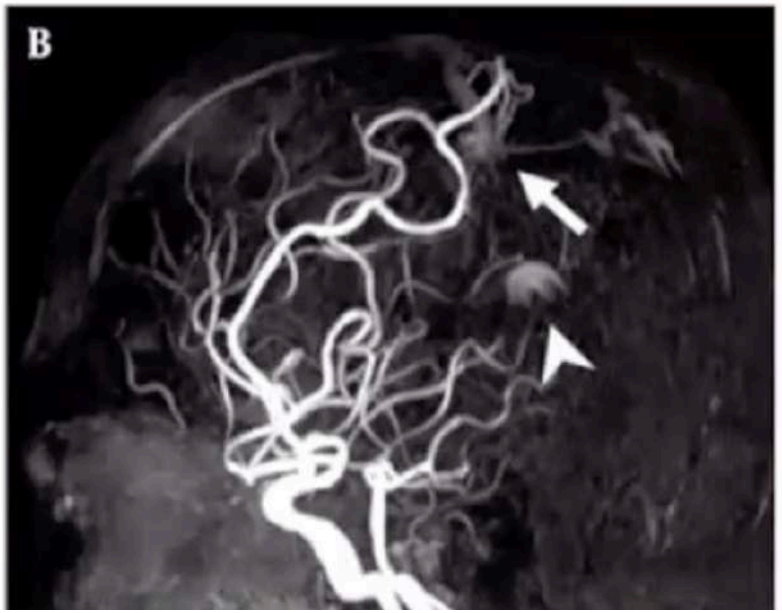
MRA

- Dilated pial vessels
- Early prominent venous filling
- Sinus enlargement or occlusion
- White matter edema

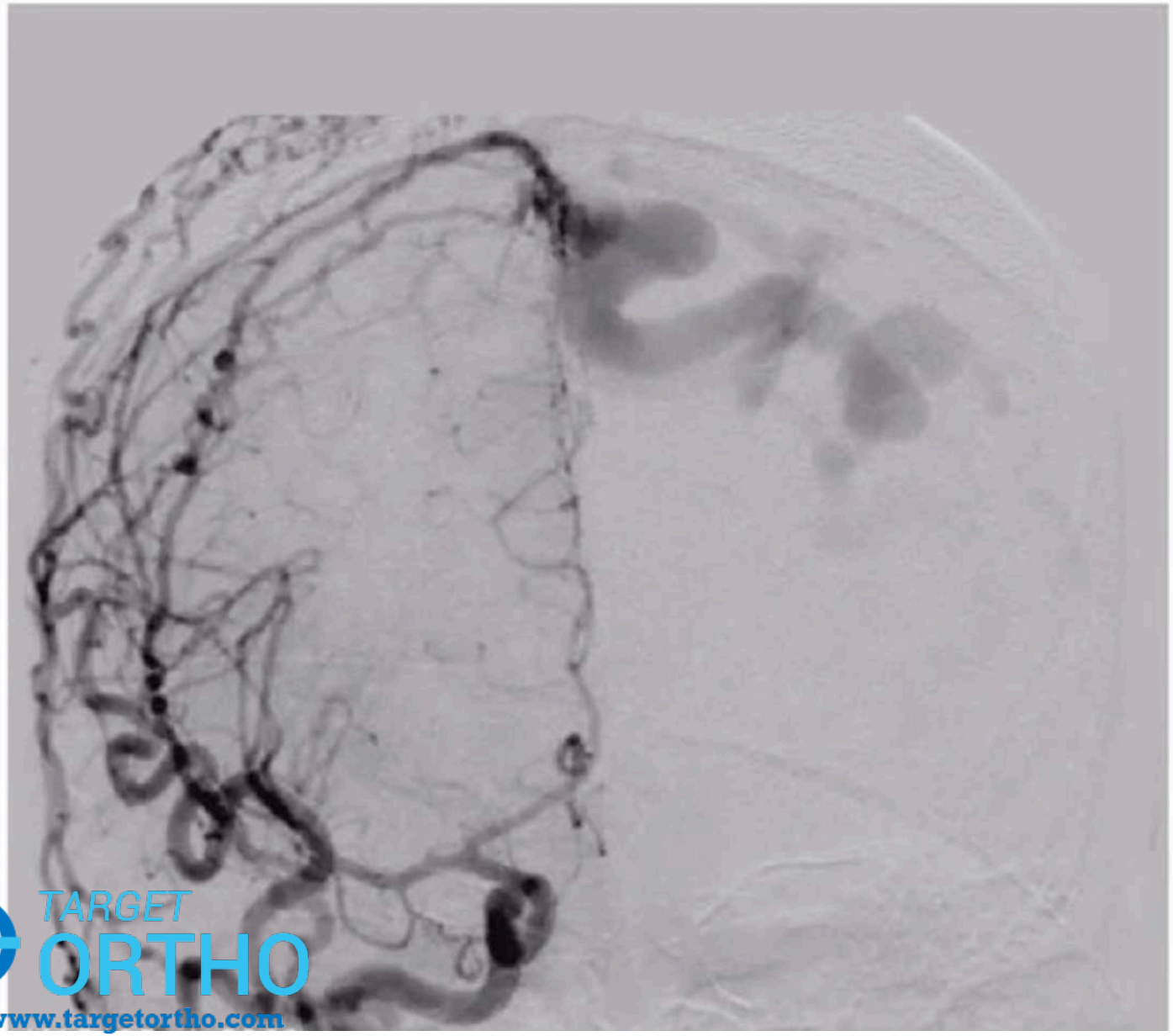








6 VESSEL CEREBRAL ANGIOGRAPHY



ANGIOGRAPHIC CLASSIFICATIONS

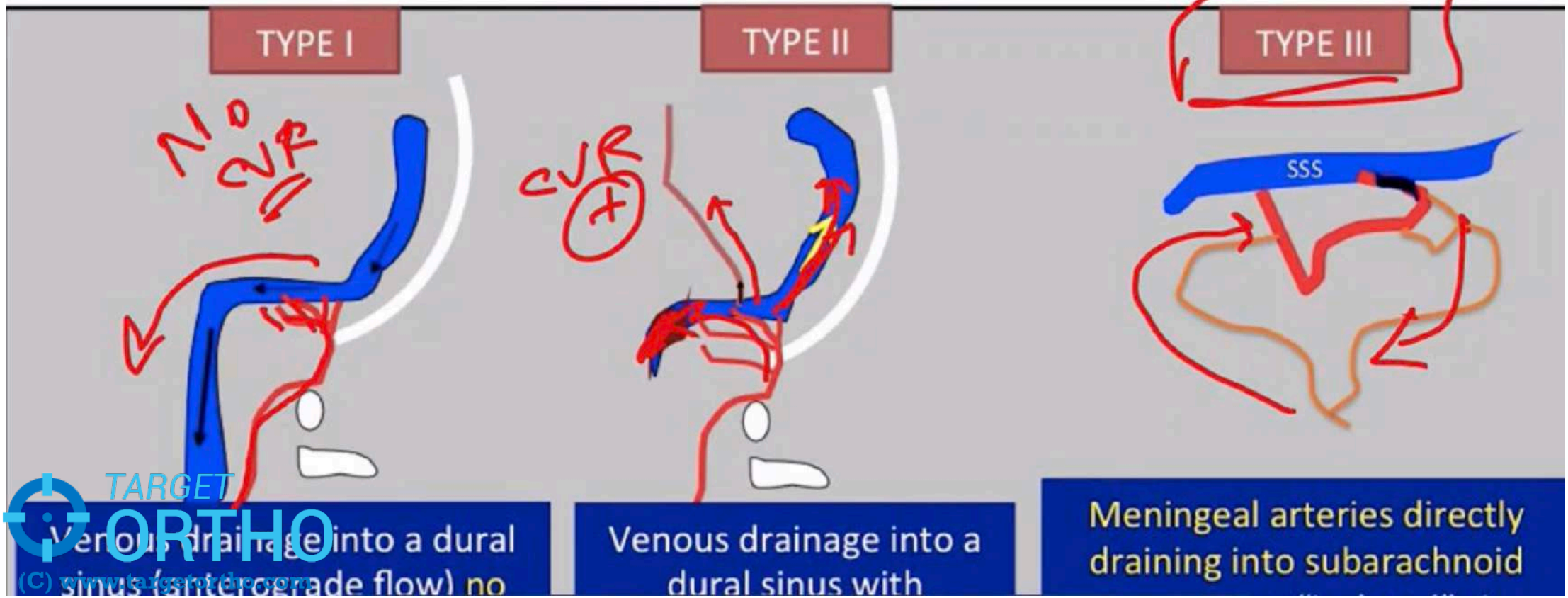
Cortical venous drainage – key factor

Distinguish:

- Benign (low – grade) 54% (no CVR)
- Aggressive (high – grade)

BORDEN CLASSIFICATION

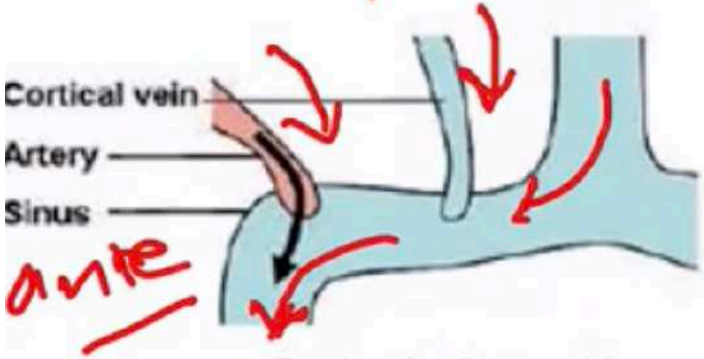
Type	Features
I	DAVF drainage into a dural venous sinus or meningeal veins, with normal anterograde flow. Usually clinically benign.
II	DAVF draining anterograde into dural venous sinus, but with retrograde flow into cortical veins.
III	DAVF with direct retrograde flow from fistula into cortical veins, causing venous hypertension.
Above further subclassified into a: with single hole and b: multiple holes	



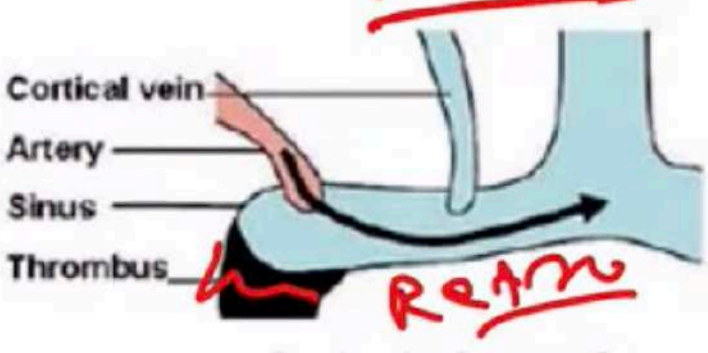
COGNARD CLASSIFICATION

Borden classification	
1	Venous drainage directly into dural venous sinus or meningeal vein
2	Venous drainage into dural venous sinus with CVR
3	Venous drainage directly into subarachnoid veins (CVR only)
Cognard *et al.*	
I	Venous drainage into dural venous sinus with antegrade flow
II a	Venous drainage into dural venous sinus with retrograde flow
II b	Venous drainage into dural venous sinus with antegrade flow and CVR
II a + b	Venous drainage into dural venous sinus with retrograde flow and CVR
III	Venous drainage directly into subarachnoid veins (CVR only)
IV	Type III with venous ectasias of the draining subarachnoid veins
V	Direct drainage into spinal perimedullary veins
	(CVR indicates cortical venous reflux)

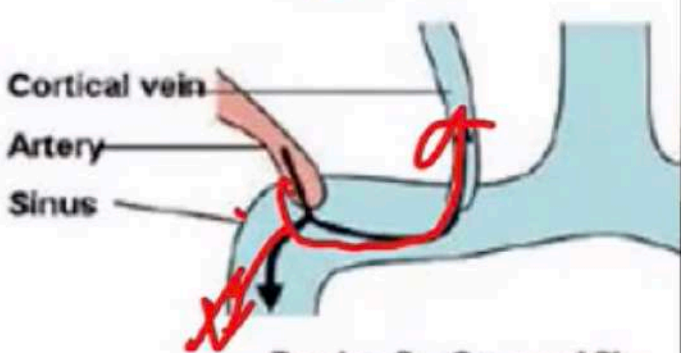
Dural Arteriovenous Fistula Classification



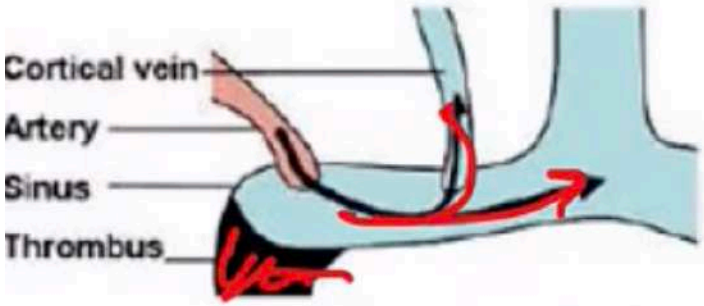
Borden 1 – Cognard 1



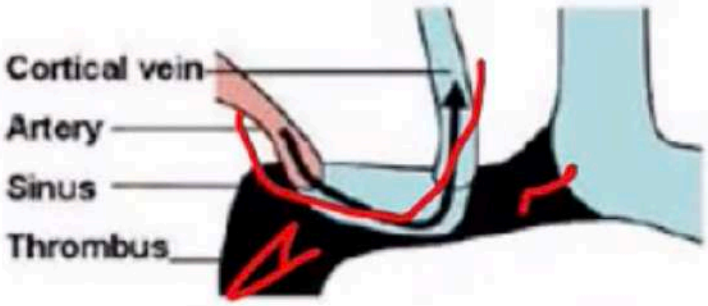
Borden 1 – Cognard 2a



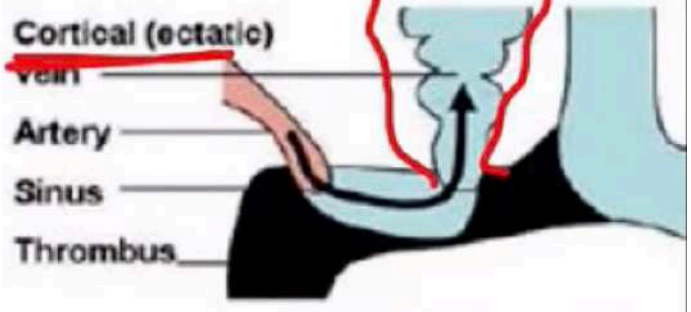
Borden 2 – Cognard 2b



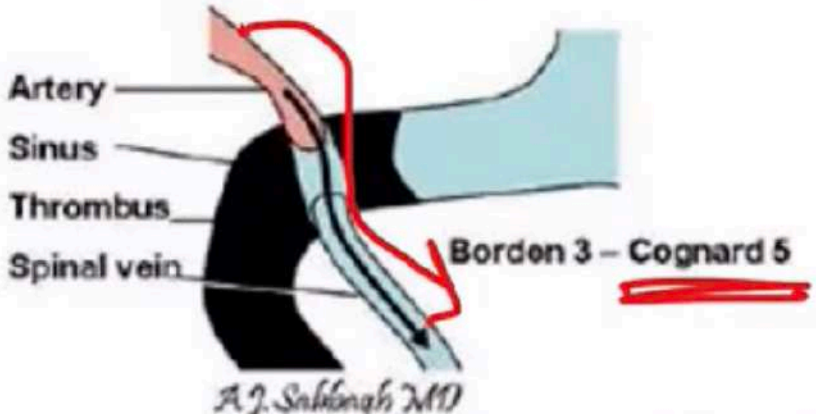
Borden 2 – Cognard 2a+b



Borden 3 – Cognard 3



Borden 3 – Cognard 4



Borden 3 – Cognard 5

NATURAL HISTORY

Benign lesions

- **98% asymptomatic – over a 3 year period**

Aggressive lesions

- **Over a 4 yr period, annual rates of**
  - Hemorrhage 8.1%
  - Non-hemorrhagic neurological deficit 6.9 %
  - Mortality 10.4%

Locations associated with aggressiveness

Aggressive:benign ratio

- Tentorial 31:1
- Middle fossa / sylvian 2.5:1
- Anterior fossa / ethmoidal 2.1:1

MANAGEMENT

Indications for intervention:

- **Presence of cortical venous drainage**
  - In absence, follow them radiographically (2% evolve to develop cortical venous drainage)
- **Neurologic dysfunction**
- **Hemorrhage**
- **Orbital venous congestion**
- **Refractory symptoms**

MANUAL CAROTID SELF COMPRESSION

Thrombosis rate 22%

Clinical improvement 33%

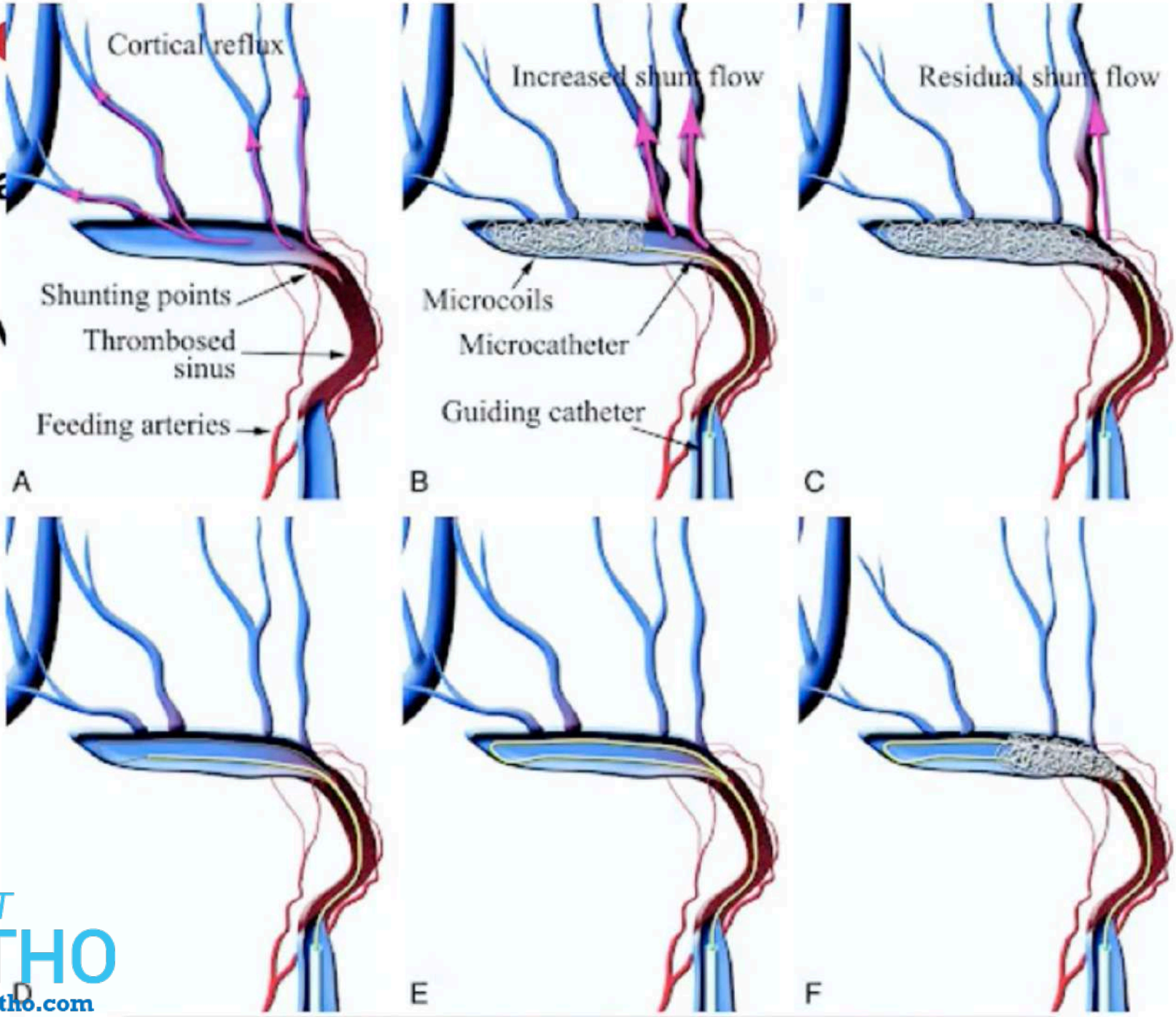
Use the opposite hand

10 min / once a day, to begin with

ENDO

Transa

Transv



SURGERY

Primary treatment – endovascular embolisation

Surgery indications:

- partial/incomplete/failed endovascular treatment
- Inaccessible by pure endovascular route
- Anterior fossa / ethmoid
- Tentorial

SURGICAL STRATEGIES

- Preoperative embolisation
- Avoid using craniotome
- Rapid transfusion
- Surgical assisted embolisation

SRS

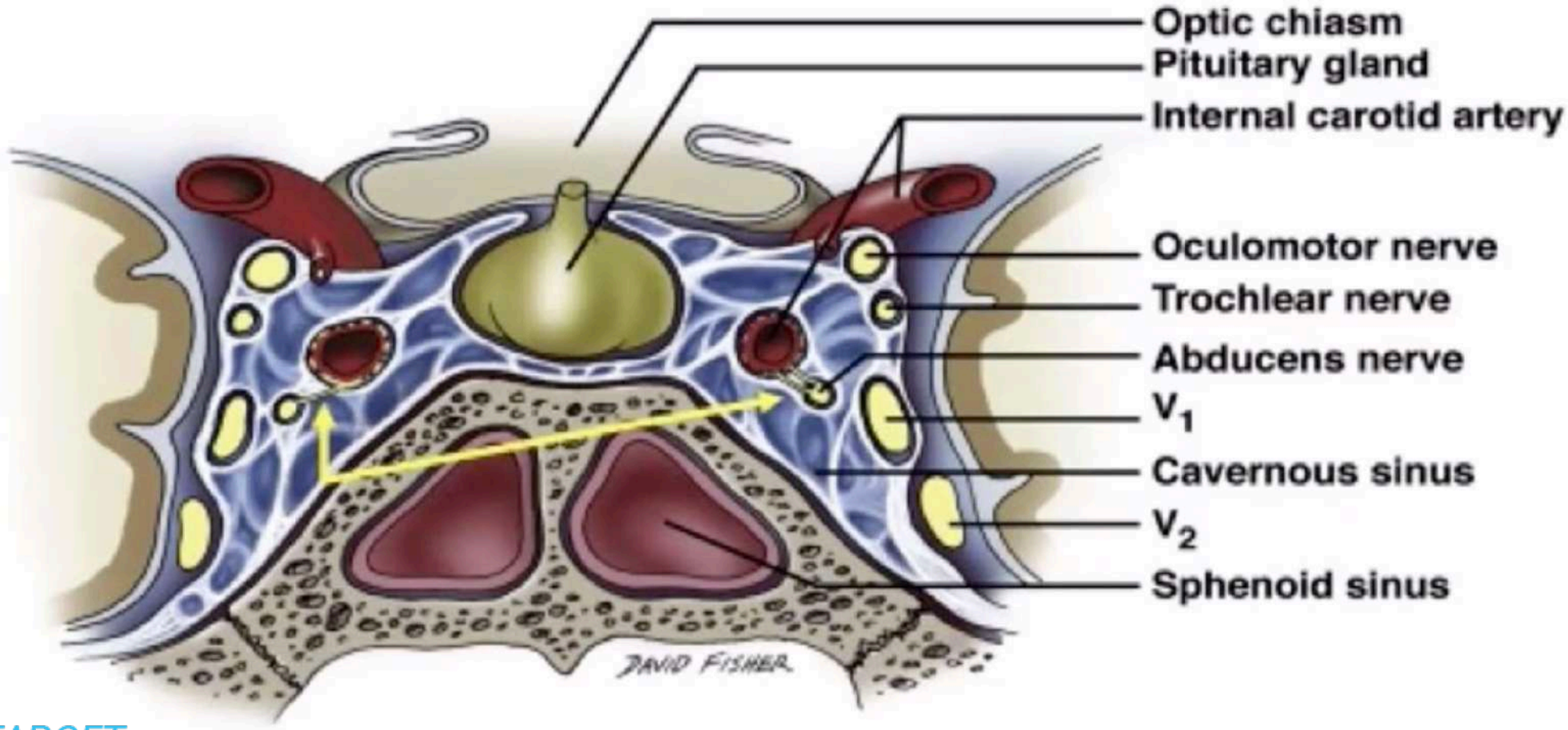
Post – embolisation

Obliteration rate 58% with only SRS

71% when SRS used after failed embolisation/surgery

CAROTID CAVERNOUS FISTULA

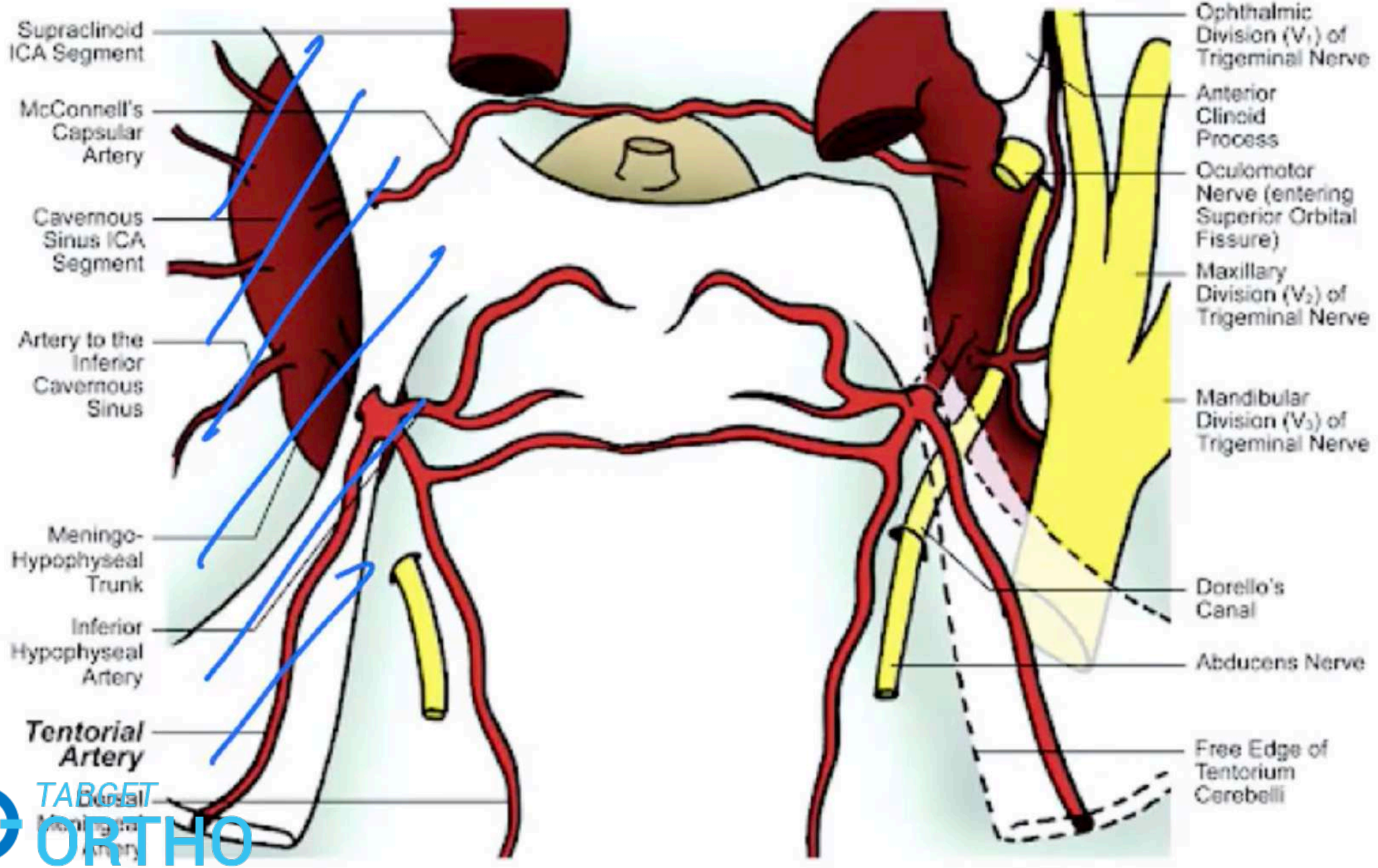
ANATOMY



ARTERIES

Branches of carotid artery within the cavernous sinus

1. **The meningohypophyseal trunk**
   - A. The tentorial artery of Bernasconi-Cassinari
     - The tentorium
   - B. The inferior hypophyseal artery
     - Posterior pituitary
   - C. The dorsal meningeal artery
     - The clival area and 6th CN
2. **The artery of the inferior cavernous sinus**
3. **McConnell's capsular arteries**



ICA – ECA anastomoses

- The branches of ICA arising within the cavernous sinus anastomose with the branches of ECA

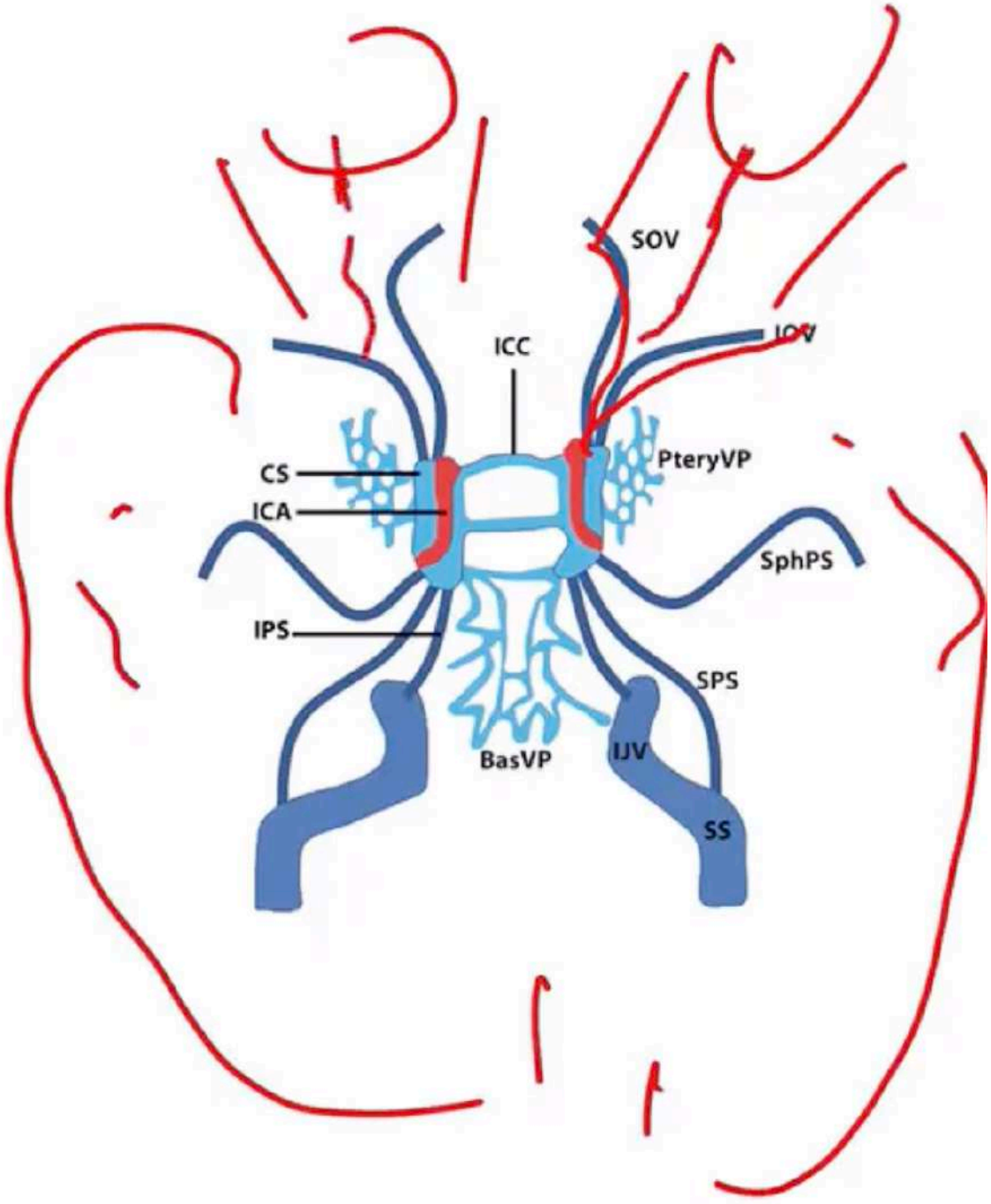
ECA branches

- The neuromeningeal trunk of ascending pharyngeal artery
- The recurrent and anterior meningeal branches of the internal maxillary artery
- The cavernous branch of middle meningeal artery

VEINS

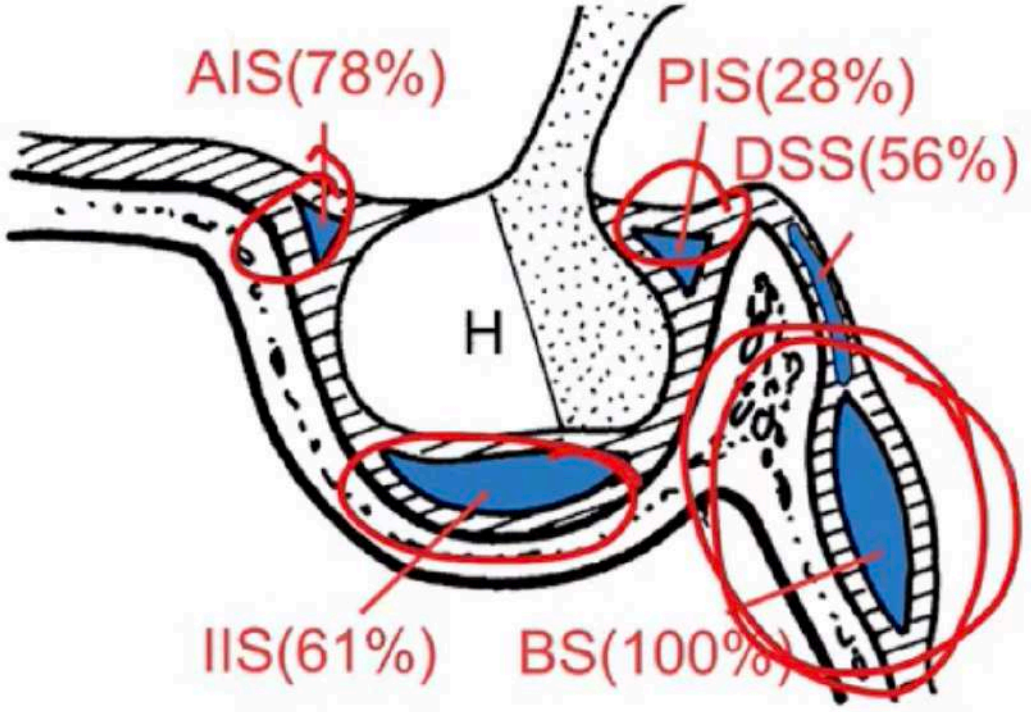
Tributaries of the cavernous sinus:

1. The superior and inferior ophthalmic veins → the facial veins
2. The middle and inferior cerebral veins
3. The central retinal vein, the middle meningeal veins, the superior and inferior petrosal sinuses and the emissary veins

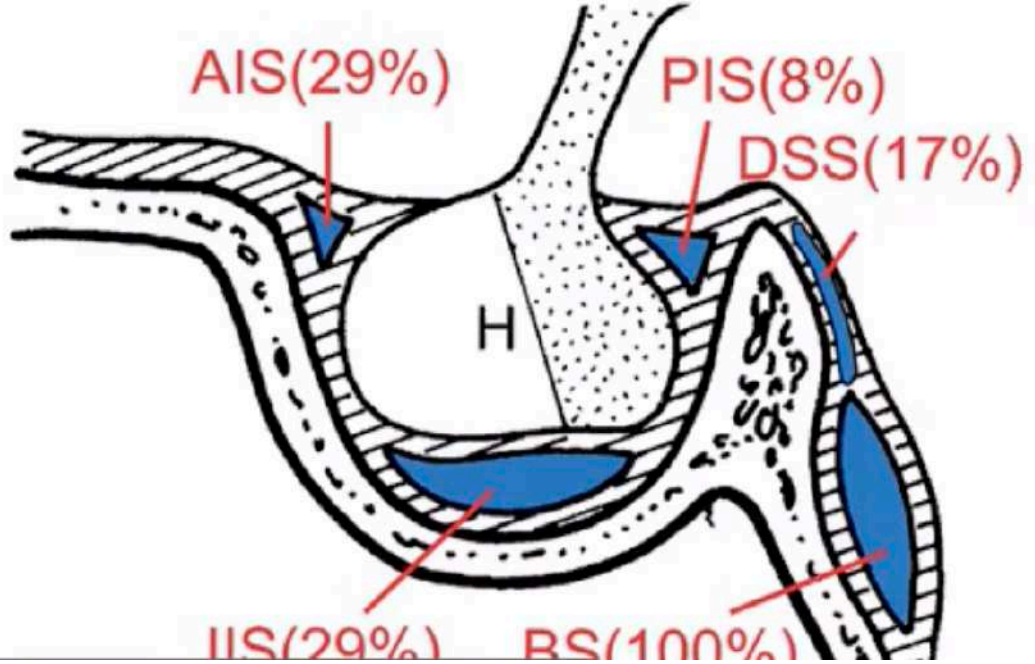


Intercavernous sinus communications:

- **The anterior intercavernous sinus: 0.5-5.4 mm**
- **The posterior intercavernous sinus: 0.7-4.1 mm**
- **The inferior cavernous sinus**
- **The basilar plexus – the largest intercaverous communication**
  - At the level of dorsum sellae



Cadaver



BARROW CLASSIFICATION

Direct type

Type A: direct high flow shunts between ICA and CS

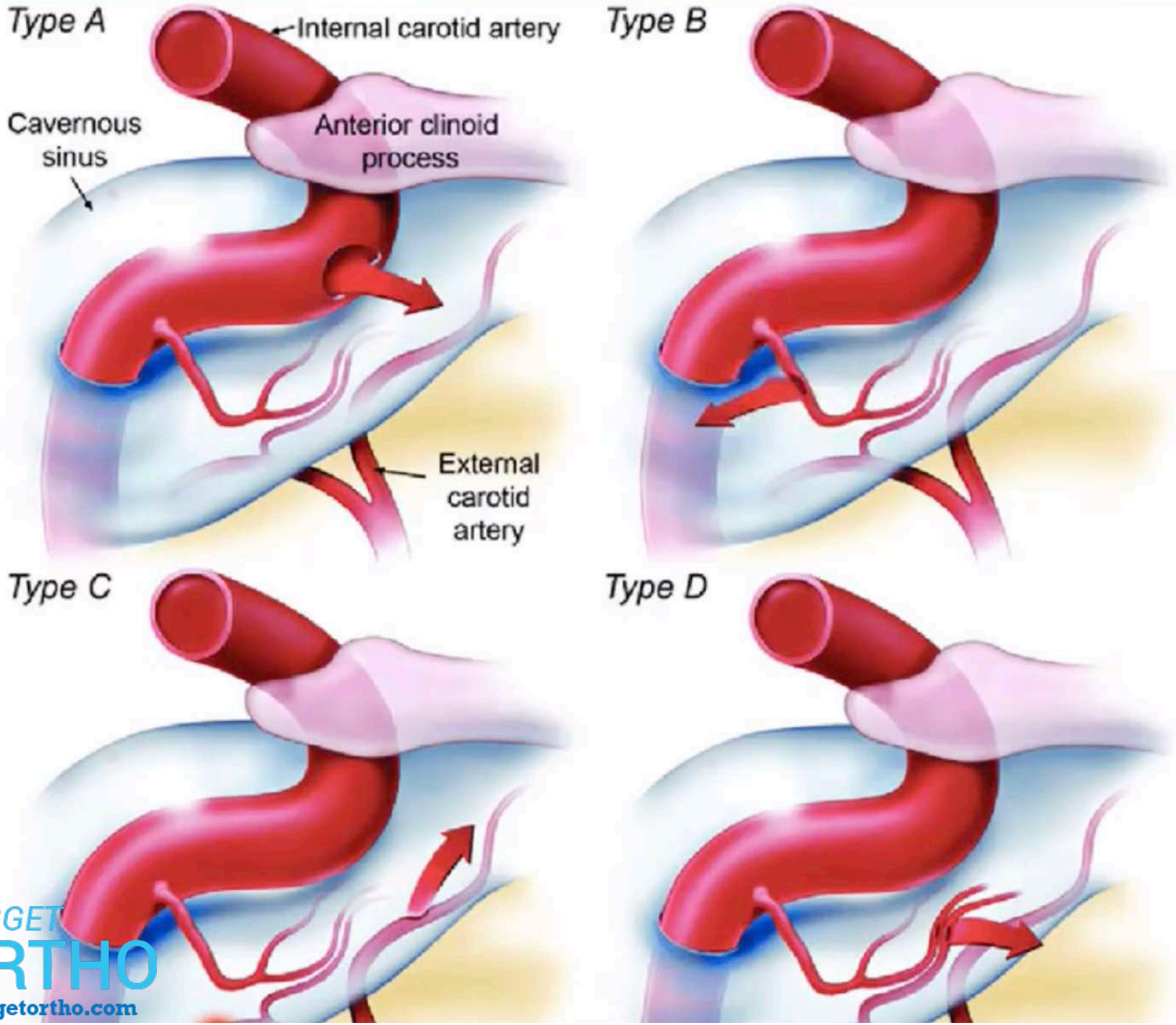
1. Traumatic: 0.2% of craniocerebral trauma; iatrogenic: following percutaneous trigeminal rhizotomy, endovascular procedures
2. Spontaneous: ruptured cavernous ICA aneurysm, connective tissue disorders

Indirect type: shunts; dural arteries, branches of ECA, except type B – low flow

Type B: meningeal branches of ICA

Type C: meningeal branches of ECA

Type D: meningeal branches of ICA & ECA



PRESENTATION

- Orbital / retro orbital pain
- Chemosis
- Pulsatile proptosis
- Ocular / cranial bruit
- Deterioration of visual acuity
- Diplopia: 6th CN palsy; most common
- Pupillary dilatation
- Ophthalmoplegia - 3.4.6
- Increased intraocular pressure
- Rarely: SAH

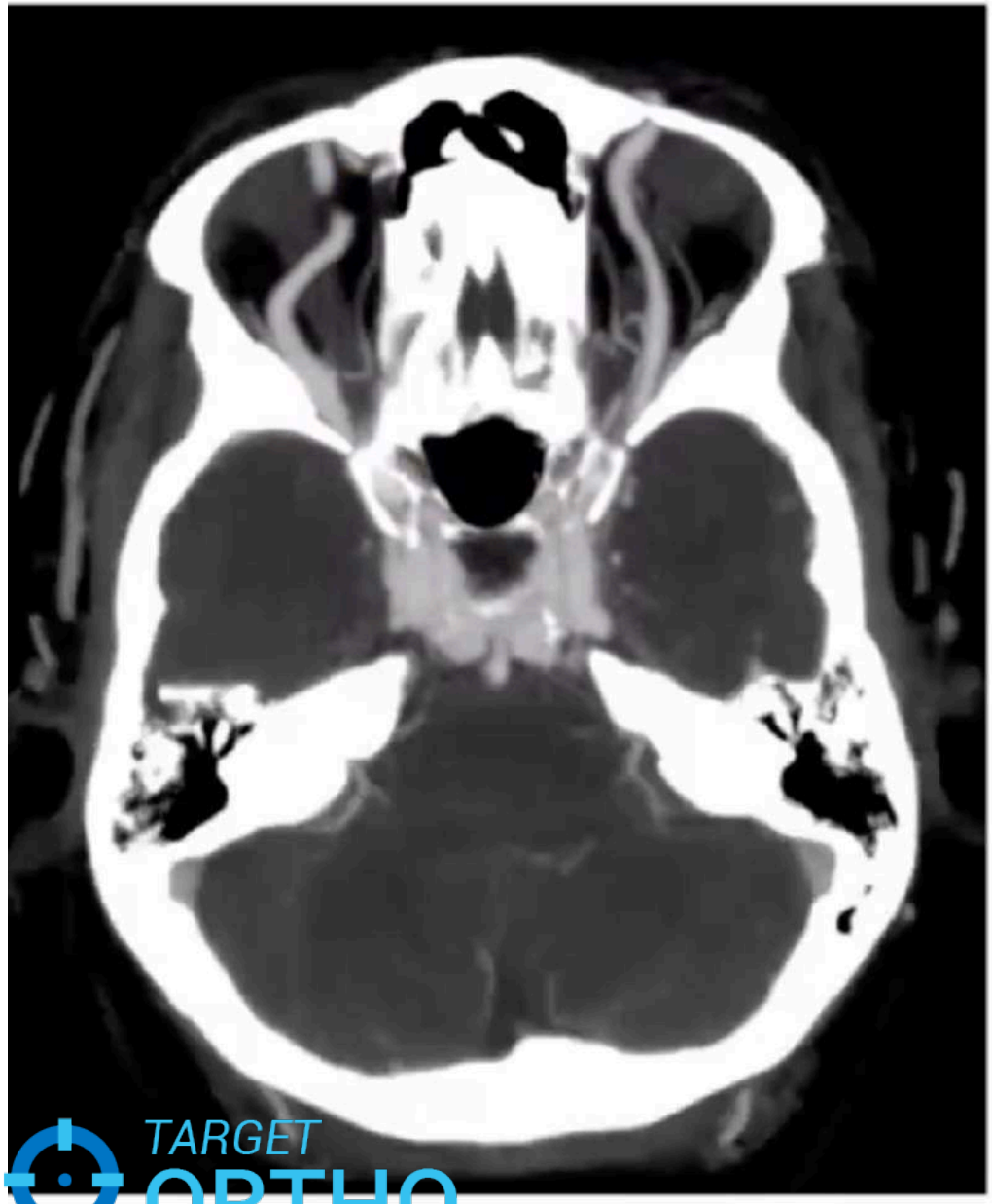
EVALUATION

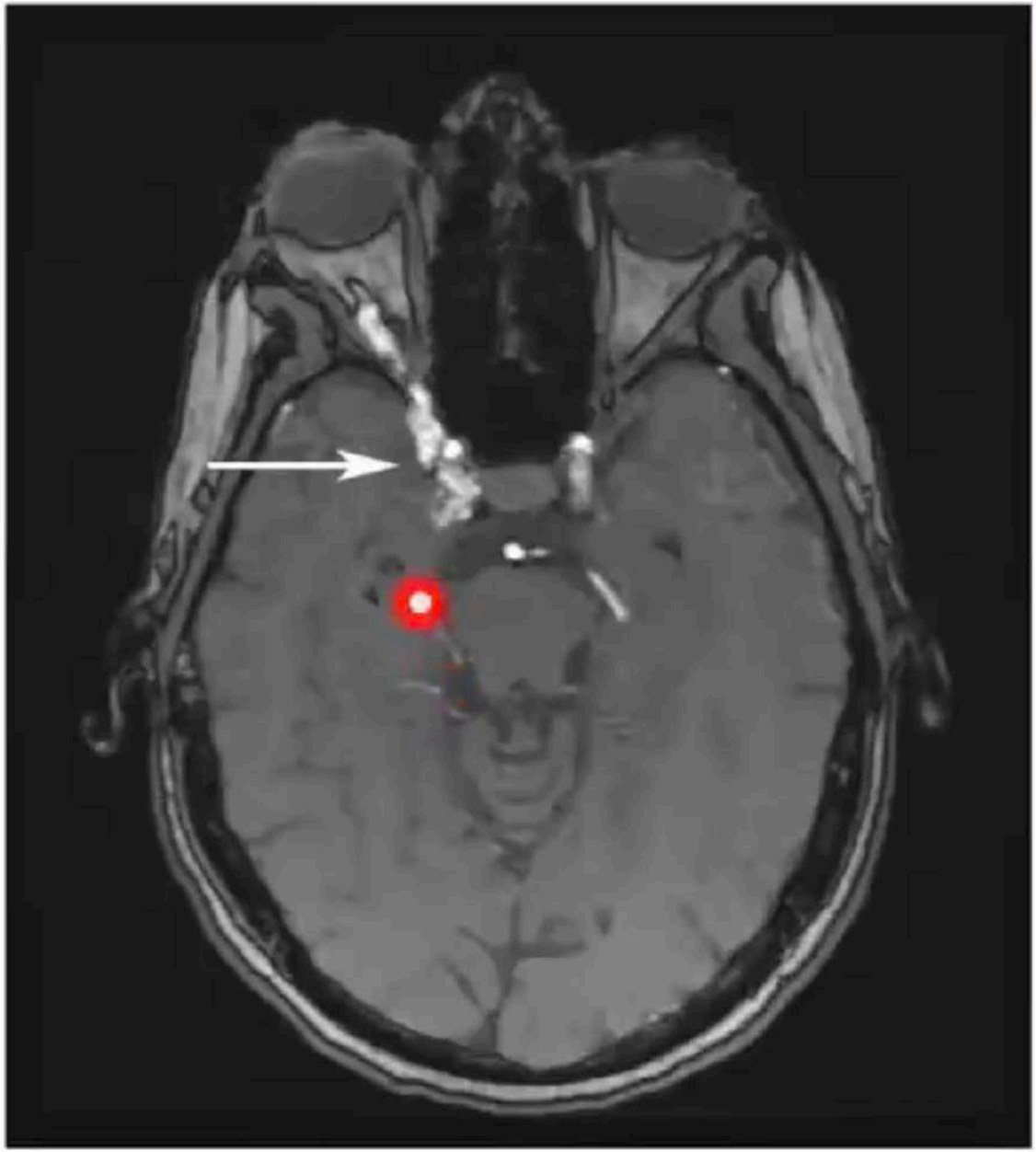
CT / MRI:

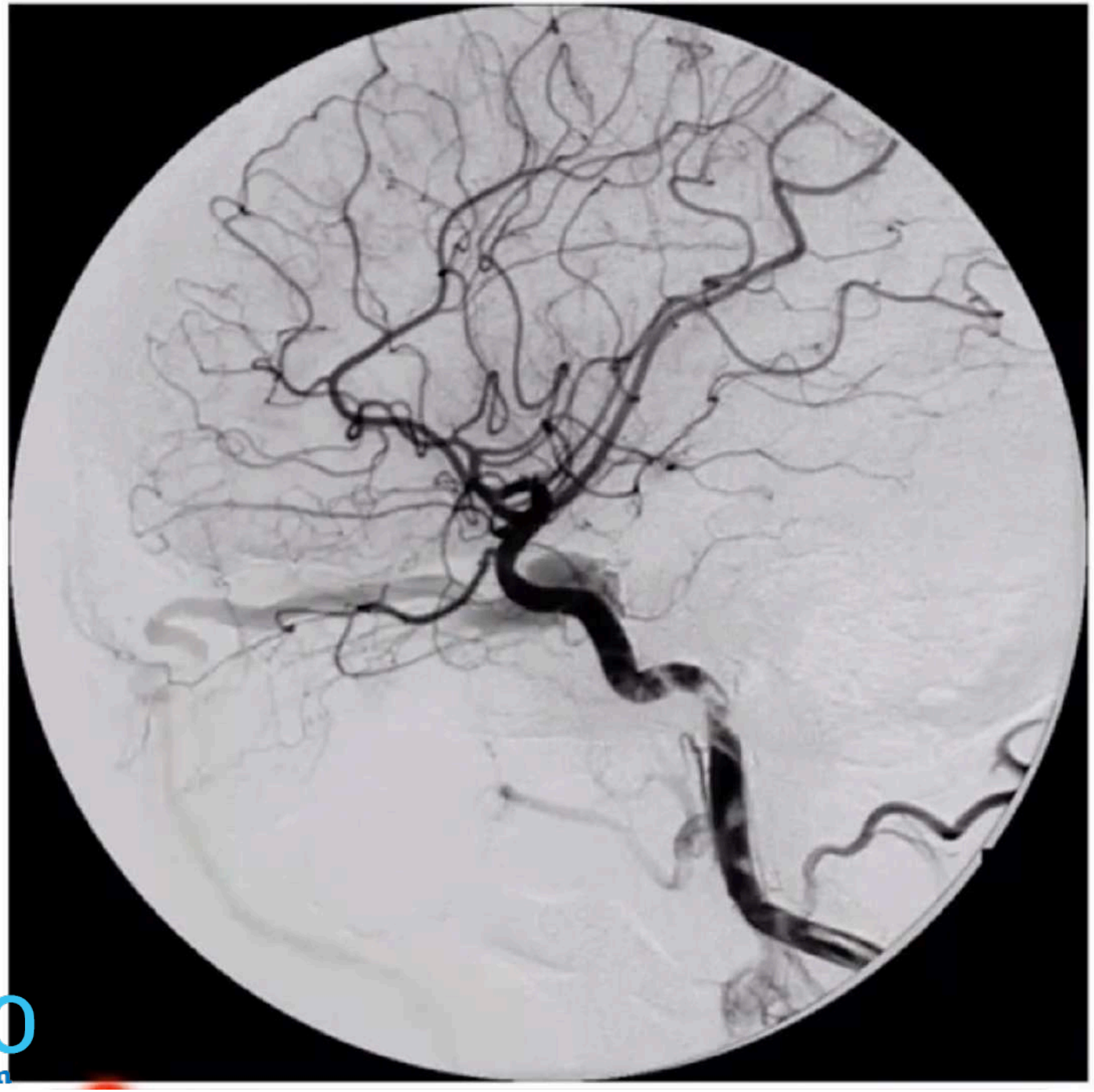
- **Proptosis**
- **Serpigenous and ingorged intraocular vessels**
  - Seen on T2WI coronals

Angiography

- **Shunting of blood from ICA into CS**
- **Rapid opacification of petrosal sinus and/or opthalmic vein**
- **Huber maneuver, Mehringer – Hieshma maneu**







TREATMENT

- 20 to 50% of low flow CCF spontaneously thombose
- Conservatively manage till
  - Visual acuity is stable
  - Intra ocular pressure is <=25
- High flow CCF – urgent treatment
- Goal: preservation of vision, ocular motility

Indications of treatment:

- Proptosis
- Visual loss
- CN 6 palsy
- Bruit
- Severely elevated IOP
- Filling of cortical veins on angiogram

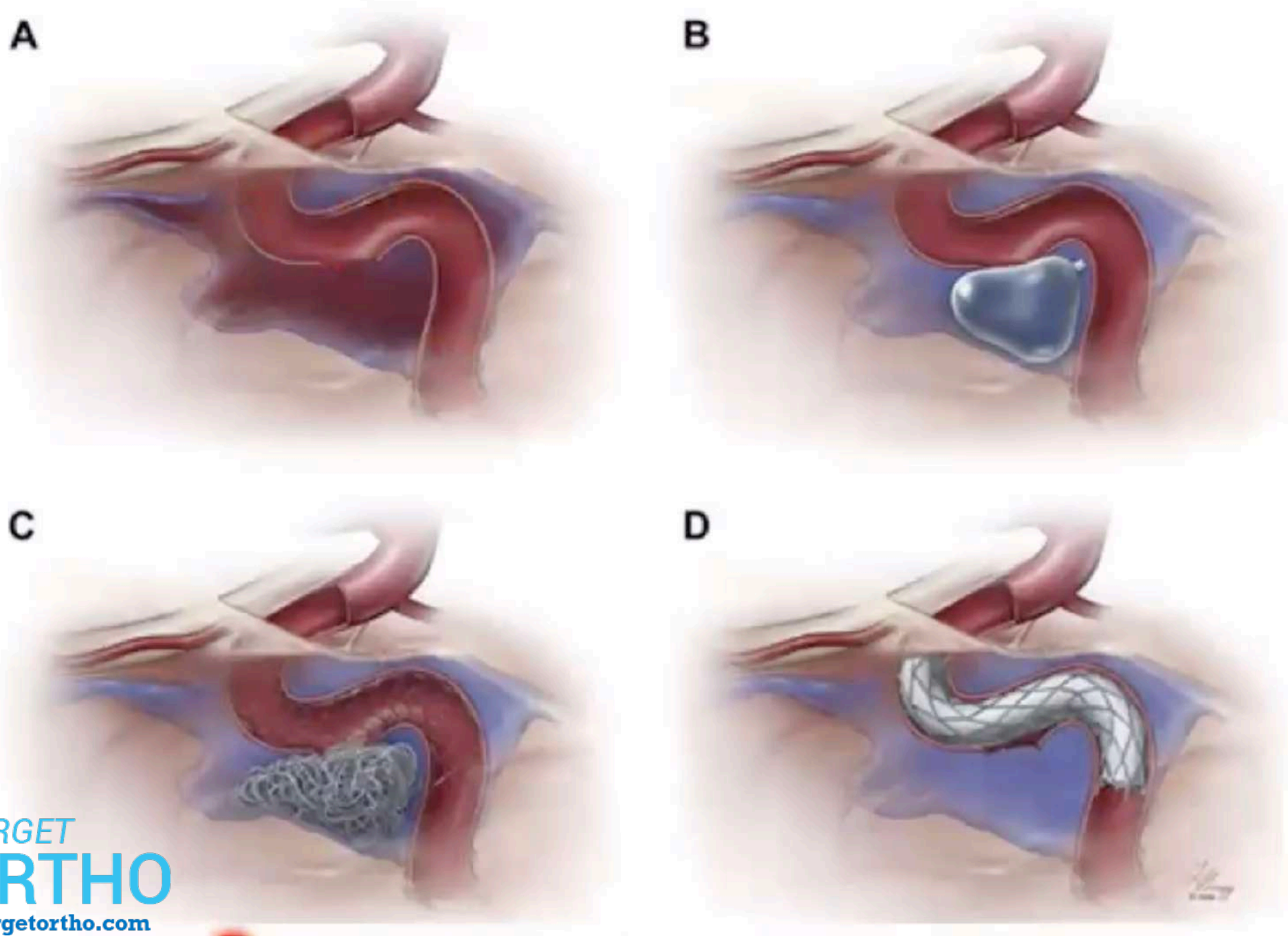
ENDOVASCULAR TREATMENT

Options:

1. Electrolytically detachable coils - platinum
2. Amplatzer vascular plug
3. Silicon balloons
4. Covered stents

Routes:

1. Transarterial through ICA
2. Trasarterial through ECA
3. Transvenous



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Type A: embolization by balloons, coils or embolic agents

Type B: very rare, direct surgical approach as it cannot be occluded with a balloon

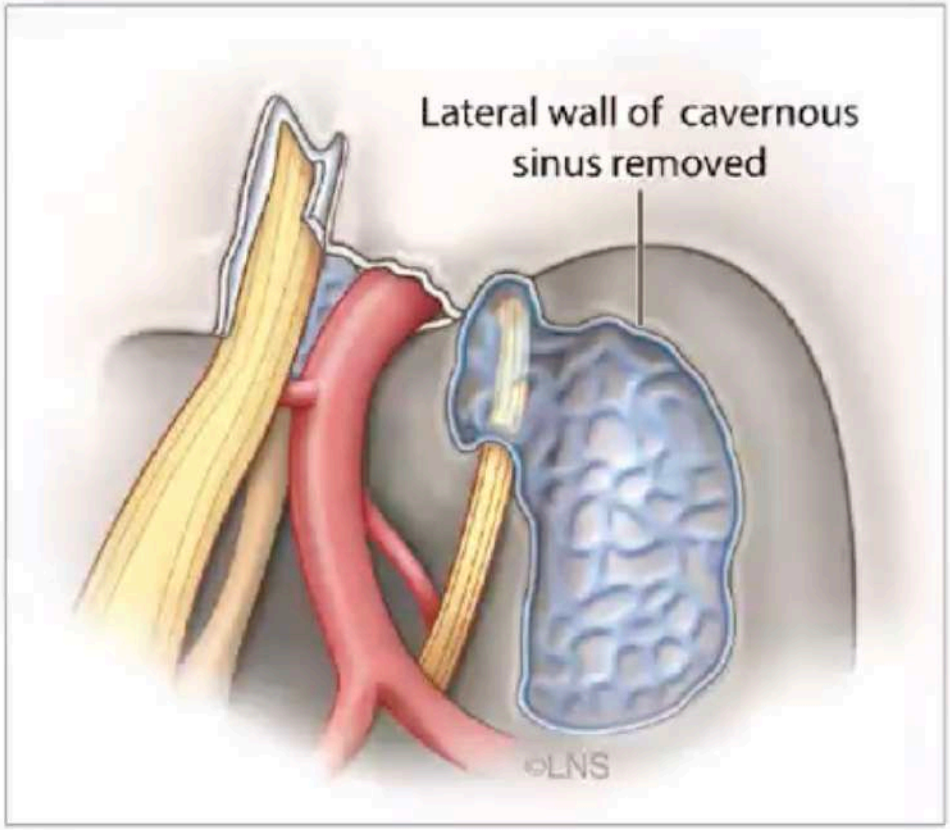
Type C: rare, embolisation of ECA

Type D: embolisation of ECA feeders → ICA feeders at a later stage

SURGICAL APPROACHES

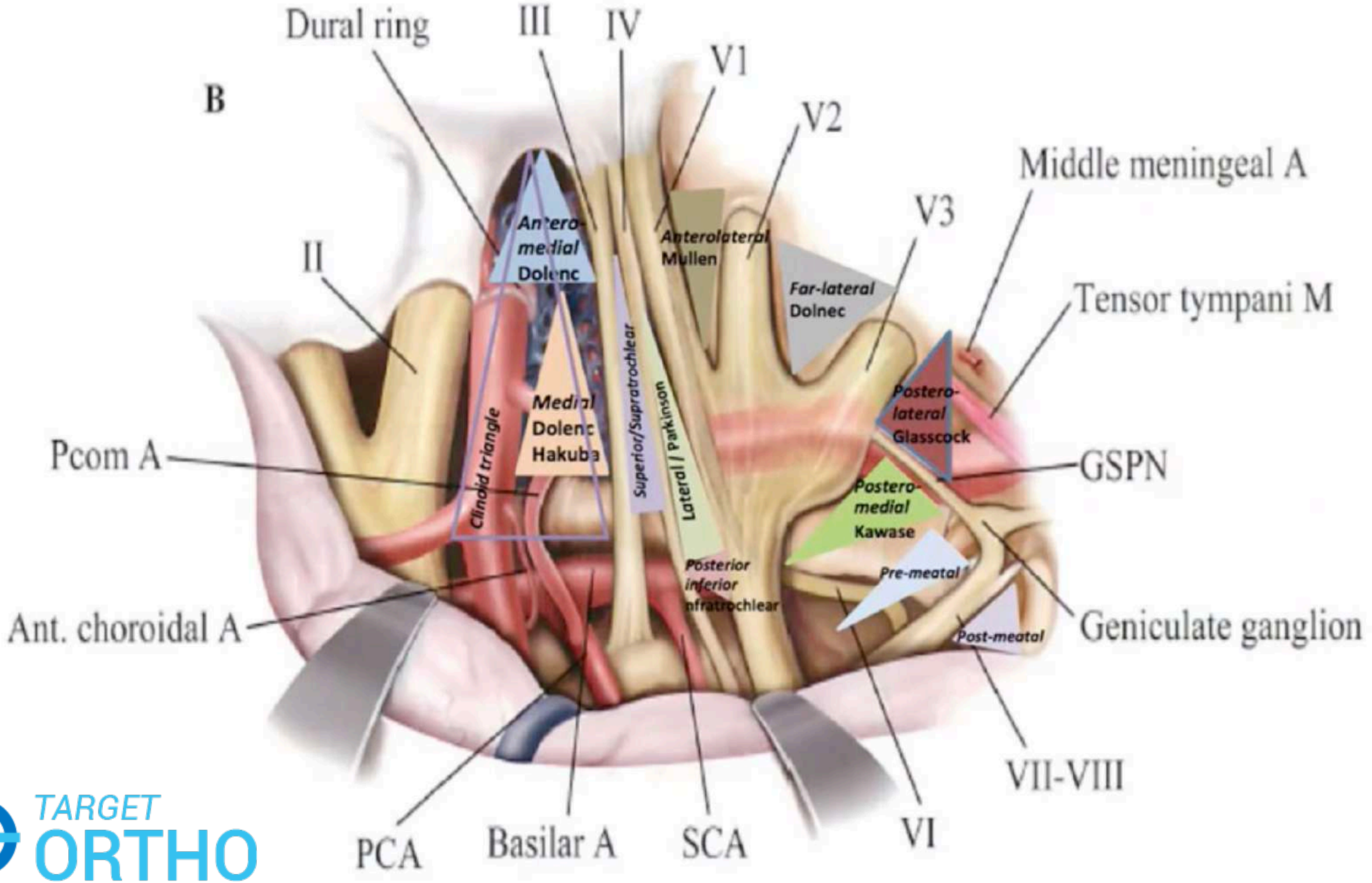
Indication:

- if embolisation fails
- embolisation is contraindicated



Parkinson – first to open Cavernous Sinus

The CS is opened between the III & IV nerves and this area is known as Parkinson's triangle



SRS

Indications:

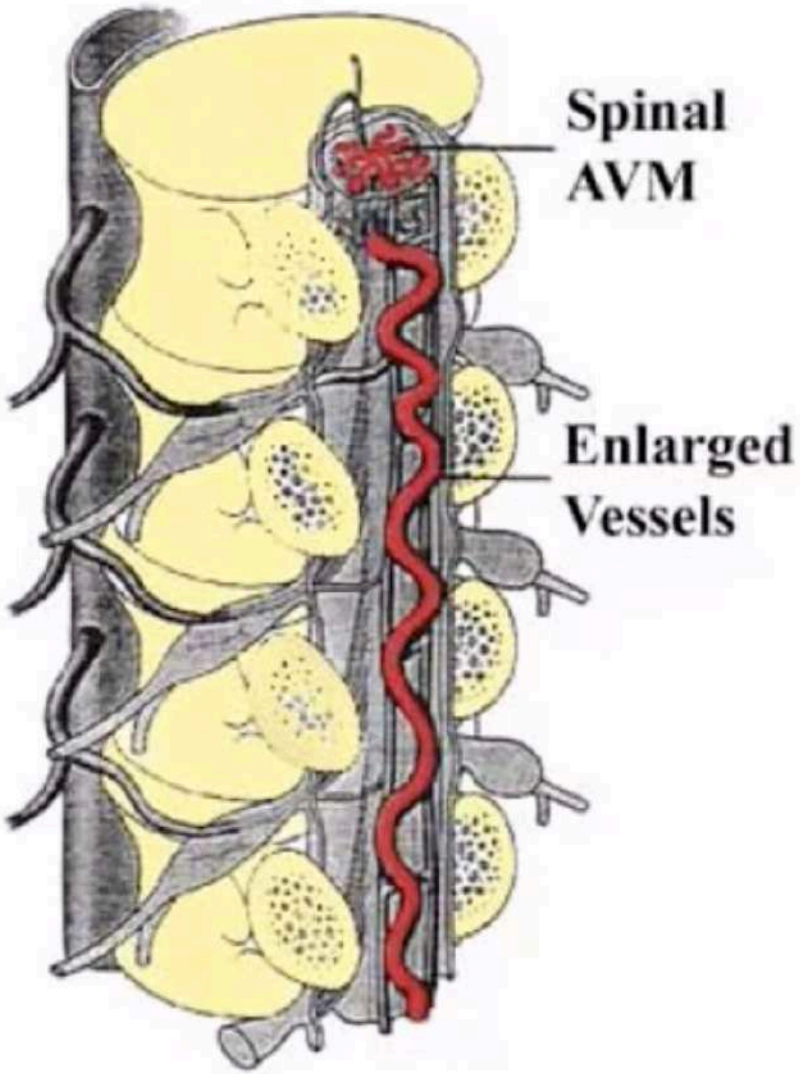
- Elderly
- Medical comorbities
- Failed endovascular therapy
- Low flow CCF

OUTCOMES

- **Mortality 1-2%**
- **Complications:**
  - Stroke due to ICA occlusion or balloon migration
  - Venous rupture during transvenous route
  - False aneurysms due to balloon shrinkage
  - Cranial nerve deficits

SPINAL VASCULAR MALFORMATIONS

- Aka spinal AVMs
- Incidence 4% of spinal masses
- 80% between 20 to 60 years

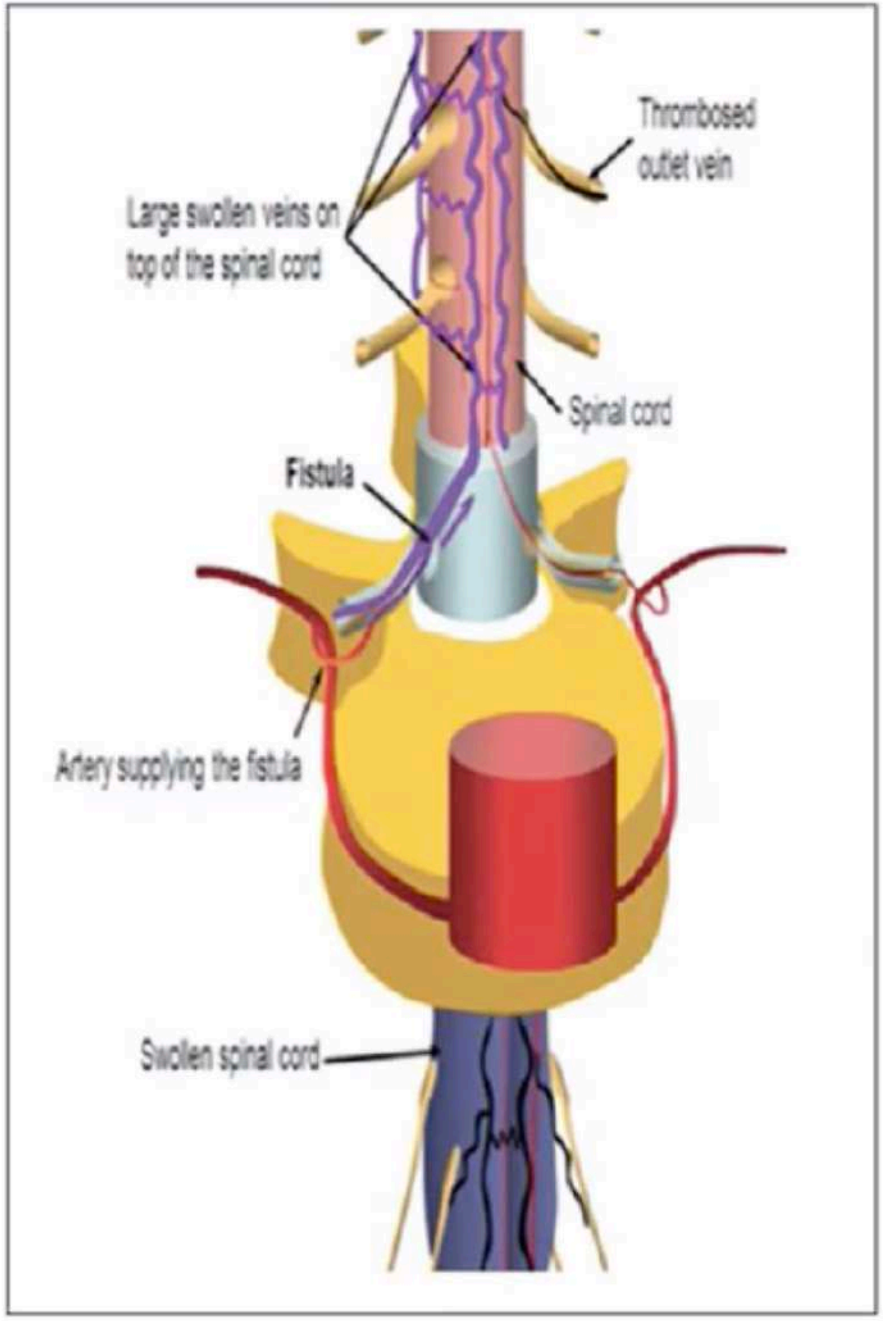


CLASSIFICATION

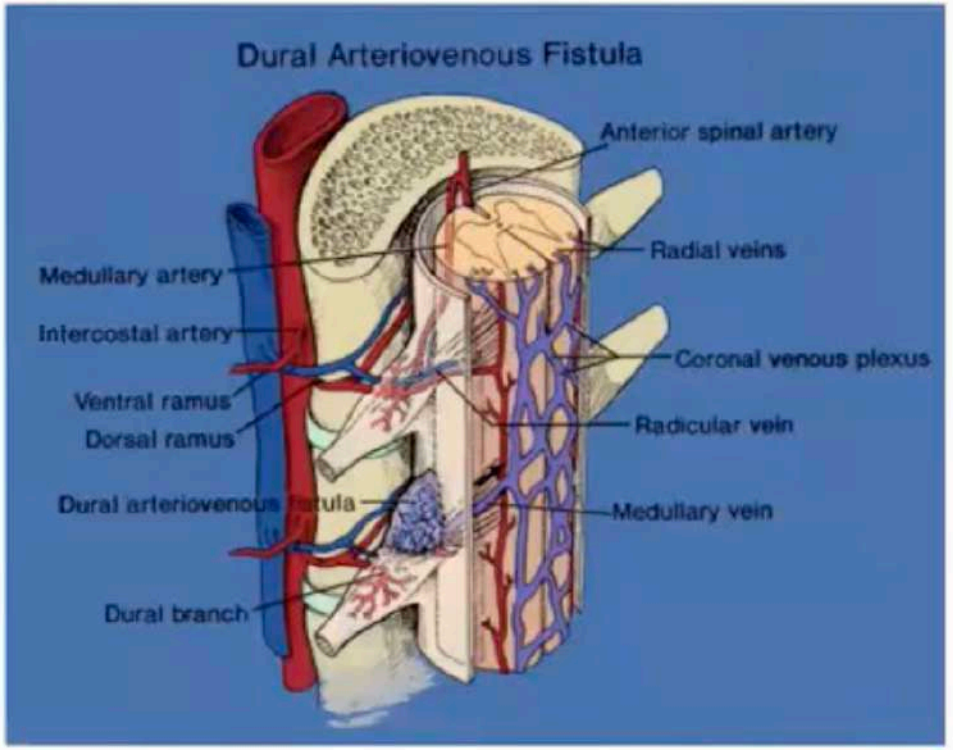
The American / English / French classification

Type I: DURAL AVM aka AVF

- Most common 80% of SVM
- Fed by radicular artery
- Forms AVF at the dural root sleeve (in the foramen)
- Drains into the spinal vein of the posterior cord
- Congestion of the cord
- Cord involvement distant to the fistula
- Slow flow



- Symptoms:
  - LBP
  - Progressive myeloradiculopathy
  - Cauda equina syndrome (due to venous congestion)
  - Urinary retention
  - Pain 35%
  - Rarely bleed
- Type IA: single arterial feeder
- Type IB: two or more arterial feeders

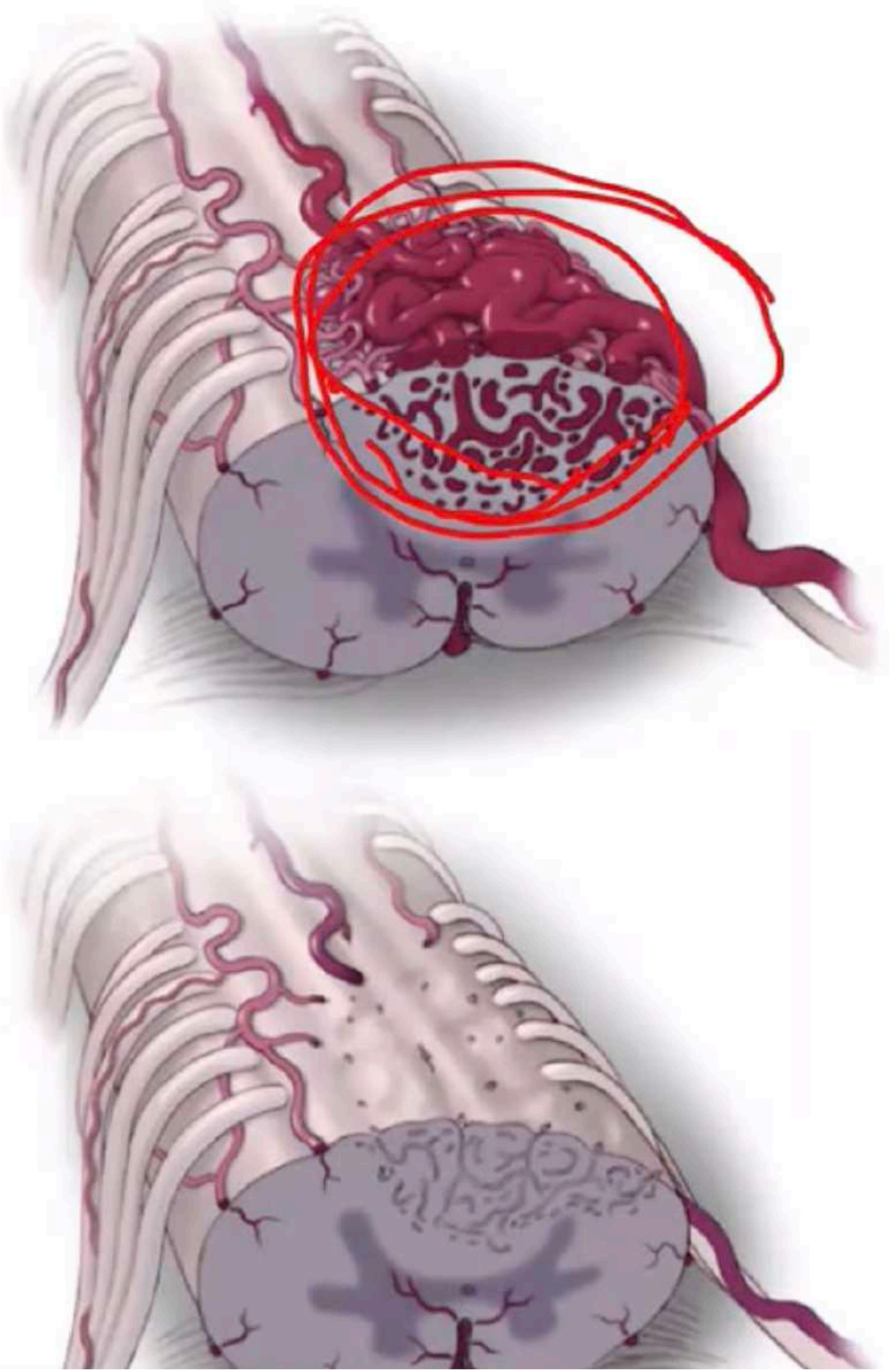


INTRADURAL AVMS

- **High flow**
- **75% acute symptoms, usually hemorrhage**

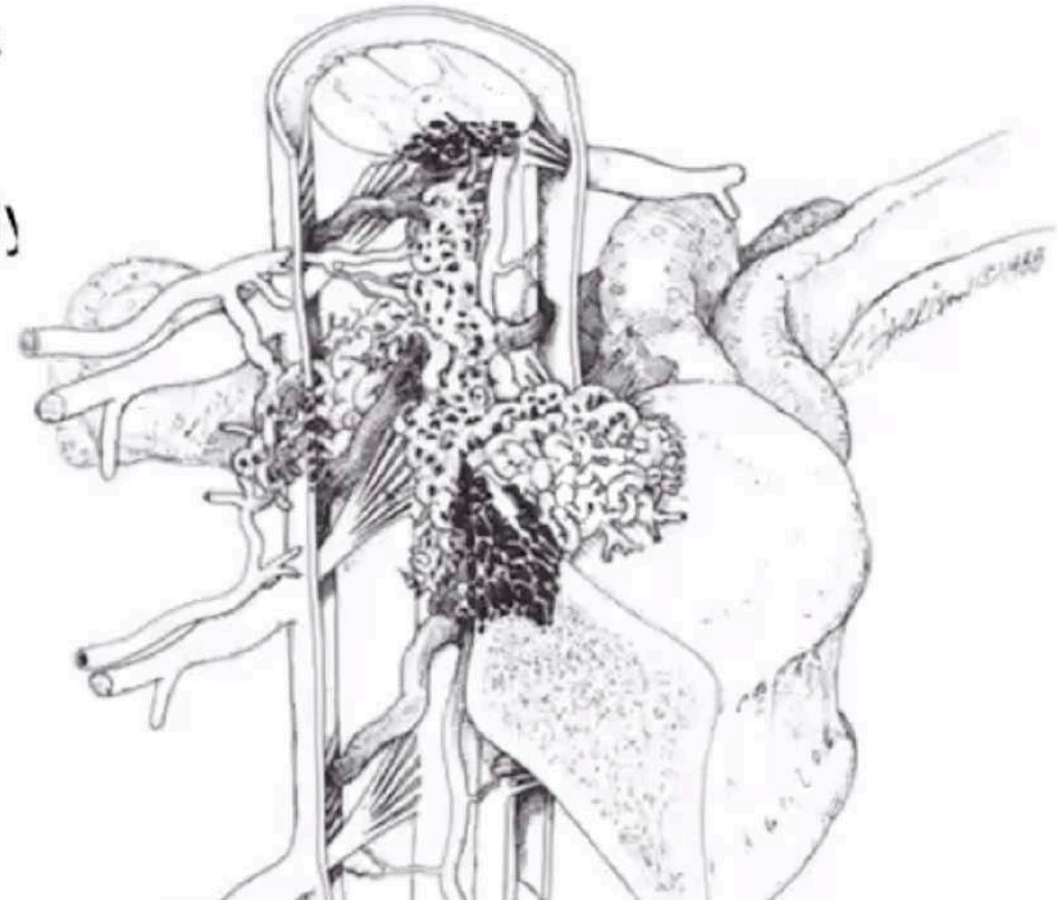
Type II: aka spinal glomus AVM

- Intramedullary
- True AVM of the cord
- 15-20% of SVMs
- Compact nidus
- Fed by medullary arteries
- Feeder artery aneurysms
- Worse prognosis than dural AVM



Type III: aka juvenile spinal AVM

- Enlarged glomus AVM
- Occupies the entire cross section
- Invades the vertebral body
- May cause scoiosis

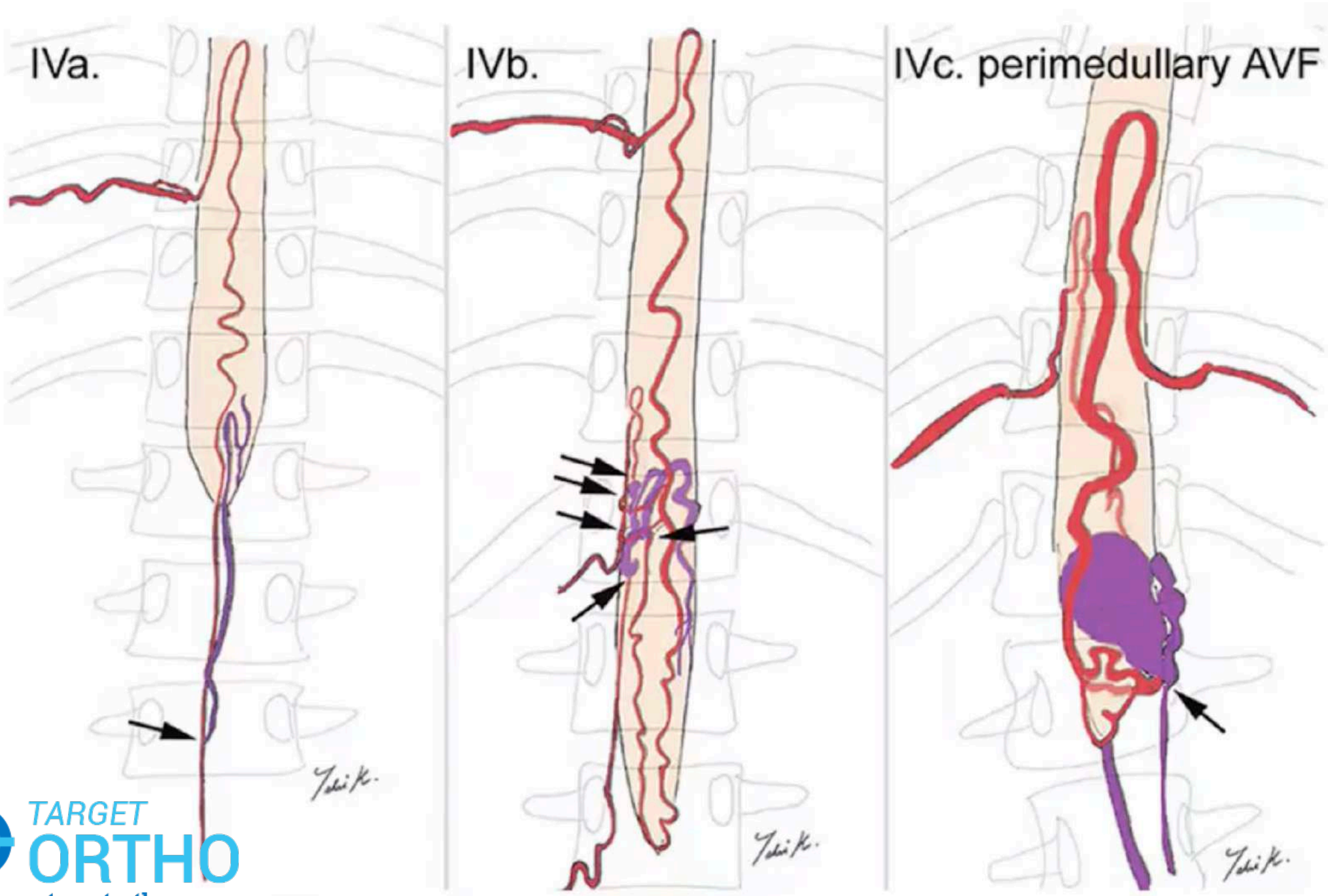


Type IV: intradural perimedullary AVM aka AVF

- Direct fistula
- Usually anterior spinal artery
- Often artery of Adamkiewicz
- Younger patients
- Catastrophic hemorrhage - SAH

Subtype	Arterial supply	AVF	Venous drainage
I	single (thin ASA)	single, small	slowly ascending perimedullary venous system
II	multiple (dilated ASA & PSA)	multiple, medium	
III		single, giant	giant venous ectasia, rapid metameric venous drainage

ASA = anterior spinal artery; AVF = arteriovenous fistula; PSA = posterior spinal artery

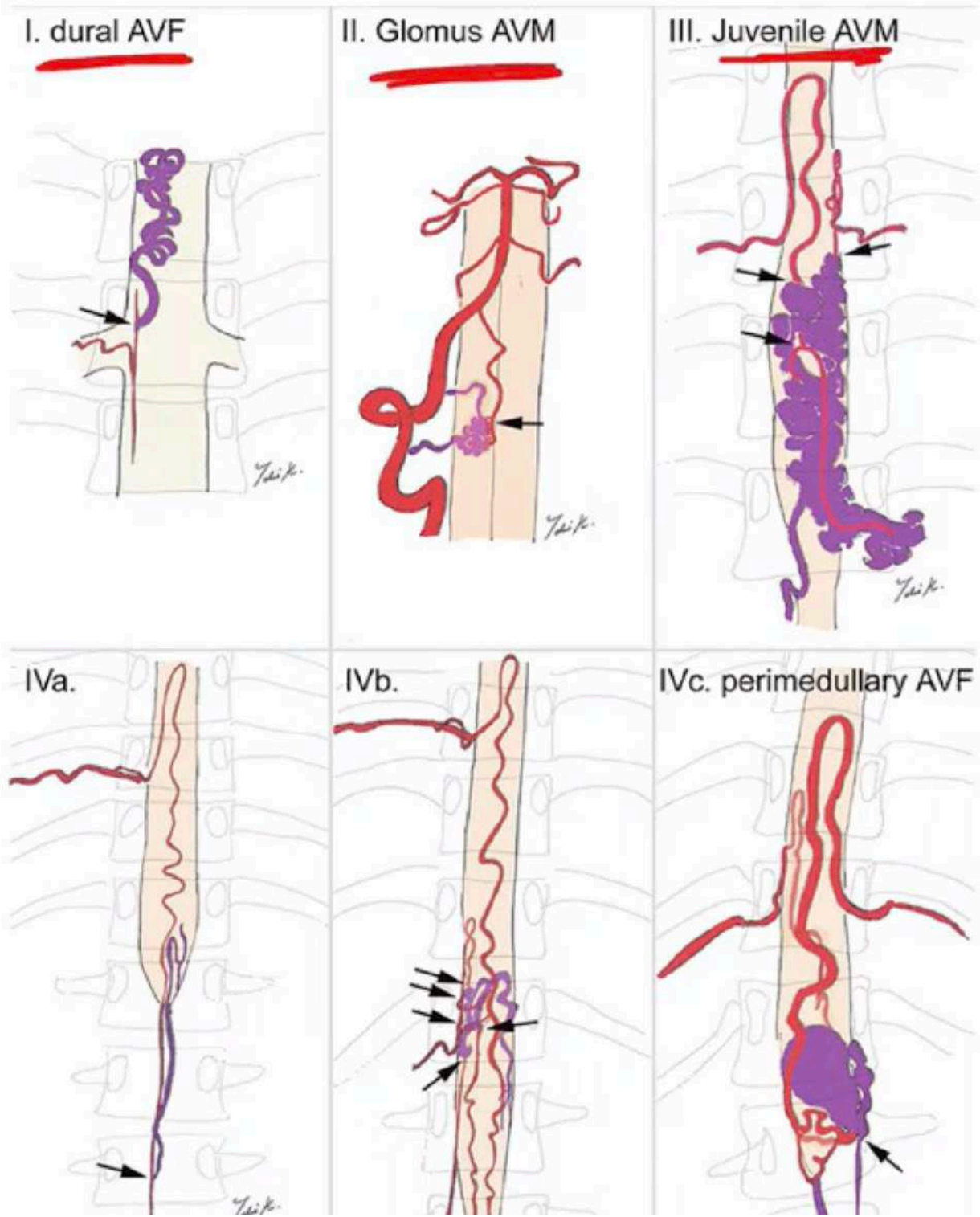


Miscellaneous spinal vascular lesions:

a) Spinal cord cavernomas

b) Spinal cord venous angiomas: rare, DSA negative

c) Vertebral body hemangiomas



Spetzler classification

1. **Neoplastic vascular lesions**
    1. Hemangioblastoma
    2. Cavernous malformation
2. **Spinal aneurysms (rare)**
3. **AV lesions**
    1. AVFs: extradural / intradural (dorsal/ventral)
    2. AVMs

PRESENTATION

- **85% progressive neuro deficit**
  - Back pain
  - Progressive sensory loss
- **10-20% sudden onset myelopathy (usually <30 yrs)**
  - Secondary to hemorrhage
- **Foix-Alajouanine syndrome**
  - Subacute neurotic myelopathy
  - Acute/subacute neurologic deterioration in SVM without hemorrhage
  - Spastic→ flaccid apraplegia
  - Ascending sensory level & loss of sphincter
  - Due to venous hypertension with secondary ischemia

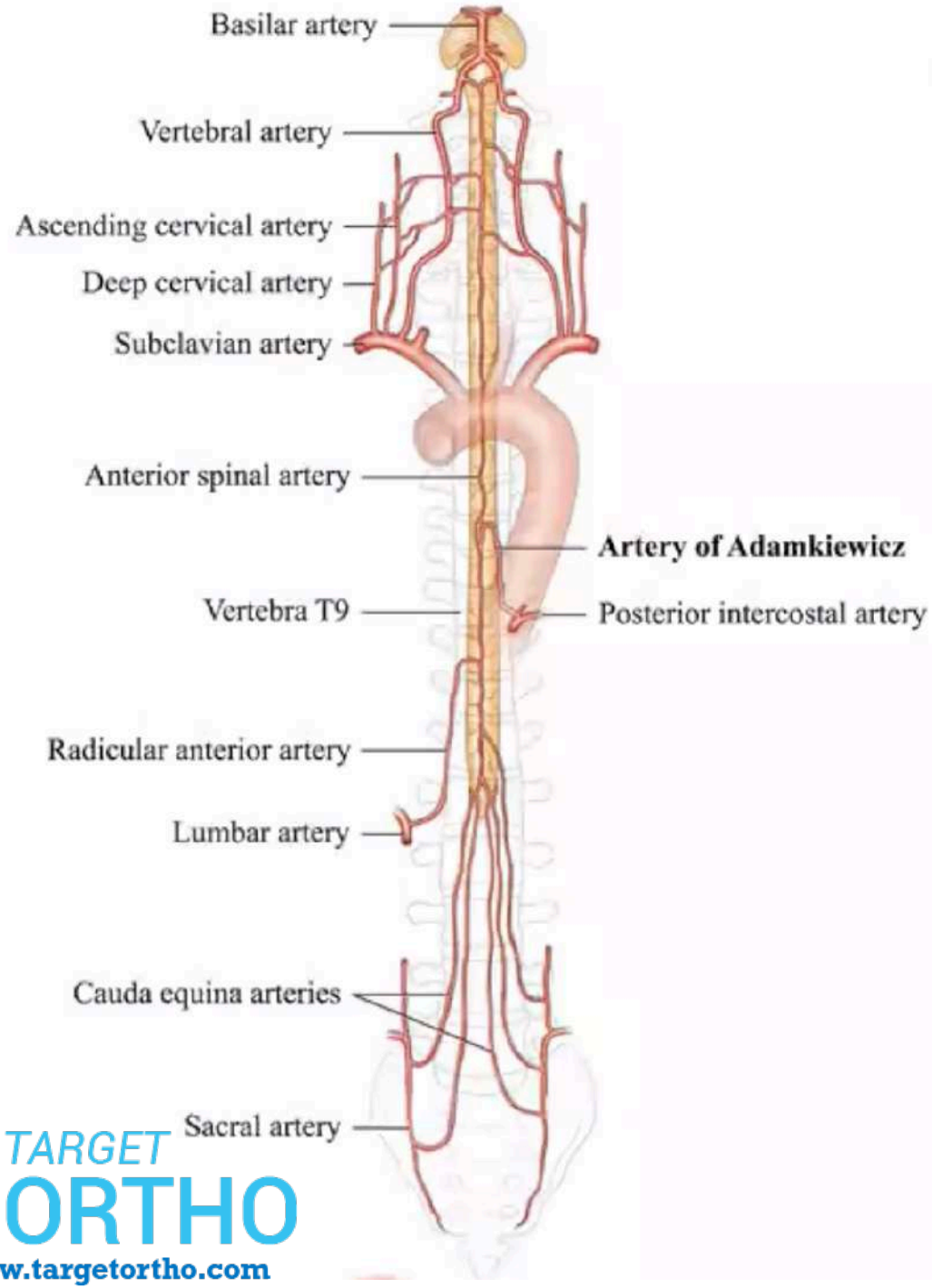
- Bruit over spine 2-3% cases
- Cutaneous angioma over back 3-25%

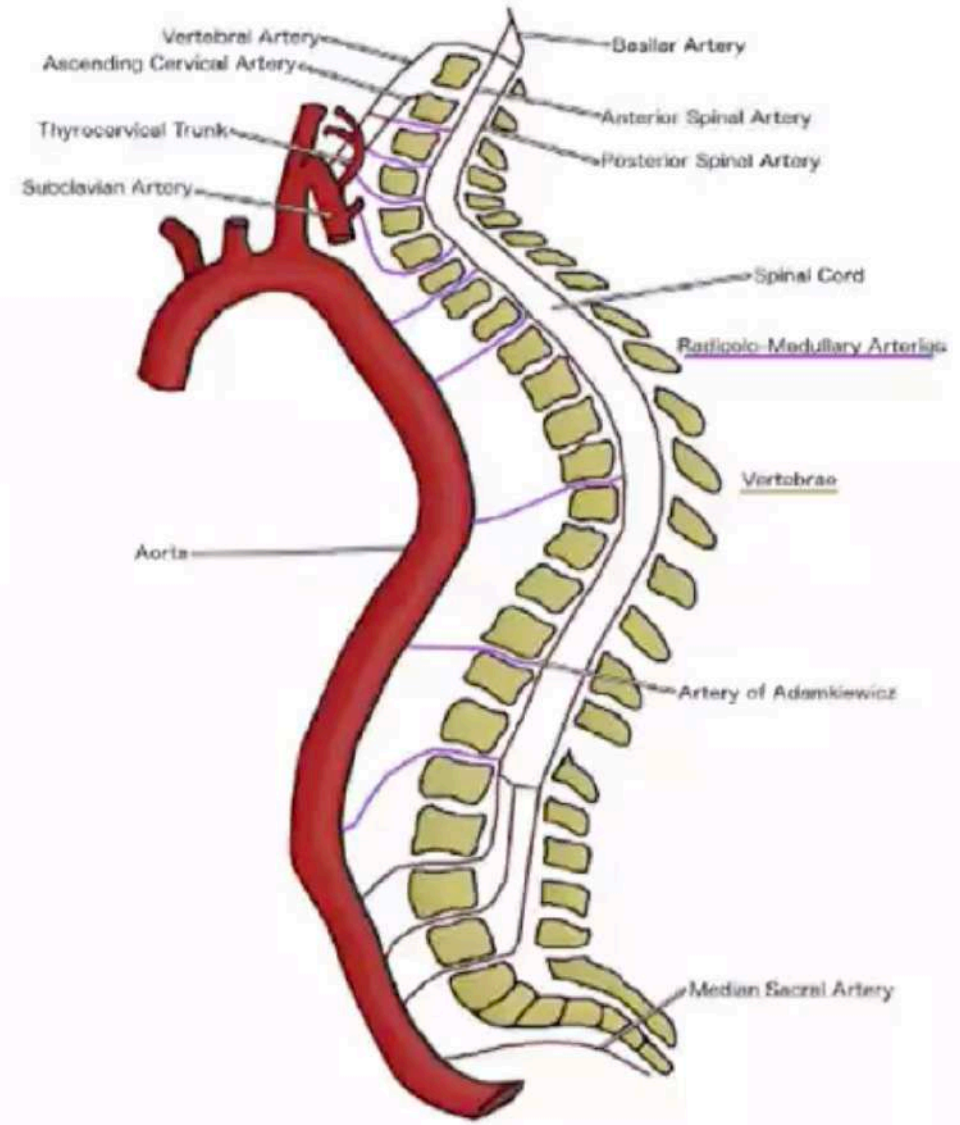
EVALUATION

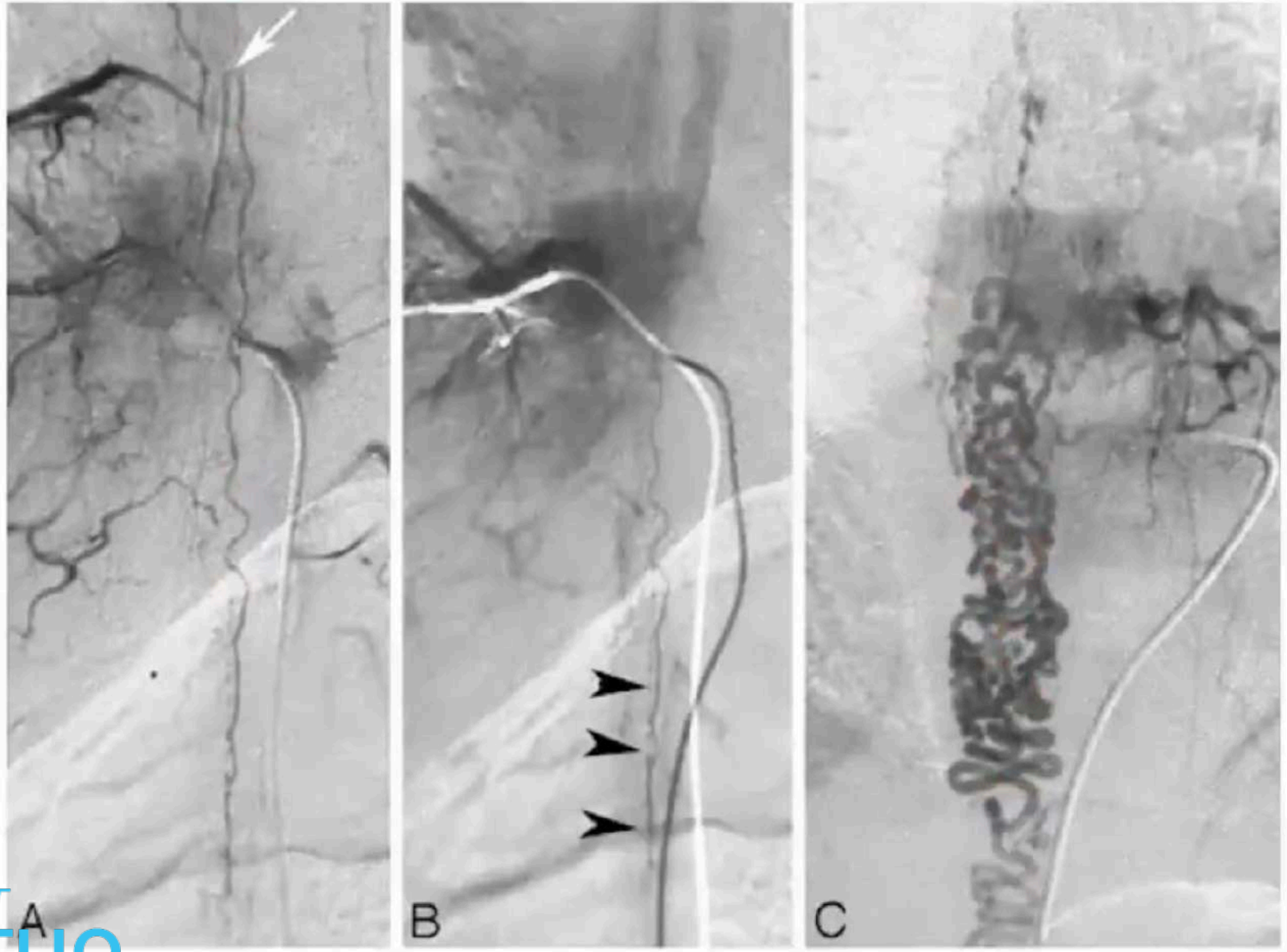
Spinal angiography

For type I dural AVMs, the DSA must encompass all feeders of neuroaxis, includes:

1. ICAs: artery of Bernasconi & Casinari
2. Every radicular artery incl artery of Adamkiewicz
3. Internal iliac artery: for sacral feeders





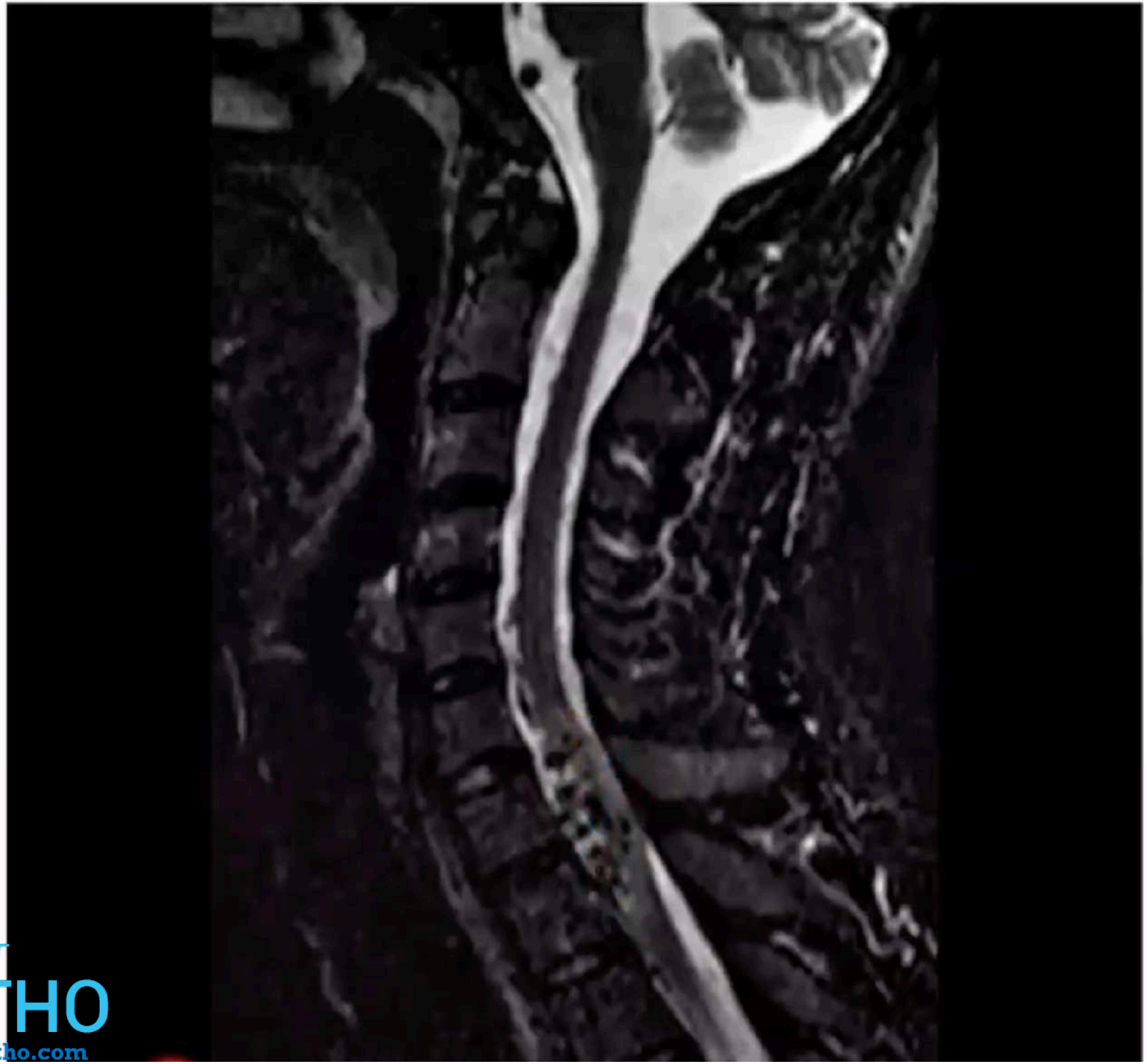


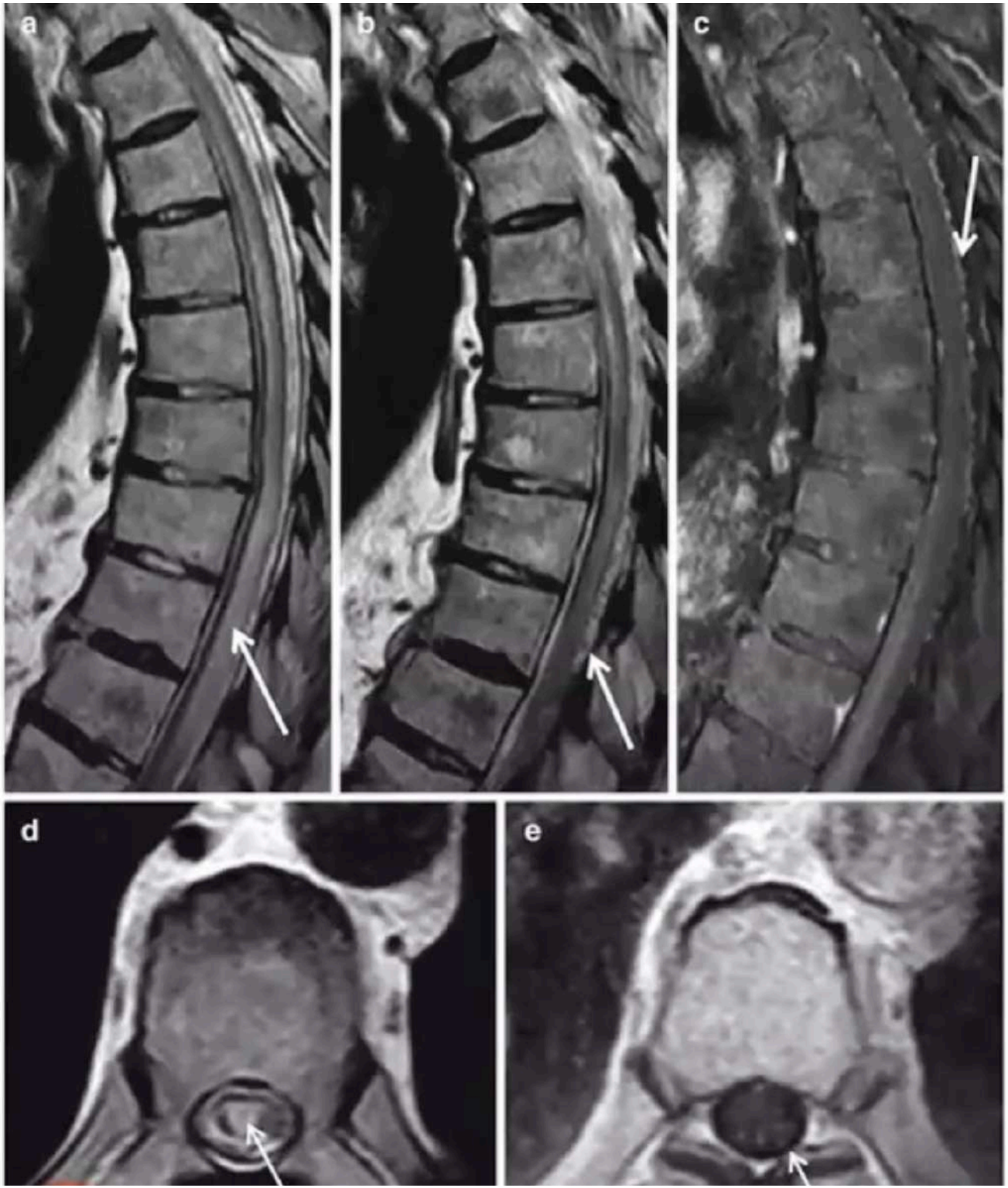
MRI

- Greater sensitivity and safety
- Inadequate for treatment planning
- 82% show extramedullary flow voids
- Negative MRI does not rule out the diagnosis

Myelography

- Intradural serpiginous filling defects
- Prone and supine
- Risk of rupture by the needle





TREATMENT

Type I (dural AVMs)

- Require treatment
- Endovascular techniques using glue
- Take down the proximal vein as well

Type II : spinal glomus AVM

- Endovascular procedures : embolisation esp type IIA (single feeder)
- Recurrence may be higher
- Surgery preferred for Type IIB (>=2 feeders)
- Strategy: same as IC AVMs except that parenchyma cannot be retracted
- Preserve arteries
- Intraop ICG angio helpful

Type III : Juvenile AVMs

- Natural history is better than any other treatment

Type IV : perimedullary fistulae

Merland's subclassification

Subtype	Diagnosis	Embolization	Surgery
Subtype I	difficult; ? reliability of MRI[a]; tomomyelography; angiotomomyelography	difficult	easy on filum terminale; difficult on conus medullaris
Subtype II	easy: MRI or myelography	incomplete occlusion	on posterolateral AVFs
Subtype III		effective	difficult, dangerous

[a]due to inaccuracy, do not delay angiogram to get MRA, etc.