Vascular Dissection (Carotid, Vertebral)

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PATHOPHYSIOLOGY

Dissection – blood extravasation between intima and media → lumen compromise
Dissecting aneurysm – blood dissection between media and adventitia → aneurysmal dilatation.
Pseudoaneurysm – rupture of adventitia → encapsulated extravascular hematoma.

- dissection can produce second intimal tear, allowing blood clot to reenter lumen → embolization.
- clot is absorbed within several weeks, and lumen usually returns to its normal size.
- most commonly involved – **ICA high in neck** (between C_2 and skull base) - carotid artery is stretched over transverse process of C_2 by any injury involving hyperextension and rotation of head and neck.
- less frequently involved – vertebrobasilar system (most mobile V_1 and V_3 segments), intracranial ICA, MCA.

ETIOLOGY

– **trauma** (blunt*, penetrating, angiography complication, or even trivial**; see also p. TrS21 >>), but may occur **spontaneously**.

*e.g. fall on popsicle in mouth, abuse with whiplash-shake injuries
**e.g. prolonged neck holding in eccentric position, chiropractic manipulation, coughing

- usually occur in **young** people (spontaneous dissection – in slightly older people).

RISK FACTORS

- nontraumatic (congenital / degenerative changes in vessel wall) - **fibromuscular dysplasia (!)** ("string of beads" on angio), Marfan's syndrome, Ehlers-Danlos type IV syndrome, pseudoxanthoma elasticum, atherosclerosis, migraine, pronounced vessel tortuosity, moyamoya, cystic medial degeneration, pharyngeal infections, α_1-antitrypsin deficiency, luetic arteritis (associated with 60% of dissections before 1950)
- traumatic:
  1) skull fractures – involving carotid canal, Le Fort II-III
  2) near hanging, esp. with anoxic brain injury
  3) clothesline-type neck injuries
  4) cervical fractures (vertebral body, involving foramen transversarium, any fracture of C1-3)
  5) cervical ligamentous injuries and subluxations

CLINICAL FEATURES

1. **Pain** (important symptom that helps to diagnose this cause of brain ischemia!!!):
   carotid dissections → ipsilateral throbbing headache (forehead, eye, face), intense local sharp pain in neck (carotidynia).
   vertebral dissections → pain in occiput, posterior neck.
2. **Ischemia** – TIAs (due to luminal compromise), stroke (due to embolization within first few days).
   Arterial dissection is important cause of ischemic strokes in young people!
   N.B. ischemia may be delayed by > 1 hour or even by > 24 hours after dissection!
   ICA injuries often give TIAs before stroke (VA injuries do not)
   VA injury strokes happen ≈ 4 days after injury (range 8 hrs – 12 days)
   Published data indicate that VA injury may have a lower risk of stroke than ICA injury.
3. **SAH** – more common with intracranial VA dissections (thin media and adventitia).
4. Other associated symptoms:
   1) Horner syndrome (in carotid dissection)
      N.B. in ICA dissection Horner syndrome is ***incomplete*** (oculosympathetic palsy) – ptosis and miosis without anhidrosis - sympathetic fibers to face sweat glands and blood vessels travel along ECA (esp. to lower face) see p. Eye19 >>
   2) tenderness over neck
   3) (self-audible) bruit in patient < 50 yo (but auscultation is poor screening tool)
   4) pulsatile tinnitus.

DIAGNOSIS

CT / **MRI** can directly visualize intramural bleeding and expansion.

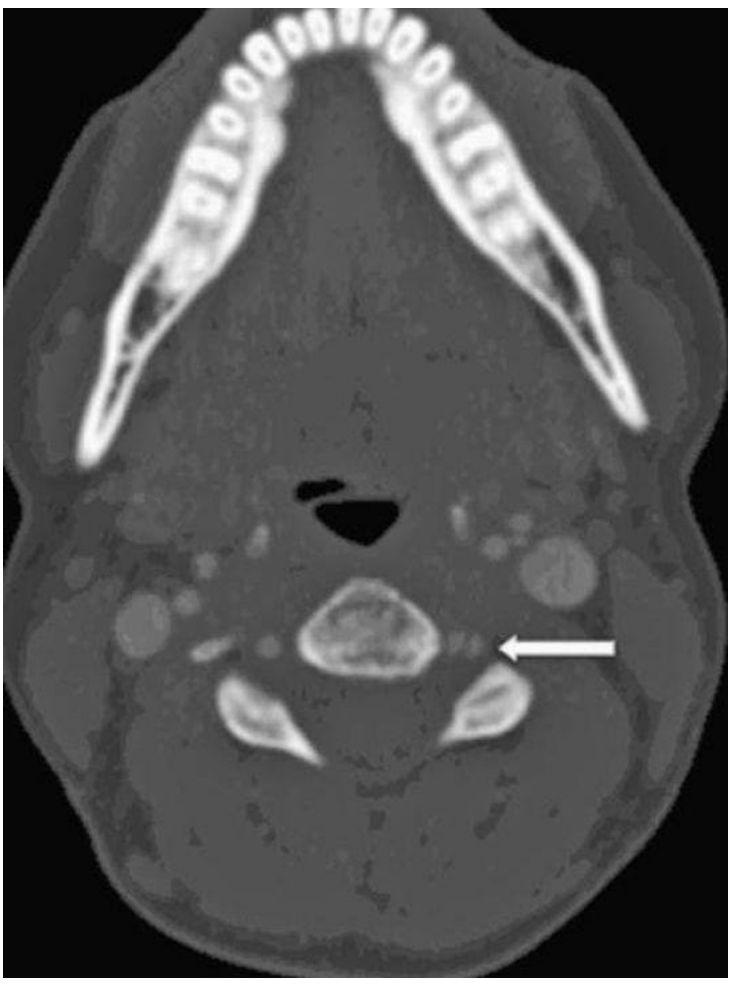
- MRI *in acute stage* - intramural hematoma is isointense to muscle - difficult to detect.
- MRI *after few days* - rim of high signal (subacute intramural hematoma) expanding outer diameter of artery and narrowing its lumen.
  Crescent sign: bright T2 signal in ICA wall on axial images (hematoma in vessel wall).

CTA – most reliable noninvasive diagnosis!

- **CTA** on scanners with ≥ 16 detectors (16-slice multidetector CTA) have accuracy near 99% & equivalent predictive value to DSA.
  Modern CTA = DSA
- **intimal flap** (at proximal end of dissection) ± **double lumen** (pathognomonic - retention of contrast within false lumen well into venous phase).
- **gradually tapering** leading to *severe narrowing* ("string sign") or *total occlusion*
- starts > 2 cm distal to ICA origin.
  N.B. cervical ICA dissection typically spares carotid bulb whereas cervical ICA atherosclerosis tends to involve bulb
- high **cervical VA** dissections can readily propagate intracranially through foramen magnum (vs. **cervical ICA** dissections - tend not to propagate intracranially through carotid canal).
- characteristic of arterial dissections is that they often change configuration on repeat imaging (some resolve, and some worsen).

Any *trauma patient* having focal neurological deficits (esp. with Horner's syndrome) that cannot be explained from imaging studies → early CTA to diagnose carotid artery dissection!

Left vertebral artery intimal flap (*arrow*) secondary to vertebral artery dissection:



Ultrasound - reliable screening tool:

B-mode ultrasound - tapering of ICA lumen, irregular membrane crossing lumen, true and false lumens.

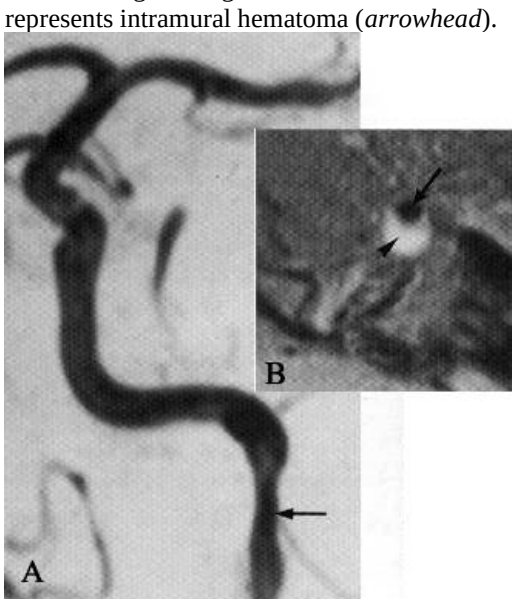
Duplex scans - decreased pulsatility, intravascular abnormal echoes, decreased flow.

TCD - effect of neck pathology on poststenotic intracranial circulation:
Diminished intracranial velocities in young patients who have normal ICA bifurcations → diagnosis of dissection is quite likely.

MRA and ultrasound are not considered adequate for BCVI screening.

MRA - reliable noninvasive diagnosis for extracranial ICA.

ICA dissection (3D TOF MRA):
A. Focal narrowing as ICA enters skull base (*arrow*).
B. Axial image through that level - flow void in residual vessel lumen (*arrow*) and high signal crescent, which represents intramural hematoma (*arrowhead*).



Conventional **angiography** (more useful for VA) – indicated if CTA does not resolve clinical suspicion.

GRADES

Denver grading scale

Grade	Description	Risk of stroke with ICA injury*	Fate
I	luminal irregularity with < 25% stenosis	3%	70% heal 25% persist 5% progress to ↑ grade
II	≥ 25% luminal stenosis or intraluminal thrombus or raised intimal flap	11%	70% progress to ↑ grade
III	pseudoaneurysm	44%	most persist
IV	occlusion	lethal	most persist
V	transection with free extravasation	lethal	

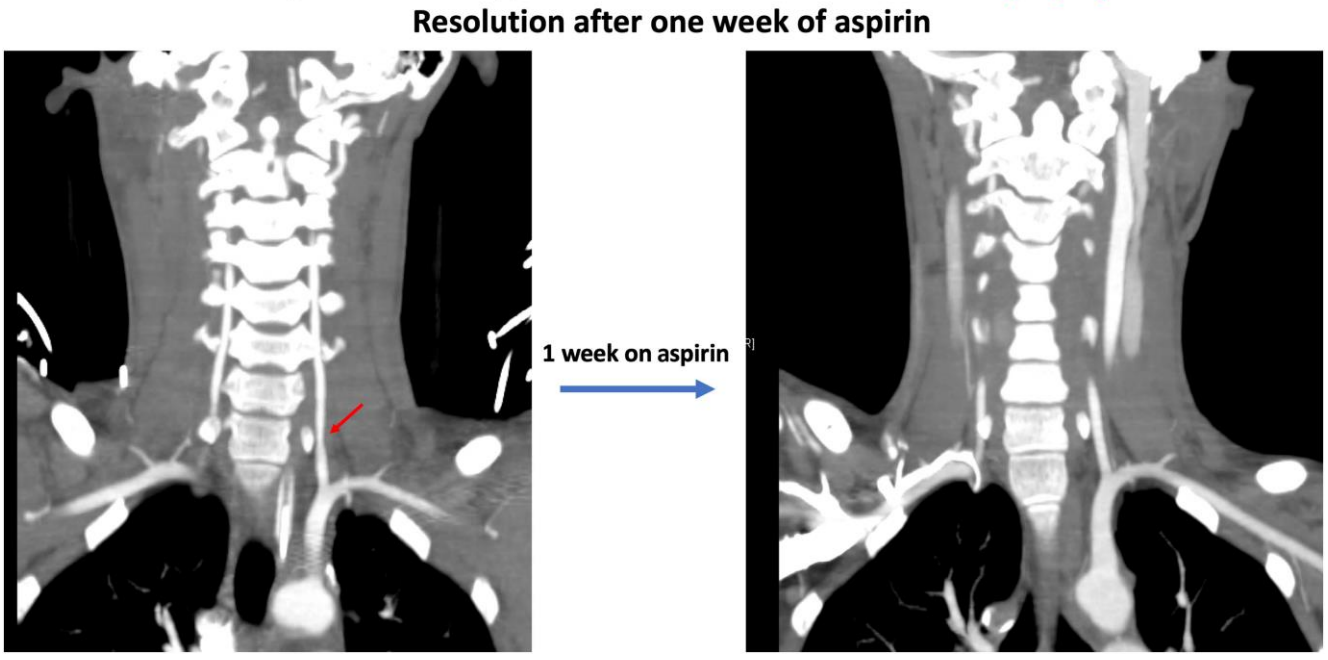
*risk of stroke increases with increasing grade for ICA injuries;
not true for VA injuries – depends on status of contralateral VA:

grade I – many cases probably just vasospasm or compression with external hematoma and not true intimal injury.

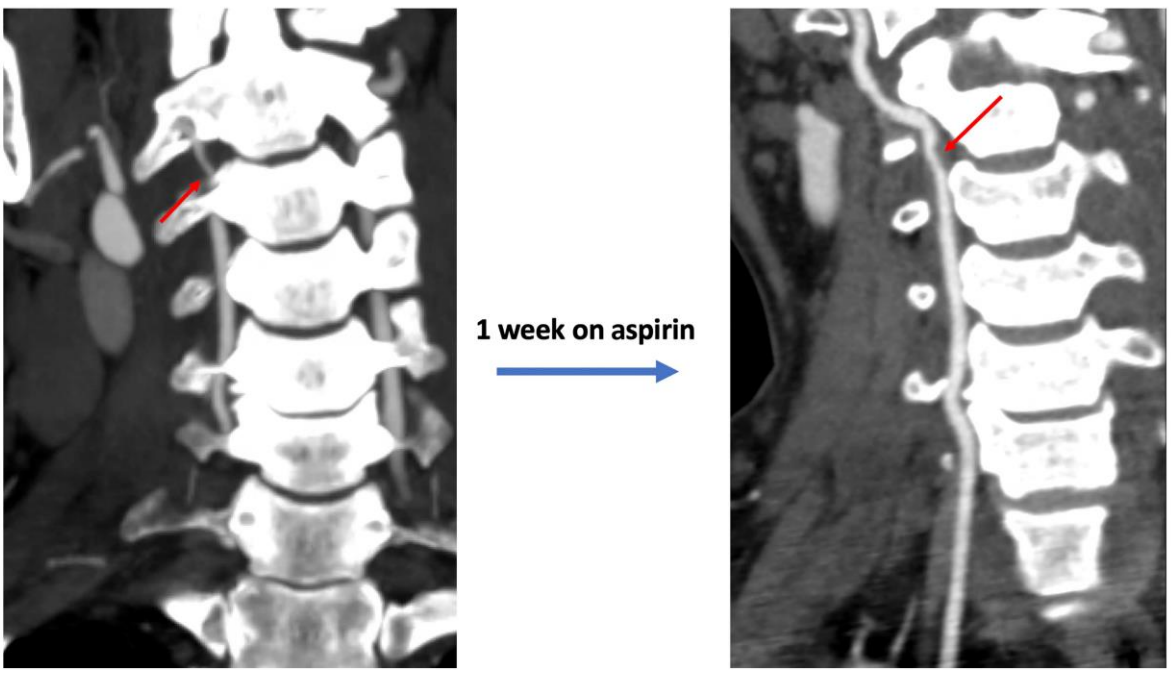
grade IV, if tolerated, has low risk of embolic stroke as flow supplied from contralateral side becomes retrograde to fill PICA.

grade II-III – risk of stroke is higher

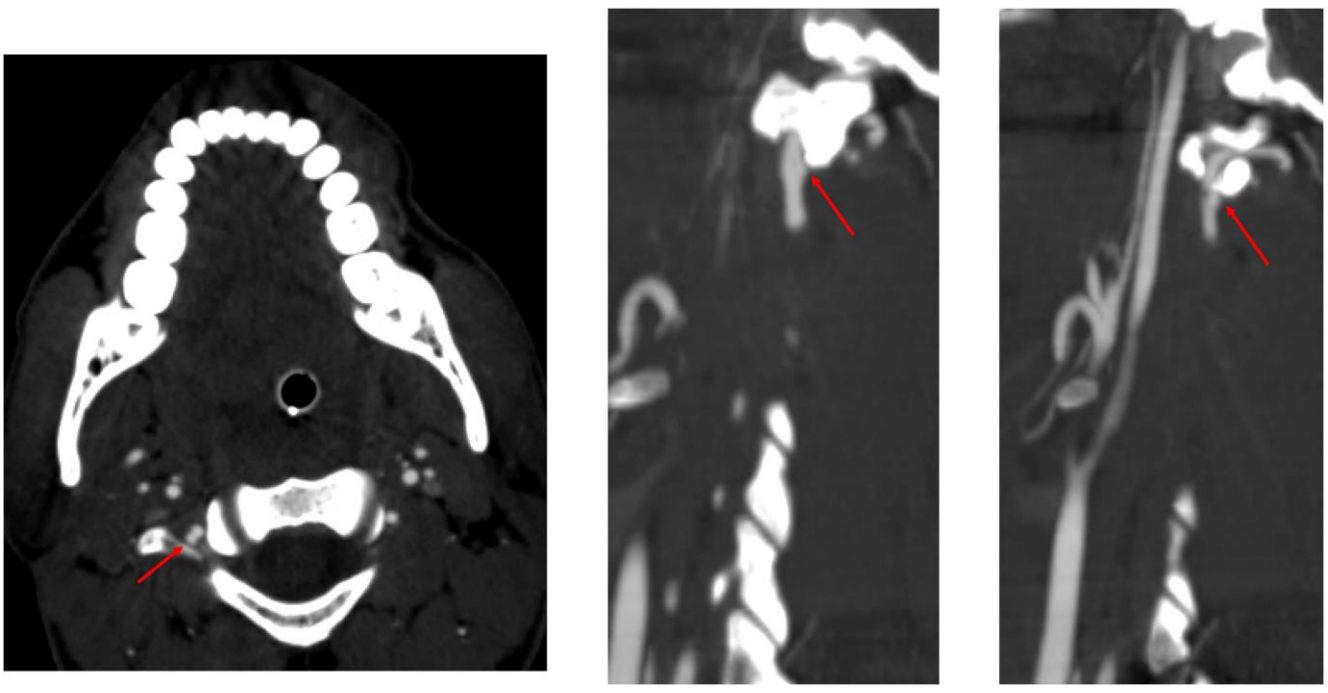
19-year-old man s/p MVC. Grade 1 left vertebral artery injury
Resolution after one week of aspirin



40-year-old women s/p auto-ped. Grade 2 right vertebral artery injury at C2-C3. Improvement after one week of aspirin



21-year-old man s/p MVC. Grade 3 right vertebral artery injury



TREATMENT

95% treatment is medical; DSA indication – need for stent:

a) flow limiting stenosis
b) crescendo strokes
c) growing aneurysm

N.B. single antiplatelet agent (not DAP)

Grade specific therapy (imaging = angiogram or 16MD-CTA) – for ICA*

Grade I & II – ASPIRIN → CTA at 6 weeks

Grade III – HEPARIN → repeat imaging in 7–10 days:

a) healed: stop anticoagulation
b) nonhealed: switch heparin to ASPIRIN / ANTICOAGULATION (may consider stenting for severe narrowing or expanding pseudoaneurysm) → repeat imaging in 3-6 months:
   - healed: stop Aspirin.
   - nonhealed: lifelong (or just additional 6 months) ASPIRIN.

Grade IV - **endovascular occlusion** to prevent embolization.

Grade V (highly lethal):

a) accessible lesions → urgent **surgical repair** (anecdotal)
b) inaccessible lesions (the majority):
   - incomplete transection → **endovascular stenting** + antithrombotic drug
   - complete transections → **ligate** (or **endovascular occlusion**)

*no specific guidelines for VA (from no treatment to endovascular therapy); recommendation: treat **V1-3 injuries** with ASPIRIN (for grades II-III – higher risk of stroke – may consider HEPARIN) → CTA at 6 weeks (expert opinions range from 1 week to no F/U at all) – mainly to detect intraluminal thrombus and not worsening of grade; some experts only follow grade II-III injuries (other grades - just treat with 12 weeks of ASPIRIN).

N.B. **V4 injuries** carry risk of SAH – ASPIRIN / ANTICOAGULATION may increase this risk further.

N.B. anticoagulation has a risk of dissection extension and SAH, or infarct hemorrhagic transformation.

Other sources of data

Most extracranial dissections ***heal spontaneously*** – treated medically!

- if *complete occlusion* has occurred, arteries often do not recanalize.
- arteries that *retain some residual lumen* invariably heal and become normal.
- indications for intervention (stenting)
  a) dissections that *progress angiographically*
  b) *flow-limiting stenosis* - brain perfusion asymmetry on angio
  c) *persistent symptoms* in spite of adequate medical therapy

Most intradural dissections (and all with **SAH***) – treated with intervention.

*high rebleeding rate

ANTICOAGULANTS / **ANTIPLATELETS** shortly after dissection should prevent stroke;

N.B. risk of embolization exists *only during acute period*! TIAs often precede infarction, leaving time for therapeutic intervention!

- HEPARIN → WARFARIN is continued until lumen is not severely compromised (e.g. for 3-24 months; target INR 2-3) → **ANTIPLATELET AGENTS** for at least 2 years.
- anticoagulation is contraindicated in intracranial dissections complicated by SAH.

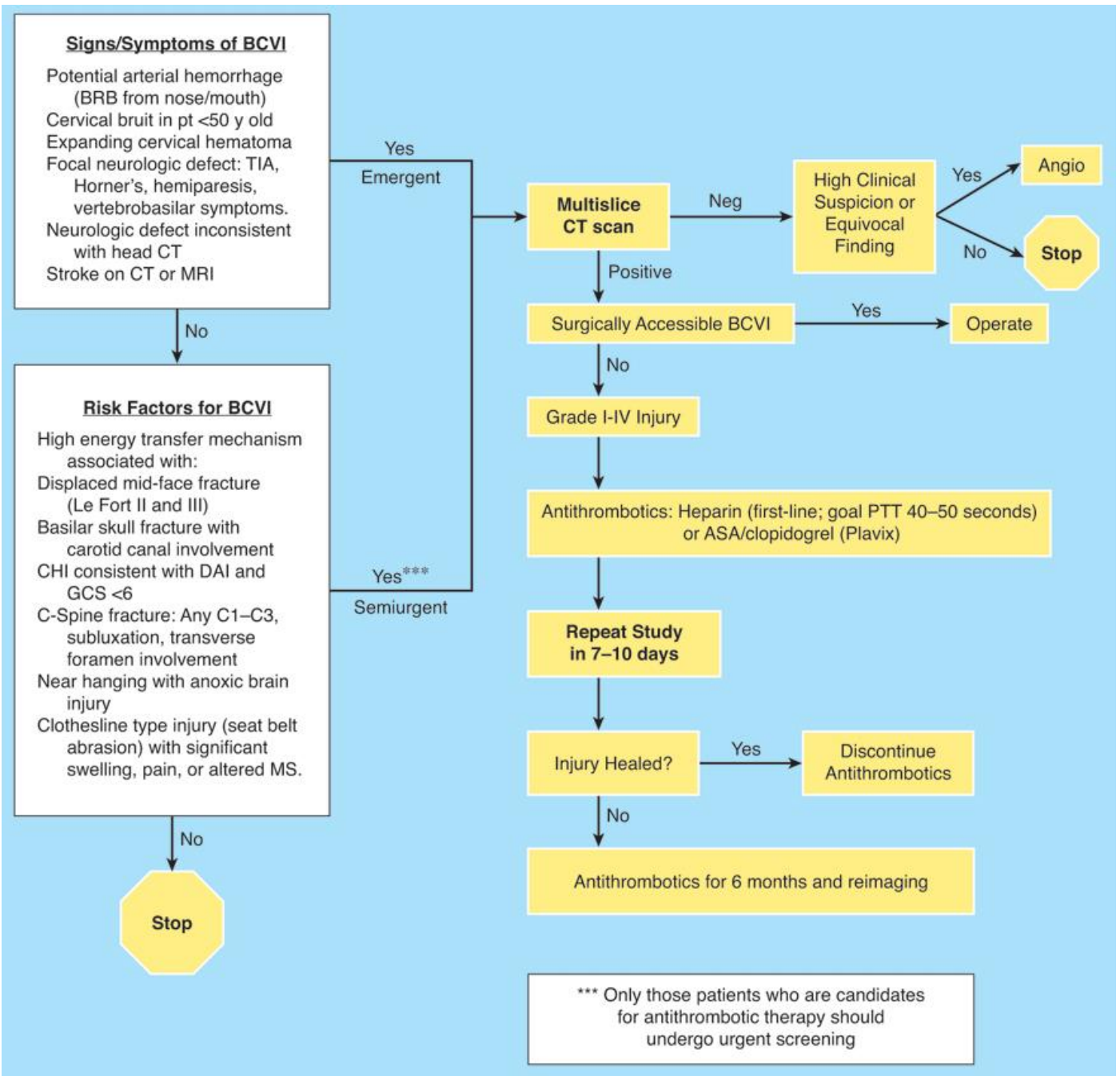
SURGICAL REPAIR indications:

a) SAH
b) persistent high-grade (s. flow-limiting) stenosis

- location high in neck makes surgical carotid repair difficult.

Endovascular stenting is a modern option.

- stenting in mobile neck carries the risk of stent fracture (cf. intracranial stenting or stenting VA ostia – much less vessel movement); solution might be a softer stent (e.g. pipeline).
- carotid dissections:
  with **complete occlusion** - observation (continue ASA for life)
  with slight contrast wisp (**high grade stenosis**):
  a. high intracranial - do not touch it if brain is well perfused (risk of even slightest dissection extension and may occlude PComA ostia --> massive stroke); if brain hypoperfused - document it with pCT and proceed with stenting.
  b. low in neck (proximal) - OK to stent (e.g. pipeline).



STUDIES

Blunt Vertebral Artery Injuries

Hussein A. Zeineddine et al. Blunt Traumatic Vertebral Artery Injuries: Incidence, Therapeutic Management, and Outcomes. Neurosurgery 90:399–406, 2022

- 156 patients
- 135/156 were treated with aspirin alone, 3 with anticoagulation therapy, and 18 did not receive treatment.
- 25% began treatment within 24 hours, 30% between 24 and 48 hours and 32.7% >48 hours from admission.
  Therapy initiation was delayed in the presence of other injuries (ie, intracranial or systemic bleeding) and/or a need for other surgical interventions.
  We did not delay aspirin in relation to need for spine surgery!
  Aspirin was delayed for 24 hours after stable head CT in significant intracranial hemorrhage
- *3 strokes were detected within 24 hours of admission and before treatment initiation* (1 patient with grade II, 1 patient with grade III, 1 patient with grade IV), no other strokes were detected during the hospitalization (incl. no strokes in grade I injuries)
- recommendations:
  - aspirin for 12 weeks for V1-3 grade I and IV injuries (for grades II-III, bilateral injuries consider anticoagulation if symptomatic or intraluminal thrombus).
  - repeat CTA at 1 week and only for grades II-III.

BIBLIOGRAPHY for ch. "Neurovascular Disorders" → follow this LINK >>