

OCCLUSIVE CEREBROVASCULAR DISEASE

NEUROSURGERY LECTURE



DR HIMANSHU CHAMPANERI

CONSULTANT NEUROSURGEON

MEDANTA HOSPITAL GURUGRAM

STROKE

- **TIA**
 - **Transient neuronal dysfunction secondary to focal ischemia without infarction**
 - **10-15% patients with TIA have stroke within 3 months, 50% within 48 hours**
- **Stroke**
 - **Permanent death of neurons caused by inadequate perfusion**

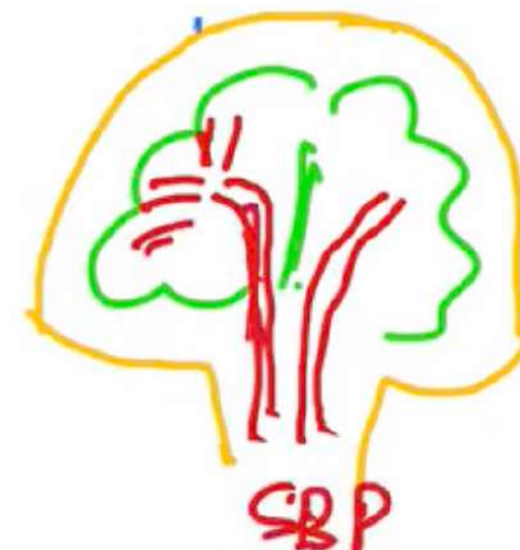
CEREBRAL BLOOD FLOW

6-10 min

CBF (ml per 100 gm tissue/min)	Condition
>60 (approx)	hyperemia (CBF > tissue demand)
45-60	normal brain at rest
75-80	<u>gray matter</u>
20-30	white matter
<20: Ischemia	
16-18	EEG becomes flatline
15	physiologic paralysis
12	brainstem auditory evoked response (BAER) changes
10	alterations in cell membrane transport (cell death; stroke)

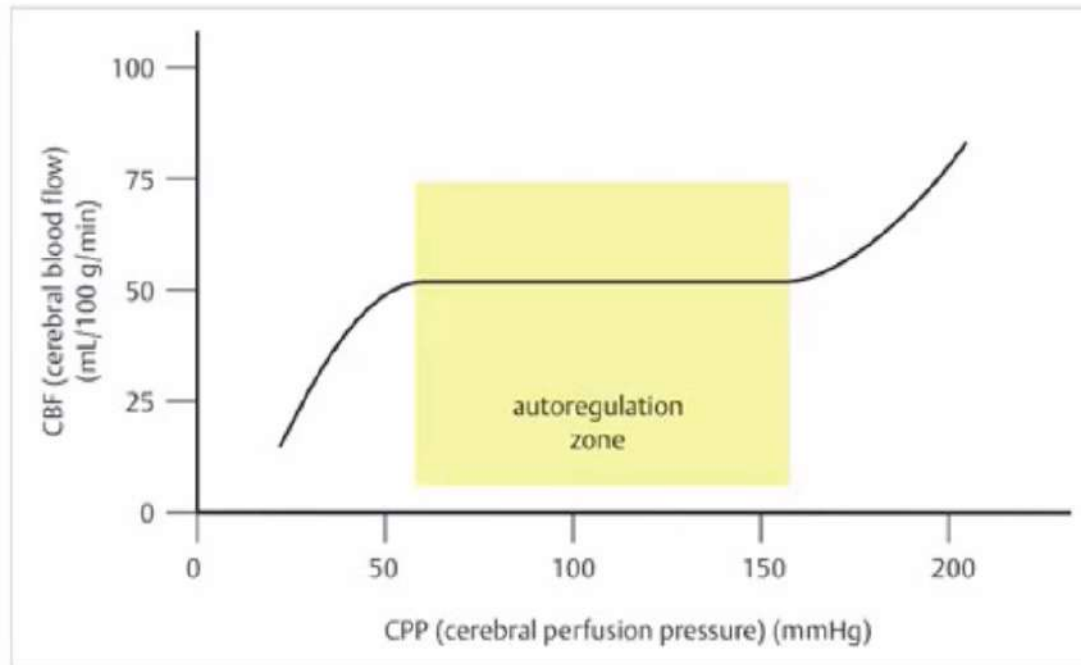
- CBF < 20 is associated with ischemia
- CBF is related to blood pressure as :

$$CBF = \frac{CPP}{CVR} = \frac{MAP - ICP}{CVR}$$



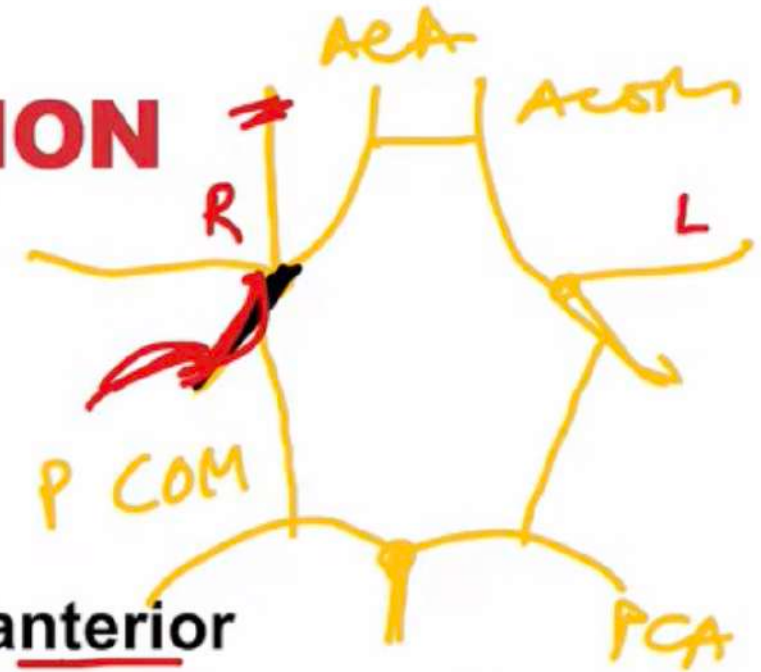
CEREBRAL AUTOREGULATION

- In the range of CPP = 50–150 mm Hg, the CVR (the resistance of the cerebral vascular bed to blood flow) of normal brain tissue varies linearly to maintain an almost constant CBF.
- This phenomenon is called (cerebral) autoregulation



COLLATERAL CIRCULATION

ICA stenosis/occlusion



1. flow through the circle of Willis

a) from contralateral ICA through anterior communicating a. b) from forward flow through the ipsilateral posterior communicating a.

2. retrograde flow through ophthalmic a. parasitizing blood from both ECAs via:

a) facial a. → angular a. → dorsal nasal a. & medial palpebral a.

b) maxillary a. • middle meningeal a. → lacrimal a. • vidian a. (a. of the pterygoid canal)

c) transverse facial a. → lateral palpebral a. d)
superficial temporal a. → supraorbital a.

3. proximal maxillary a. → anterior tympanic a. →
caroticotympanic branch of ICA

4. cortical-cortical anastomoses

5. dural-leptomeningeal anastomoses

Vertebrobasilar stenosis/occlusion

- basilar artery occlusion

1. Posterior communicating artery

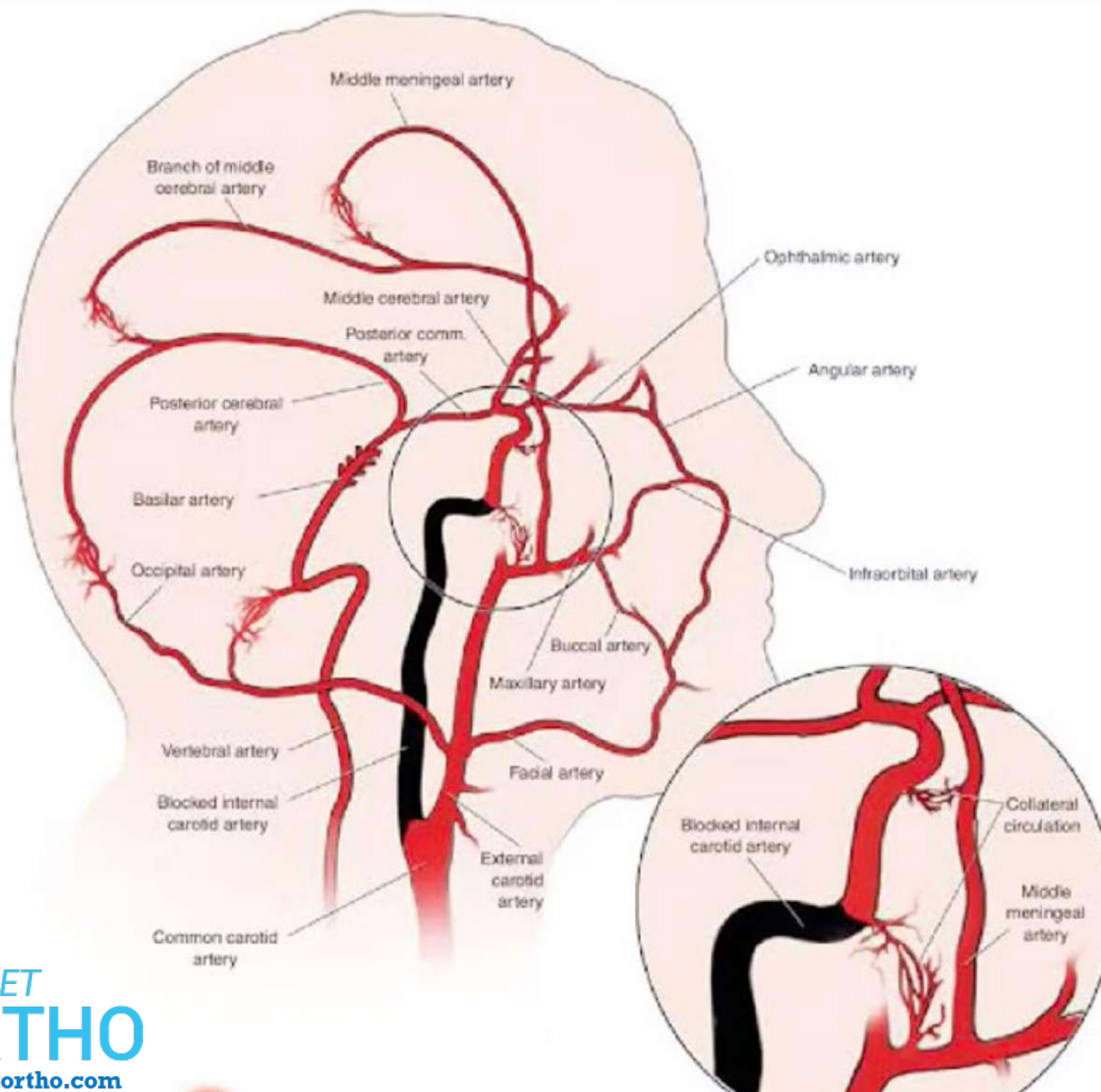
Anastomoses between SCA and PICA

- proximal vertebral artery occlusion

1. ECA → occipital a. → muscular a. Of VA-VA

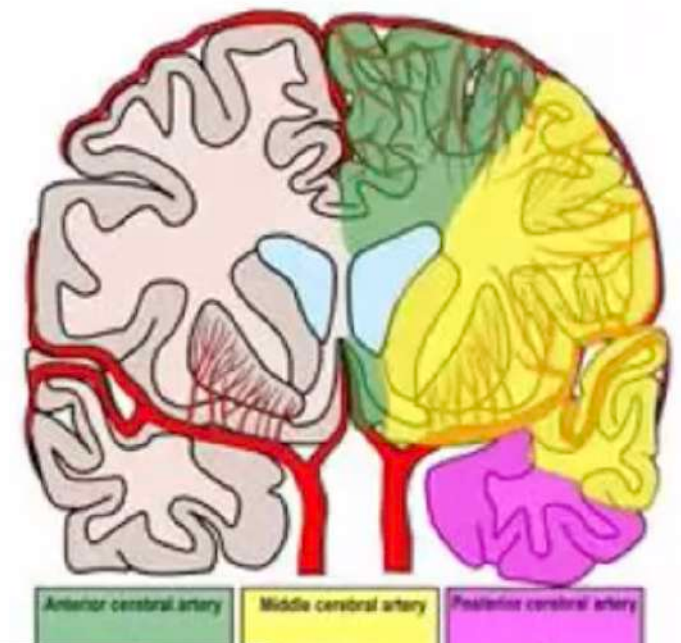
2. Thyrocervical trunk → ascending cervical a. →
direct connection or spinal radicals a. → VA

3. Contra lateral VA and/or ascending cervical a. Via
spinal radical arm



OCCLUSION SYNDROMES

- ICA occlusion
 - Acute occlusion - 26-49% risk of stroke
 - Stroke risk is less in asymptomatic ICA occlusions
 - Complete occlusion is found in 10-15%
 - Worst-case scenario - no collaterals
- ACA territory : CL weakness LE>UE





• MCA territory

Deficit ^a	Complete (M1 occlusion)	Superior division	Inferior division
[CL] weakness of UE & LF	X	X	
[CL] weakness of lower face	X	X	
[CL] hemisensory loss (UE & LE)	X	X	
[CL] hemisensory loss of face (all modalities)	X	X	
[CL] neglect ^b	X	X	
[IL] gaze preference → PEF	X		
[CL] homonymous hemianopsia	X		X ^c
receptive aphasia ^d (Wernicke's area)	X		X
expressive aphasia ^d (Broca's area)	X	X	
<u>Gerstmann syndrome</u> (p. 107): with dominant parietal lobe infarct			

^a[CL] = contralateral, [IL] = ipsilateral. An "X" indicates that the deficit is present

^bwith involvement on side of nondominant hemisphere

^cplus [CL] upper quadrantanopsia

^dwith involvement on the side of the dominant hemisphere

- **PCA territory**

a) unilateral occipital lobe infarction
→ homonymous hemianopsia with macular sparing (visual cortex of the macula receives dual blood supply from MCA and PCA)

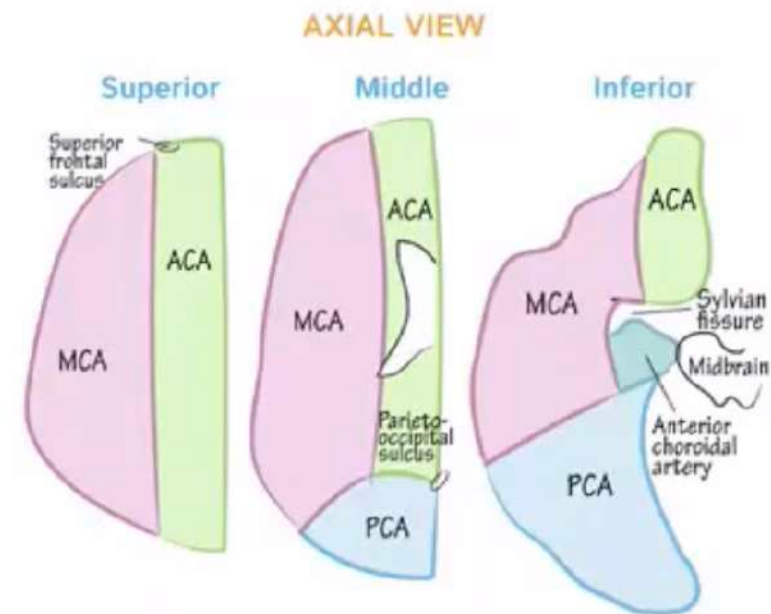
b) Balint syndrome

c) cortical blindness (Anton syndrome)

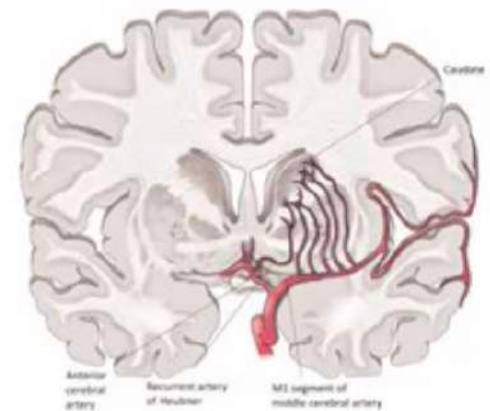
d) Weber syndrome

e) alexia without agraphia

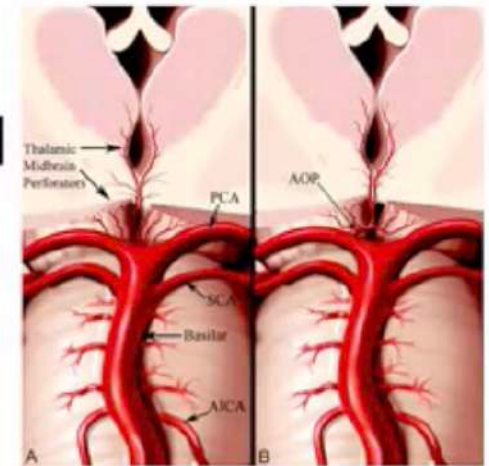
f) thalamic pain syndrome (Dejerine-Roussy syndrome)



A₁ - A₂A



- Recurrent medial striate artery of Heubner
 - Broca's aphasia with mild hemiparesis (UE>LE), proximal muscles weaker
- Anterior choroidal artery syndrome
 - Triad of CL hemiplegia, hemianesthesia and homonymous hemianopsia
- Artery of Percheron
 - Bilateral thalamic and mesencephalic infarcts



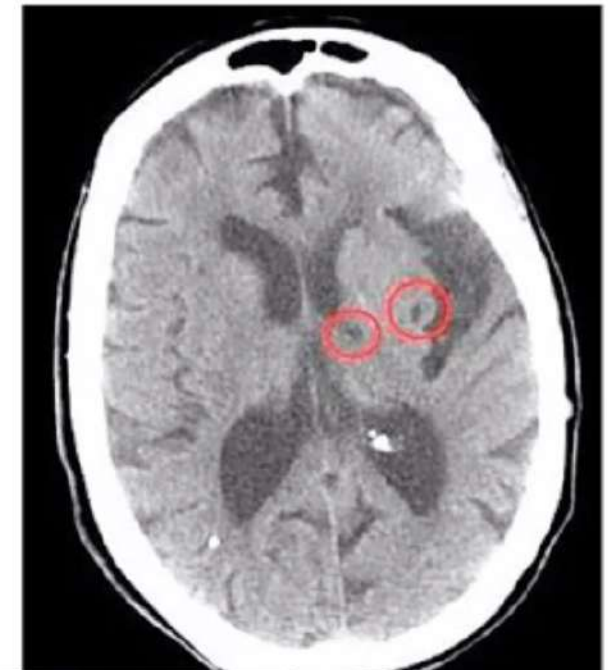
POSTERIOR CIRCULATION

- **Vertebral artery**
 - Medial medullary syndrome (Dejerine)
 - Lateral medullary syndrome (Wallenberg)
- **Basilar artery**
 - AICA: lateral pontine syndrome (Marie-Foix)
 - PICA: lateral medullary (Wallenberg)
 - SCA: superior cerebella vermis and cerebellum
 - Anterior spinal artery

Syndrome	Signs/Symptoms	Localization	Vascular Supply
Weber	Ipsilesional cranial nerve III palsy, contralesional hemiparesis (including the lower face)	Medial midbrain/cerebral peduncle	Deep penetrating artery from posterior cerebral artery (refer to Table 2-3)
Benedikt	Ipsilesional cranial nerve III palsy, contralateral involuntary movements (intention tremor, hemichorea, or hemiathetosis)	Ventral midbrain involving red nucleus	Deep penetrating artery from posterior cerebral artery or paramedian penetrating branches of basilar artery
Nothnagel	Ipsilesional cranial nerve III palsy, contralesional dysmetria, and contralesional limb ataxia	Superior cerebellar peduncle	Deep penetrating artery from posterior cerebral artery
Foville	Ipsilateral cranial nerves VI and VII (lateral gaze palsy, upper and lower facial weakness), with or without contralateral hemiparesis	Caudal pontine tegmentum involving the facial colliculus	Pontine perforator branches off the basilar artery
One-and-a-half	Ipsilateral cranial nerve VI (lateral gaze) palsy, bilateral internuclear ophthalmoplegia	Paramedian pons involving the paramedian pontine reticular formation and medial longitudinal fasciculi	Paramedian pontine perforators off the basilar artery
Wallenberg	Ipsilesional facial and contralesional body hypalgesia and thermoanesthesia; ipsilesional palatal weakness; dysphagia, dysarthria, nystagmus, vertigo, nausea/vomiting; ipsilesional oculosympathetic defect (Horner syndrome); skew deviation, singultus	Lateral medulla	Posterior inferior cerebellar artery (should raise concern for disease in parent vertebral artery)
Dejerine	Ipsilesional tongue weakness and contralesional hemiparesis with or without contralesional	Medial medulla	Vertebral artery or anterior spinal artery

LACUNAE STROKES

- Small infarcts in deep noncortical cerebrum or brainstem
- Occlusion of penetrating branches
- 3-20 mm infarcts (CT detects higher ones)
- Small 3-7 mm lacunas due to vasculopathy (HTN) - irreversible by carotid endarterectomy



YOUNG ADULTS STROKE

- Etiologies
- Atherosclerosis -20%
- Embolism with recognised source: 20%
 - Cardiac origin - RHD, ~~~~~
 - Fat embolism syndrome
 - Paradoxical embolism → septal defects
 - Amniotic fluid embolism

- **Vasculopathy** vasculitis
 - Inflammatory
 - Takayasu's
 - Infective
 - Herpes zoster
 - Mucormycosis
 - Systemic disease — SLE
 - Non-inflammatory
 - FMD
 - Dissections
 - Moyamoya disease
 - Homocystinuria

- Coagulopathy
- Peripartum - 5%
- Miscellaneous - 35%

- Uncertain
- OCPs
- CVT
- Migraine - rare
- Cocaine abuse
- PRES

← warfarin
liver dis

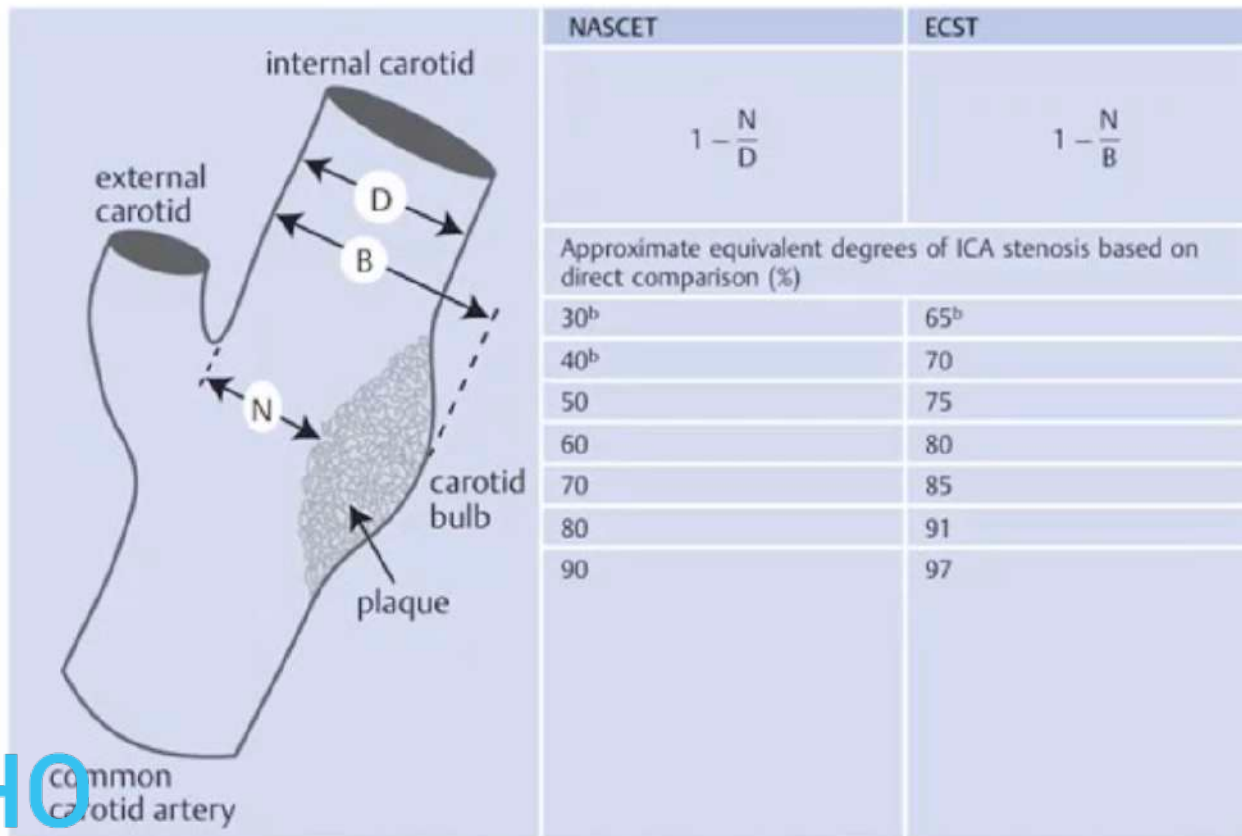
cocaine

RISK FACTORS

- Diabetes: OR 12
- HTN: OR 6.8
- Cigarette smoking: OR 2.5
- Long term heavy alcohol consumption: OR 15

EVALUATION OF CAROTID DISEASE

- **angiography**
 - Gold standard
 - Different definitions of stenosis



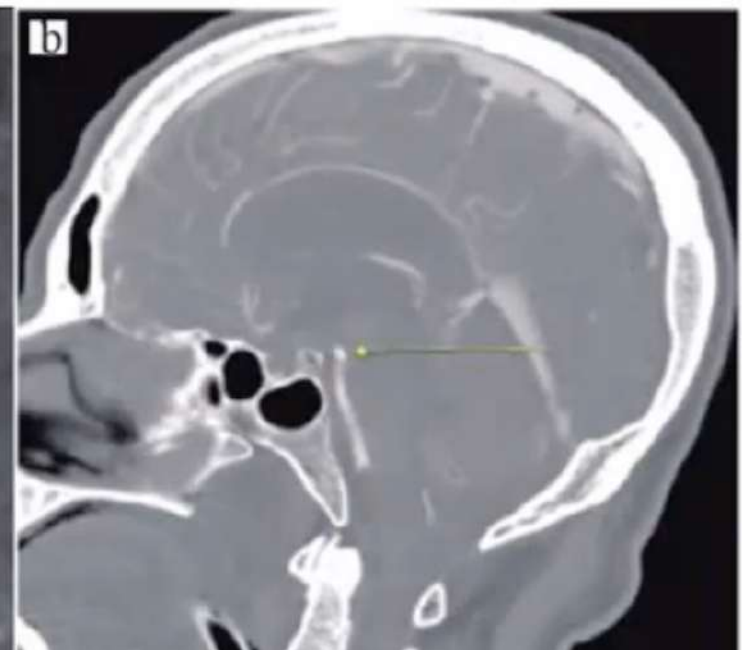
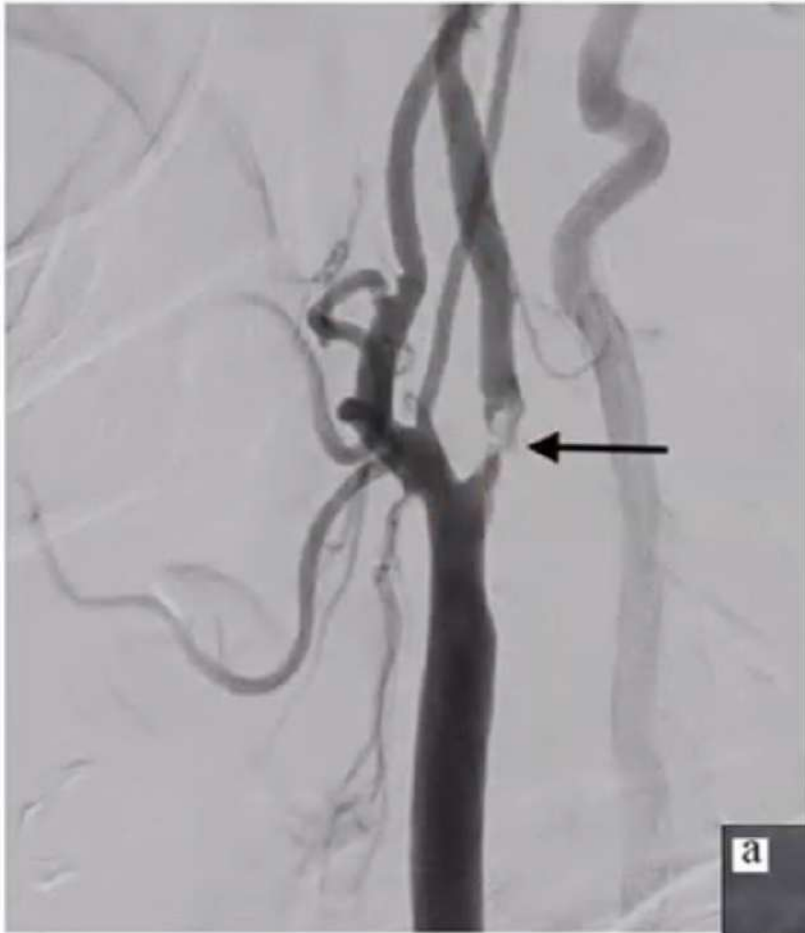
$$\% \text{ stenosis (NASCET)} = \left(1 - \frac{N}{D}\right) \times 100$$

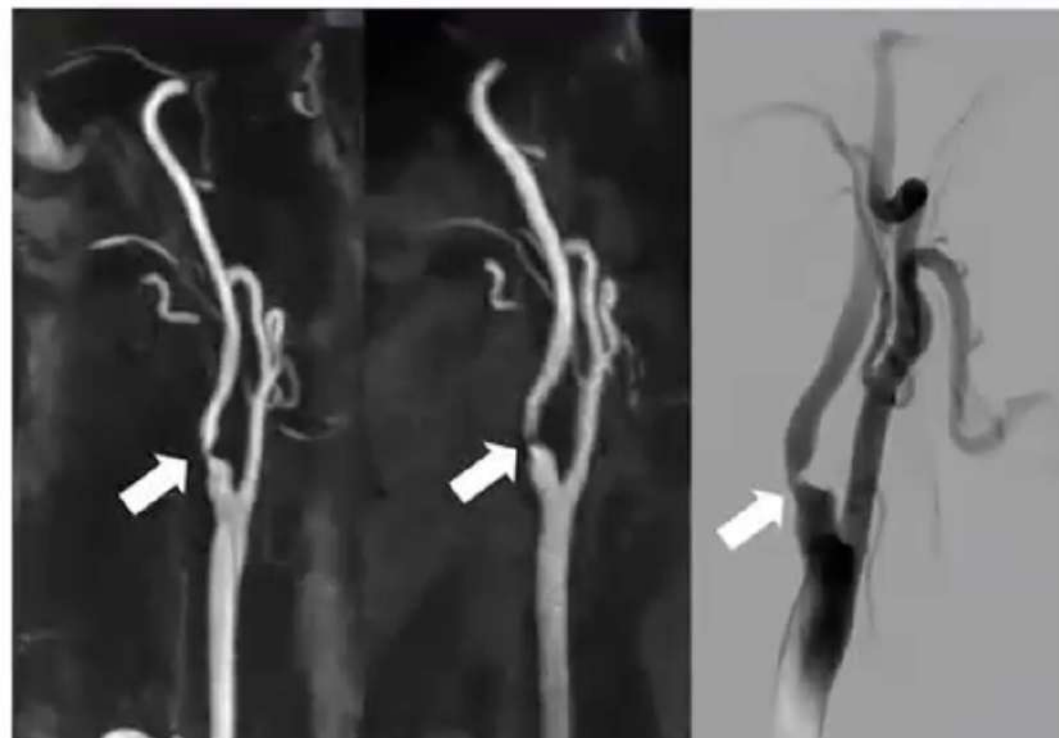
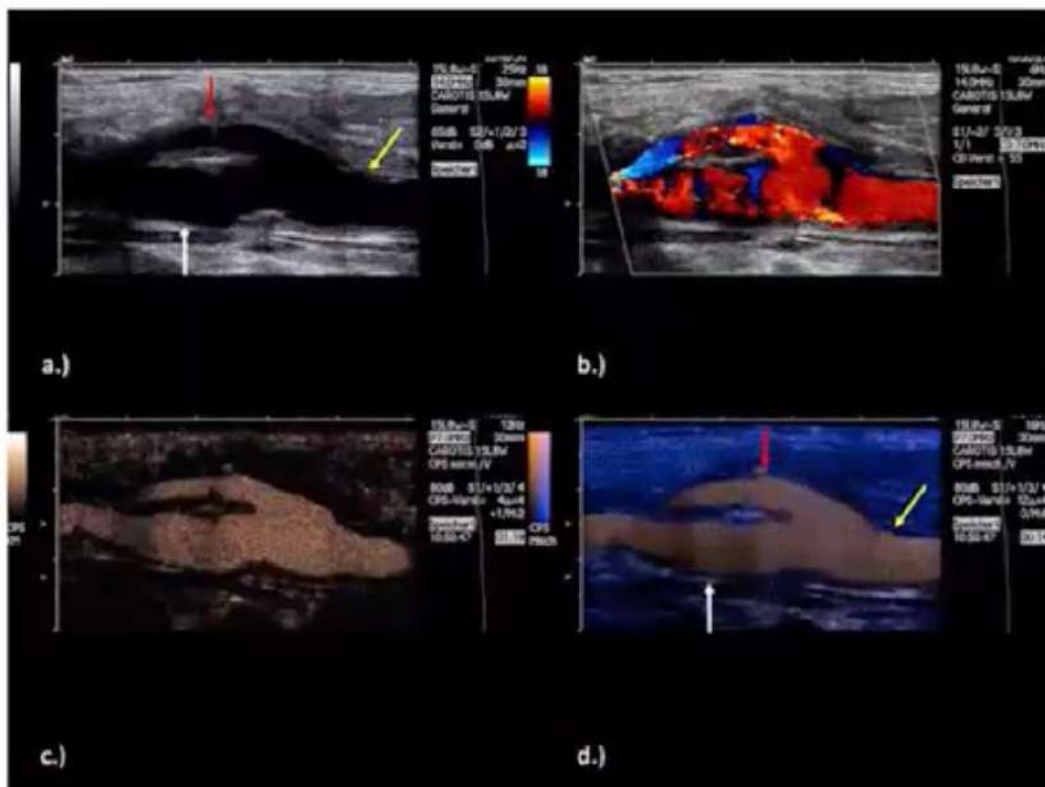
$$\% \text{ stenosis (by ECST)} = 0.6 \times \% \text{ stenosis (by NASCET)} + 40\%$$

- **Doppler ultrasound**
 - **Sens 88%; spec 76%**
- **MR angiography**
 - **Sens 91%; spec 88%**

- **CT angiography**

- **Sens 85%; spec 93%**





TREATMENT

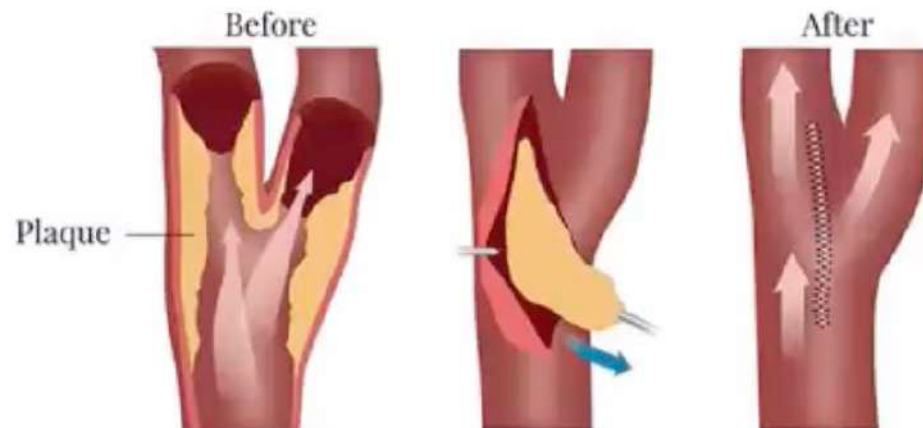
- 1. “Best medical management”
- 2. Carotid endarterectomy
- 3. Endovascular techniques

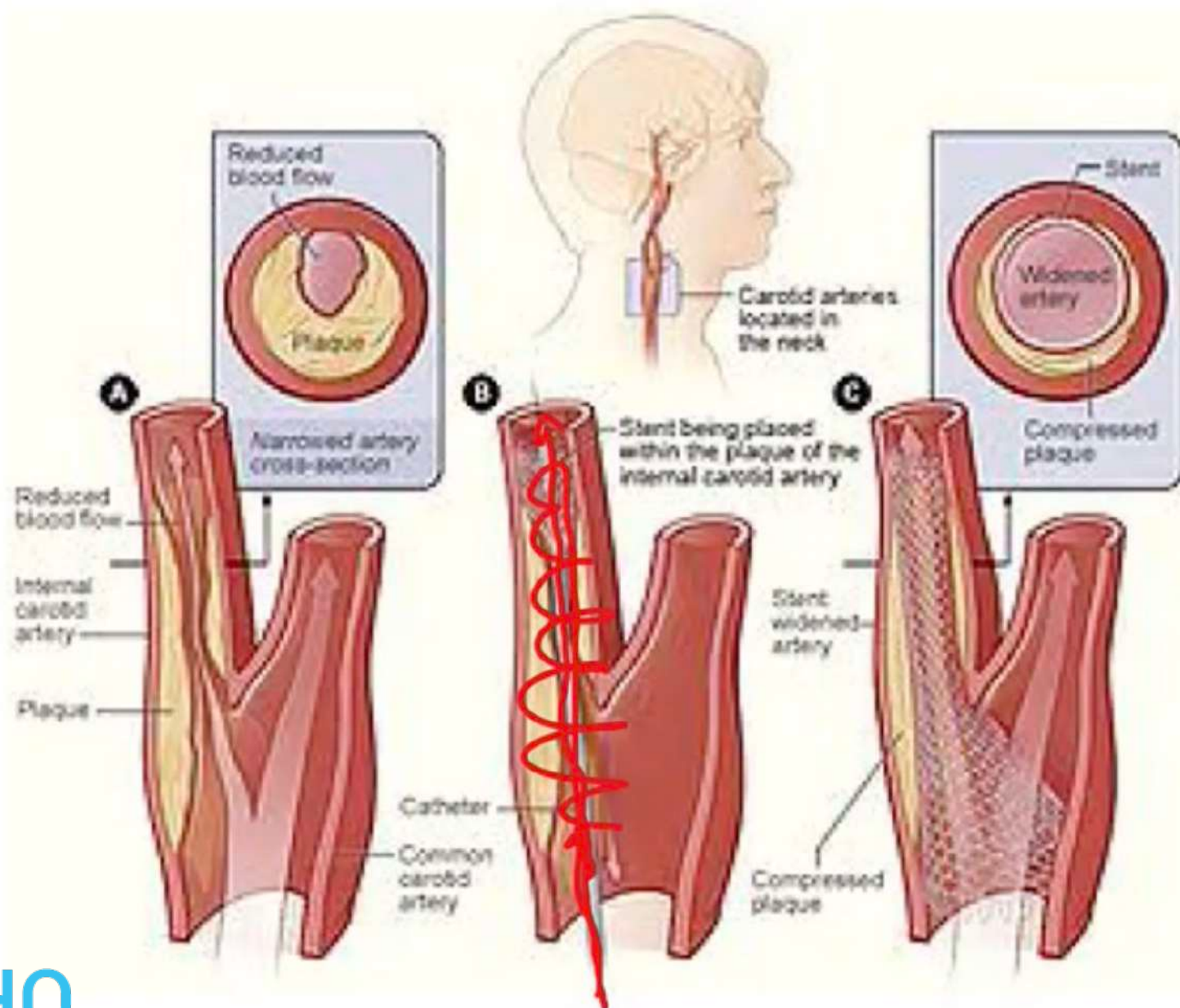
MEDICAL THERAPY

- Antiplatelet therapy
 - Aspirin, clopidogrel, combination
- Antihypertensive therapy
- Diabetes control
- Asymptomatic AF → anticoagulation
- Antilipid therapy
- Smoking cessation

ENDARTERECTOMY VS ARTERY STENTING

- CEA —> asymptomatic patients with >70% stenosis, if risk of perioperative stroke, MI and death is low
- CEA in older patients when anatomy is unfavorable for endovascular
- CAS —> when anatomy is unfavourable for surgery
- Prophylactic CAS —> highly selected patients; >60% stenosis by DSA; >70% by Doppler; effectiveness compared to medical therapy is not established





ACUTE ISCHEMIA STROKE EVALUATION

- **History & examination**
 - **Onset / Ictus time**
 - **Current deficit**
 - **NIH stroke scale score**
- **Laboratories**
 - **Blood glucose (since it affects eligibility for IV tPA)**
 - **admitting orders**

Stroke

- Imaging
 - STAT NCCT head scan
 - Within 20min
 - Rule out hemorrhage/tumor
 - Determine ASPECTS score
 - CT angiography
 - Identify potential candidates for endovascular therapy (EVT)



- **Intervention**

- **EVT - immediately**

- **IV tPA** - if there is going to be delay or not eligible candidates

GENERAL MEASURES

- Avoid hyperglycaemia > 180 mg/dl
- IV fluids: NS or half NS at 75-125 ml/hr
- Avoid over hydration in ICH, CHF or SBP>180
- Prophylactic AED not recommended
- Identify and treat MI or arrhythmia
- BP management

Table 81.2 Guidelines for lower limits of treatment endpoints for HTN in strokes

	No prior history of HTN	Prior history of HTN
do not lower SBP below	160–170 mm Hg	180–185 mm Hg
do not lower DBP below	95–105 mm Hg	105–110 mm Hg

- Osmotic therapy - IV mannitol or 3% saline
- ~~Steroids~~ - not recommended; except steroid responsive arteritis or associated brain tumor
- Stool softener
- Antiplatelet therapy

IMAGING IN ACUTE ISCHEMIA STROKE

- Hyper acute stroke - CT scan head
 - Hyper dense artery sign
 - Focal low attenuation within the gray matter
 - Loss of gray-white interface
 - Attenuation of lentiform nucleus
 - Mass effect
 - Loss of the insular ribbon sign
 - Enhancement with IV contrast

- **Alberta stroke program early CT score (ASPECTS)**
 - 2 NCCT slices
 - One at the level of thalamus
 - Second just rostral to the basal ganglia
 - MCA divided into 10 territories
 - Score ≤ 7 has worse outcome

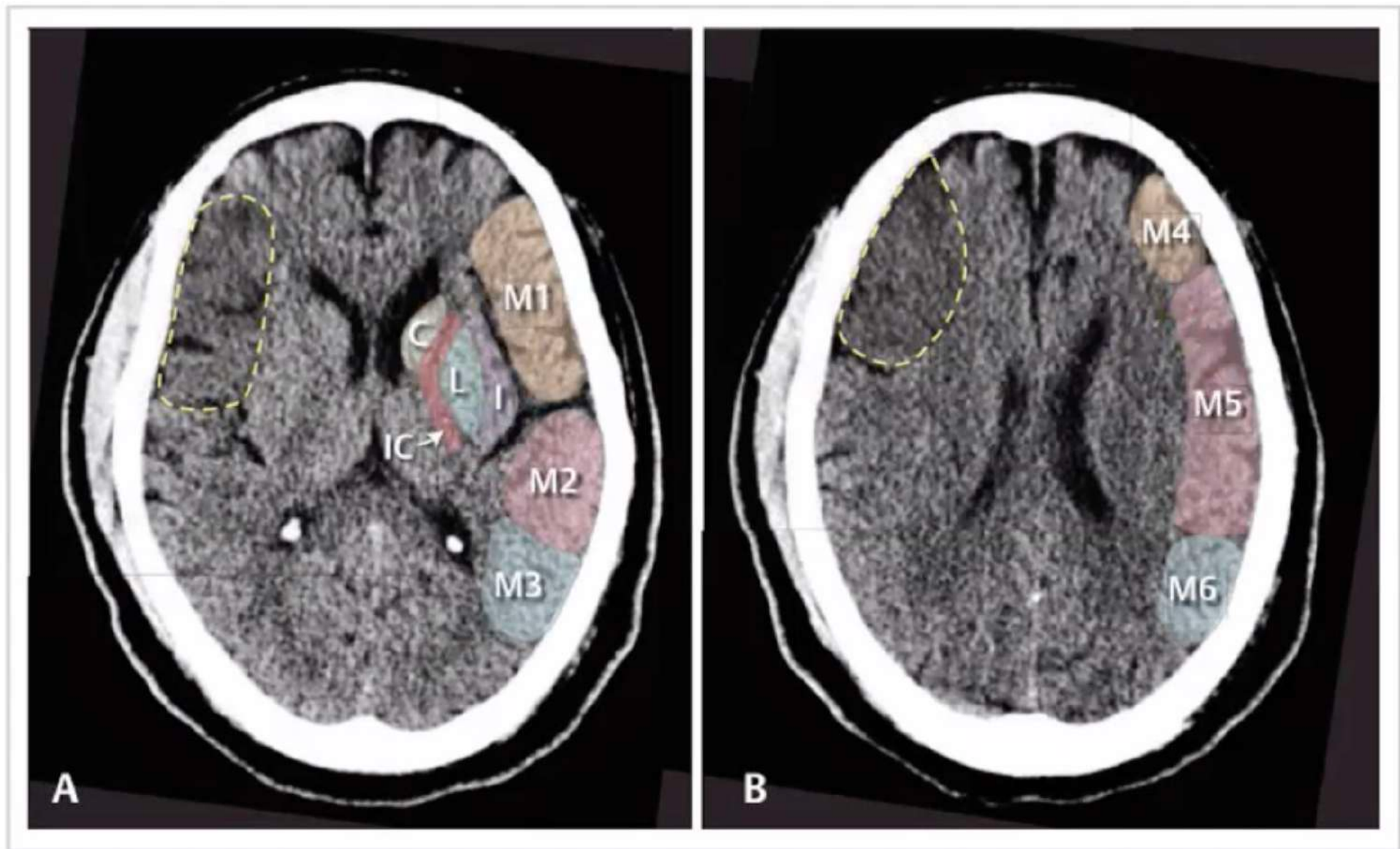


Fig. 81.1 Illustration of ASPECTS in a 71 year old male with right MCA infarct.

Noncontrast axial head CT, A: at level of the thalamus, B: just above the basal ganglia.

The 10 graded areas are shown as colored shapes on the patient's left side: C = caudate (head), IC = internal capsule, L = lentiform nucleus (BG), I = insular cortex, M1 through M6 = convexity cortical territories.

The infarct is denoted by the yellow broken lines. Also note compression of right lateral ventricle.



TREATMENT OPTIONS TIMELINE

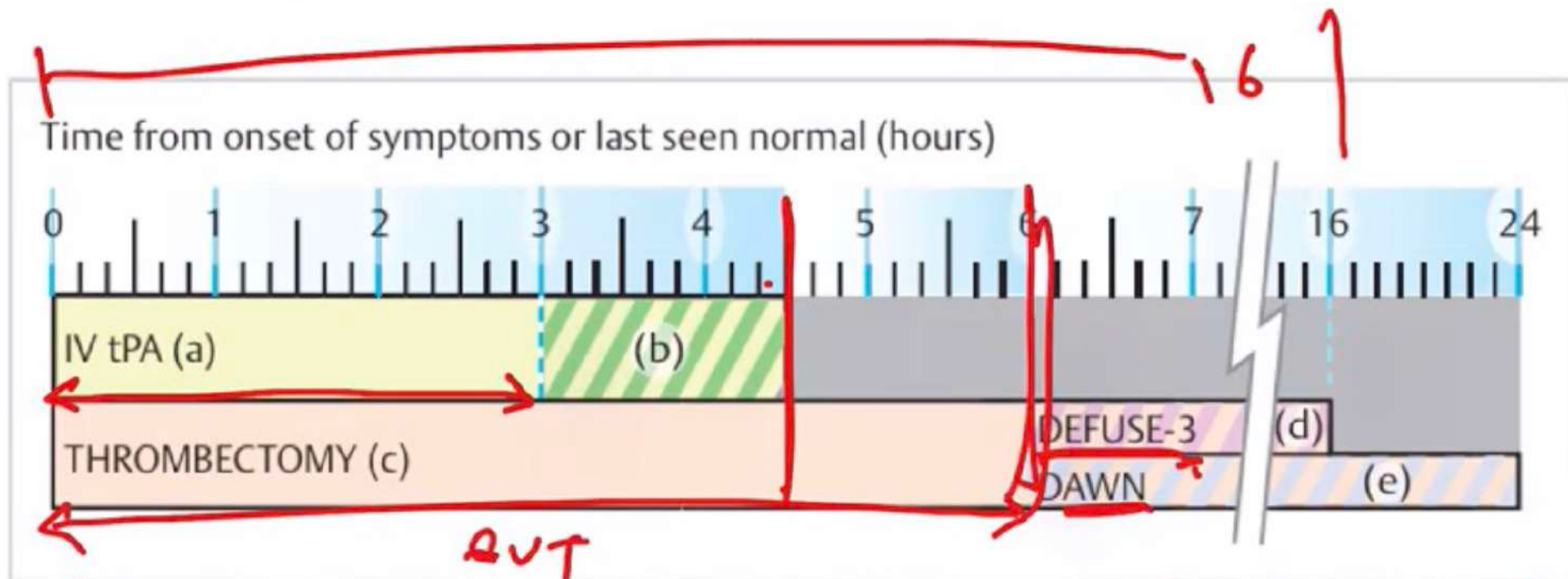


Fig. 81.3 Treatment options timeline.

(a) IV tPA: give to essentially all candidates within 4.5 hours of onset.

(b) 3–4.5 hrs: IV tPA reasonable but not studied in same patient population (see text).

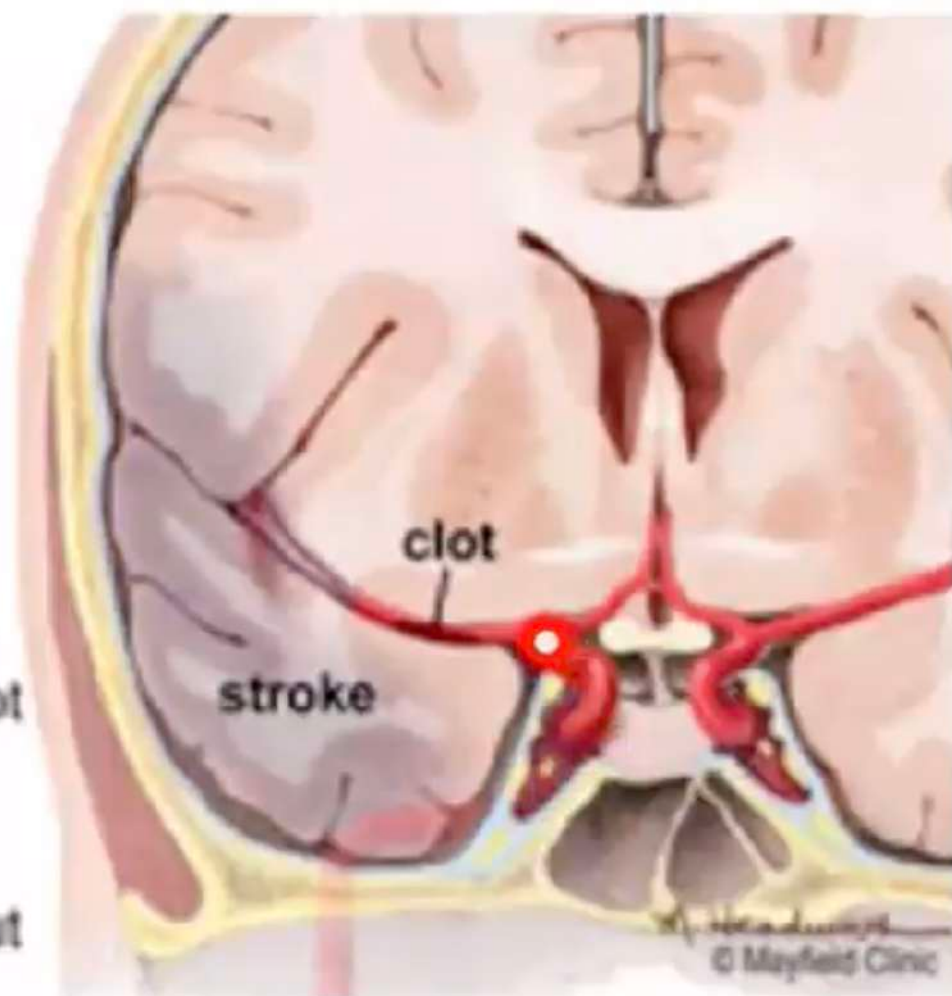
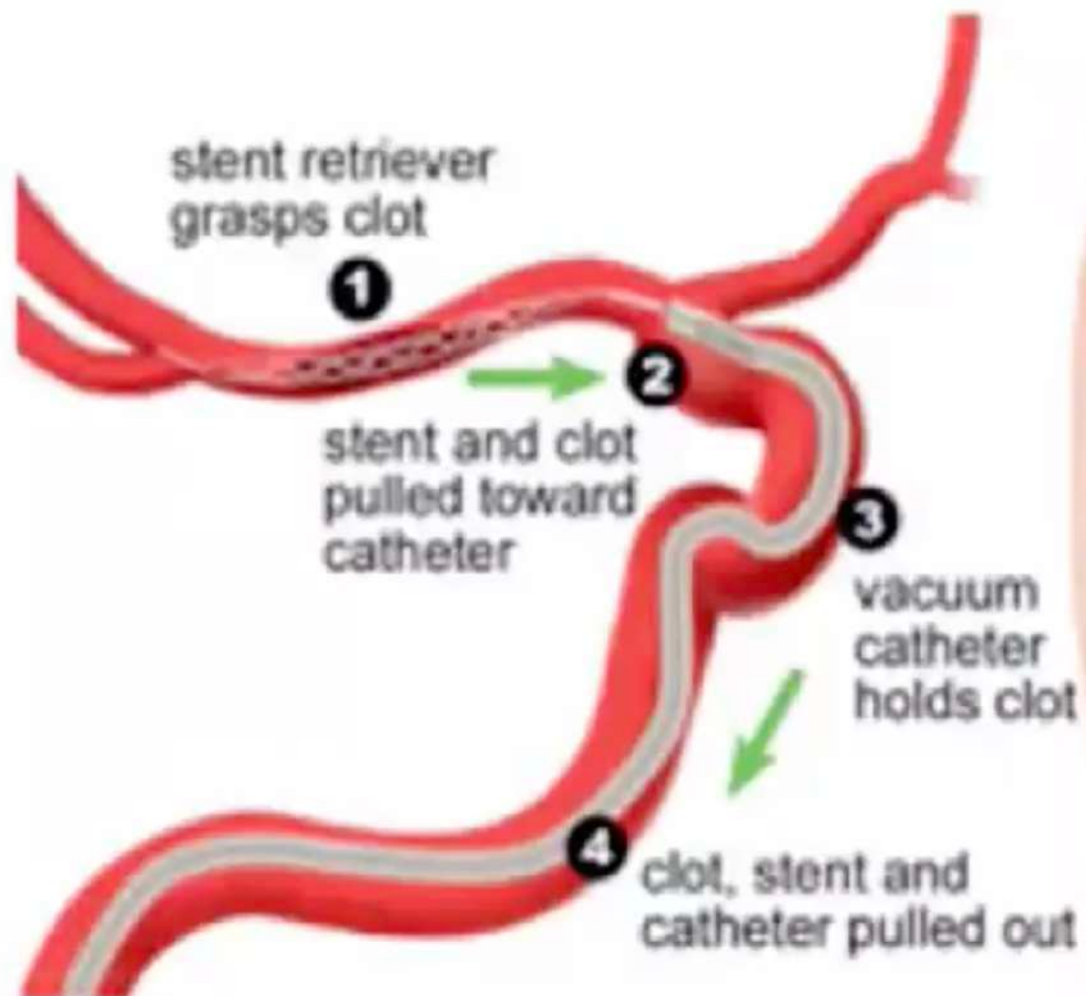
(c) thrombectomy candidates: NIHHS score ≥ 6 , ASPECTS ≥ 6 , large vessel occlusion (LVO), puncture ≤ 6 hrs from onset.

(d) thrombectomy 6–16 hrs in candidates who meet other DAWN or DEFUSE-3 (p. 1356) eligibility (see text).

(e) thrombectomy 16–24 hrs in candidates with anterior circulation LVO who meet other DAWN eligibility criteria.

MECHANICAL THROMBECTOMY

- Recommended when all criteria met:
 - Perestroika mRS 0-1
 - Causative occlusion of ICA or M1/MCA
 - Age ≥ 18 y
 - NIHSS ≥ 6
 - ASPECTS ≥ 6
 - Treatment can be initiated ≤ 6 hours o o nset



THROMBOLYTIC THERAPY

- Guidelines for administration of IV tPA
 - Age ≥ 18 y
 - Onset ≤ 4.5 hr
 - History or ≤ 10 microbleeds on an MRI
 - Known sickle cell disease
- Contraindications
 - ICH, SAH, aneurysm or AVM, active internal bleeding
 - > 10 microbleeds
 - Bleeding diathesis
 - Head trauma
 - SBP > 185 mmHg

CAROTID ENDARTERECTOMY

- **NASCET**

- Hemispheric or retinal TIA
- Mild stroke within 120 days
- Ipsilateral high grade stenosis ($>70\%$)
- CEA reduced the rate of strokes by 17% at 18 m on this and death by 7% at 18 months
- Compared to best medical management

OTHER SURGICAL MODALITIES

- Decompressive craniectomy
- Cisternostomy
- Ventricular drainage
- Stenting

