Stroke

Global mortality

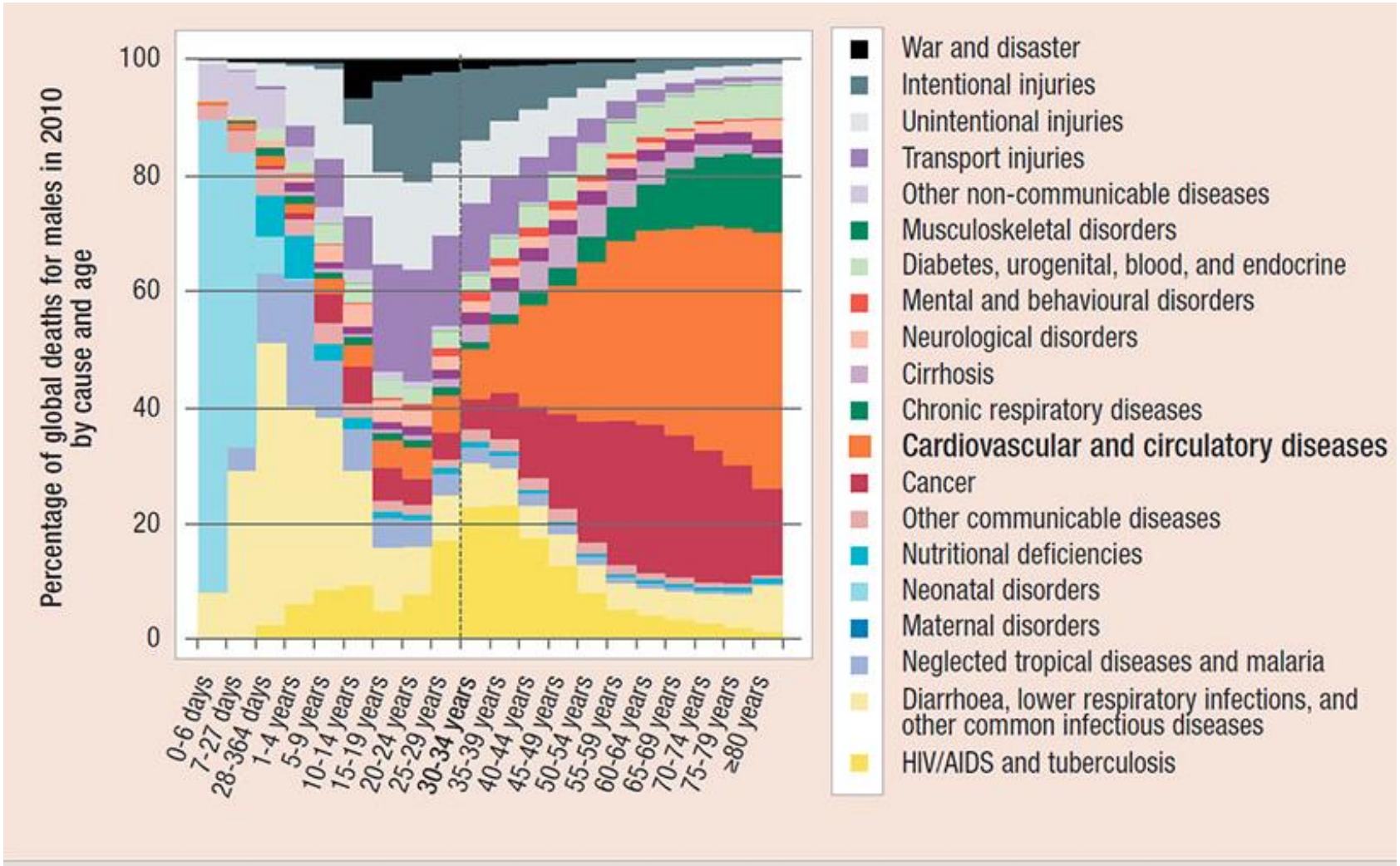
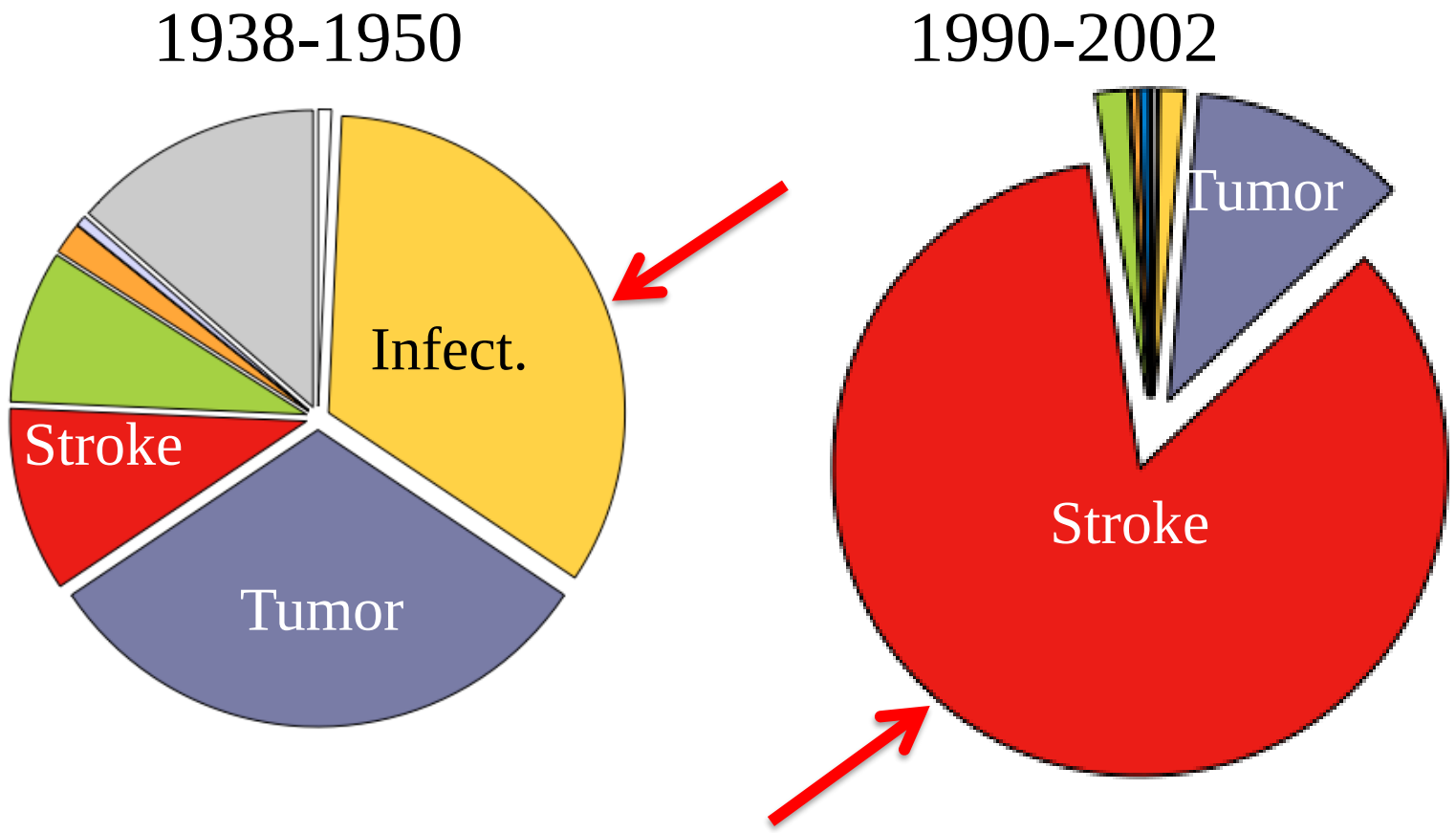


Figure 1. Percentage of global deaths for males in 2010 by cause and age according to the Global Burden of Diseases, Injuries, and Risk Factors Study 2010 (GBD 2010). (1)

Cause of death—Dept. of Neurology?



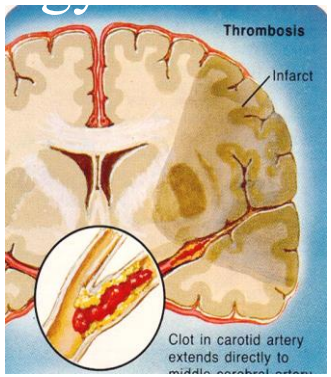
Normal lifestyle

After MI **70-80%**

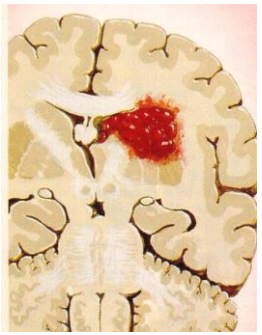
After stroke **10%**

Differential diagnosis---CT/MRI!!!

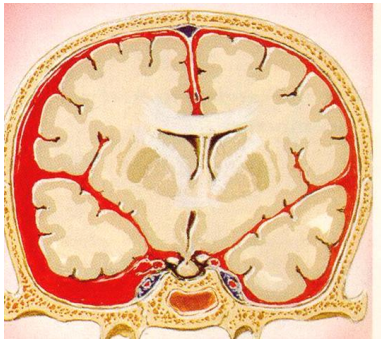
ischemia 80%



sp. Hemorrhage 10-15%

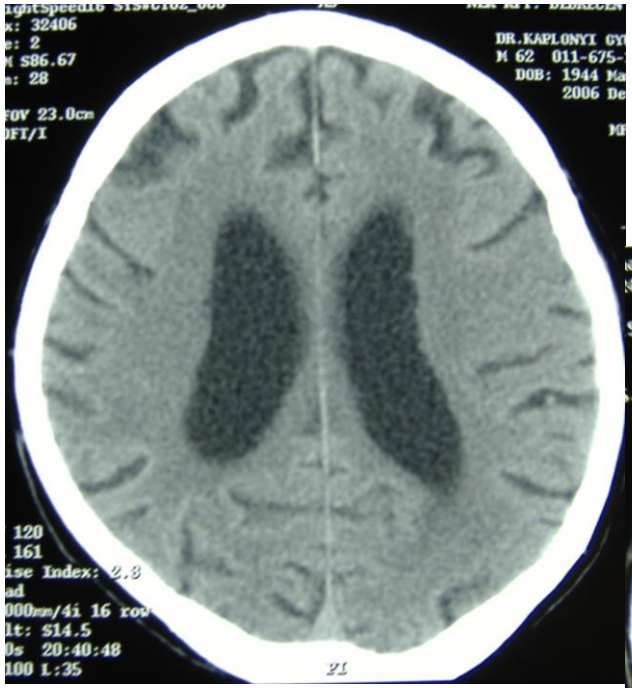


Subarachnoidal bleeding

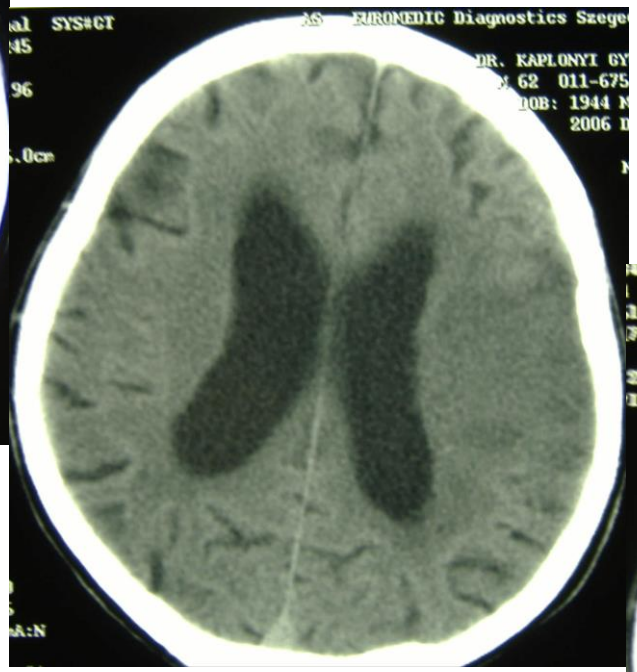


CT

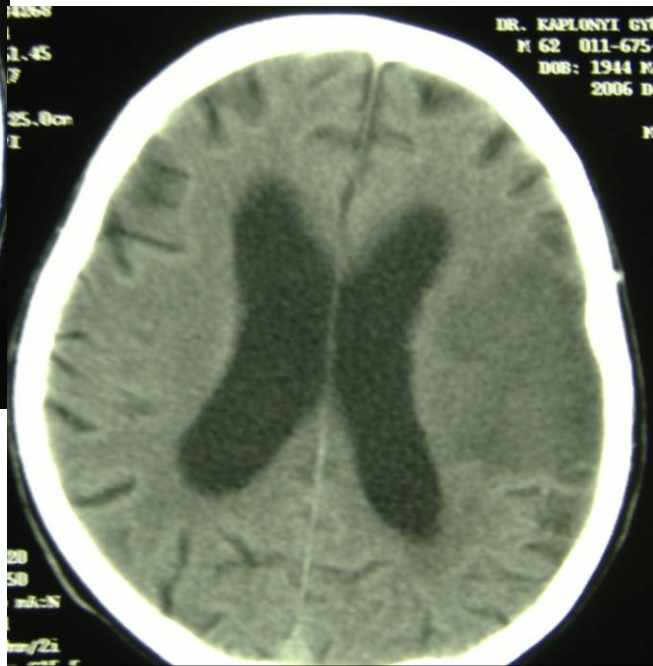
62 yrs stroke at admission



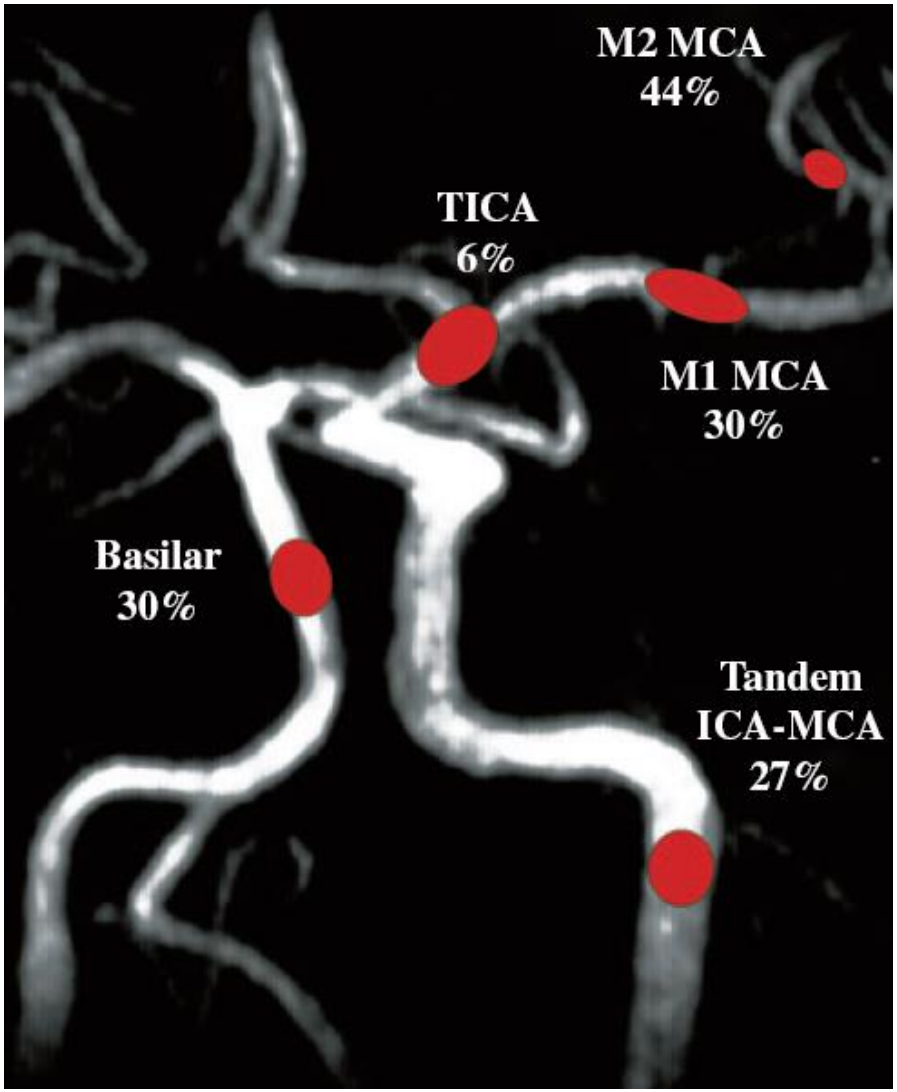
One day later



2 days later

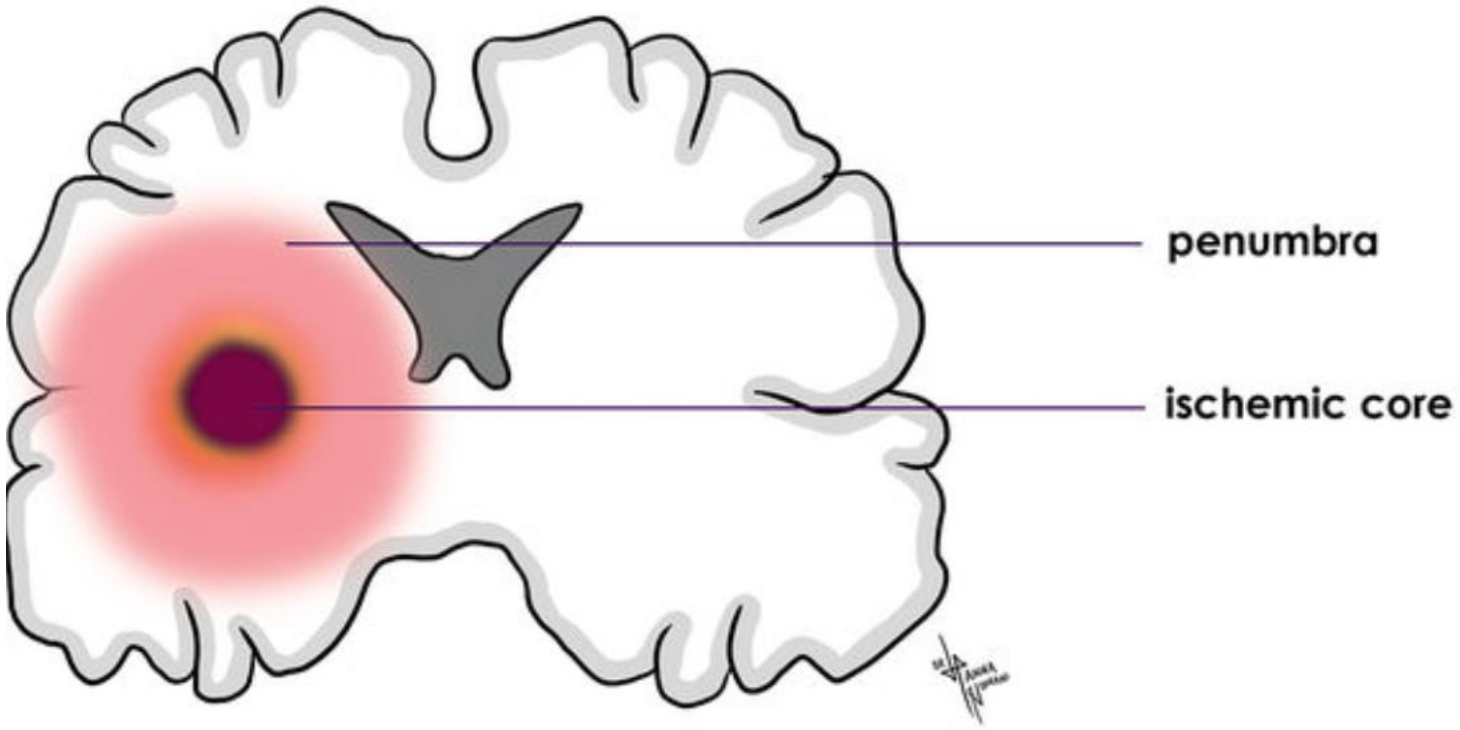


Large vessel occluded? Less chance for successful intravenous lysis..



Alexandrov , J Int. Medicine 267; 209–219 2010.

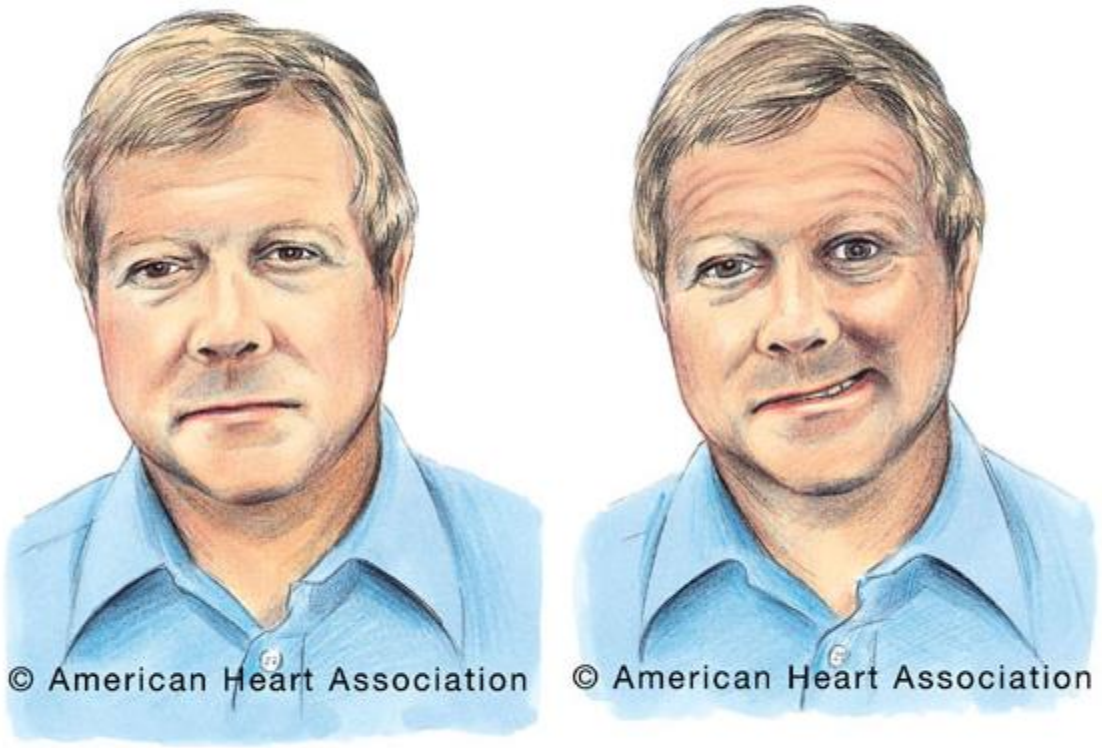
Ischemic Stroke



Cincinatti scale 1.

Cincinnati Stroke Scale

printable version PDF



Facial Droop

- *Normal*: Both sides of face move equally
- *Abnormal*: One side of face does not move at all

Cincinatti scale 2.

Arm Drift

- *Normal*: Both arms move equally or not at all
- *Abnormal*: One arm drifts compared to the other

Cincinatti scale 3.

- Slurred speech, aphasia

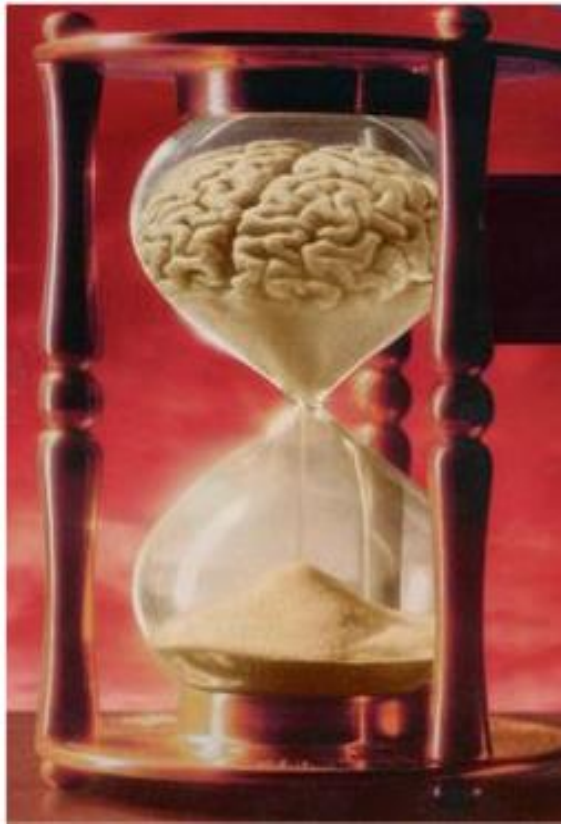
TIA (TRANSIENT ISCHEMIC ATT.)

- Transient symptoms
- Minutes
- No residual tissue deficit (MRI)

TIA is emergency!!! High risk of devastating stroke

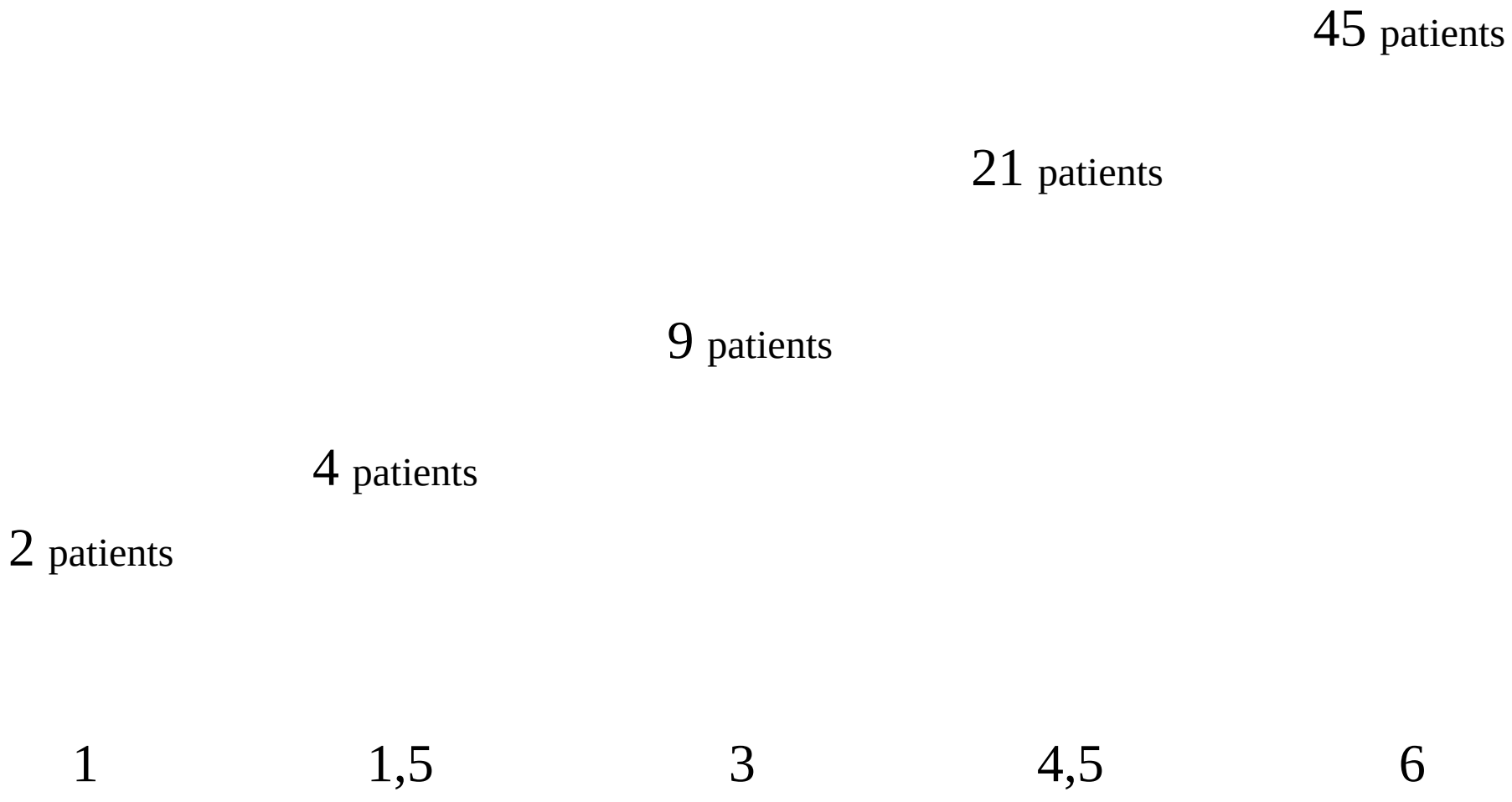
Ischemic Stroke

- 700 km axon/hour↓
- 2 millió neuron/min ↓



Time is brain!

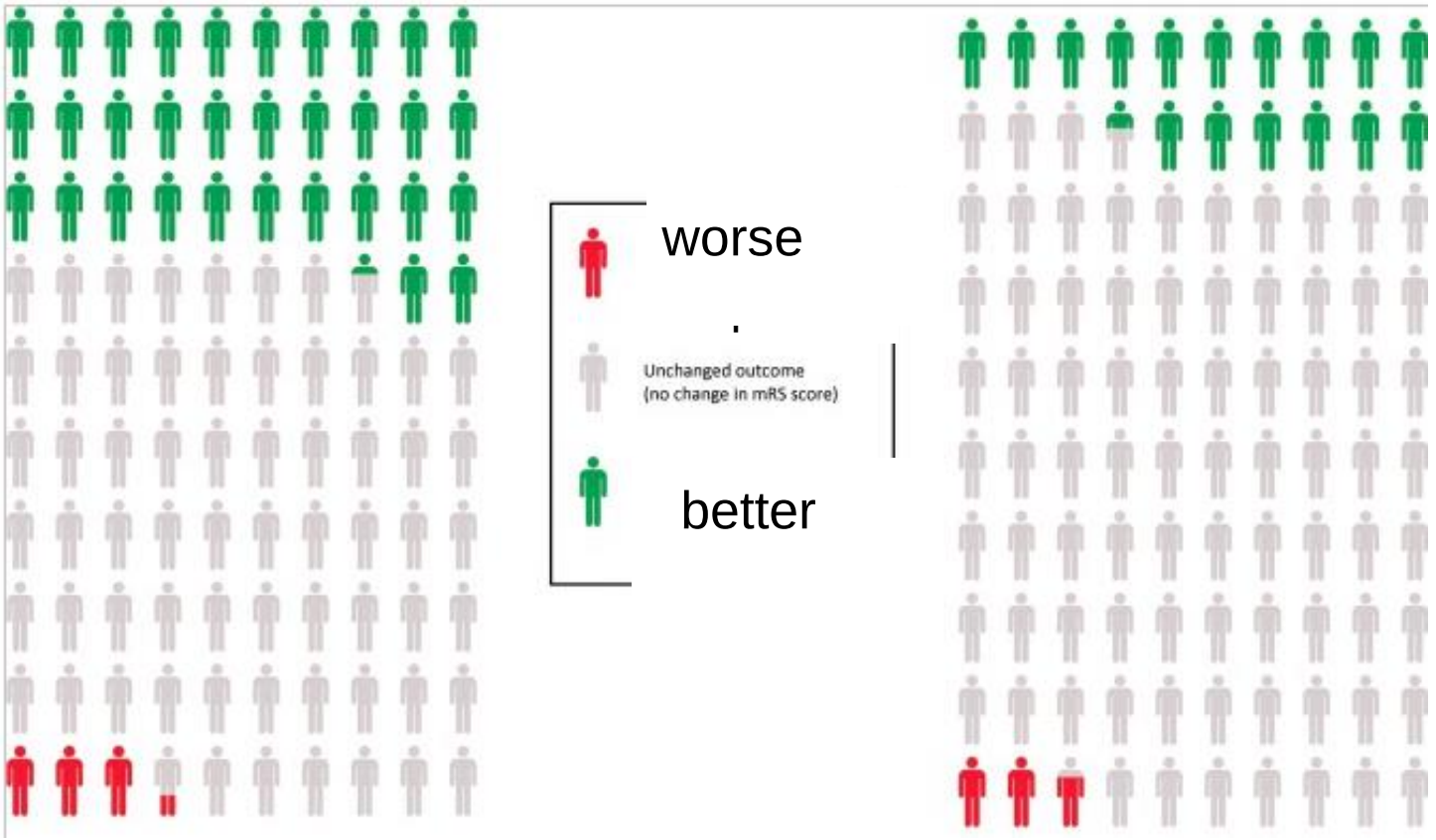
Open the artery as soon as possible



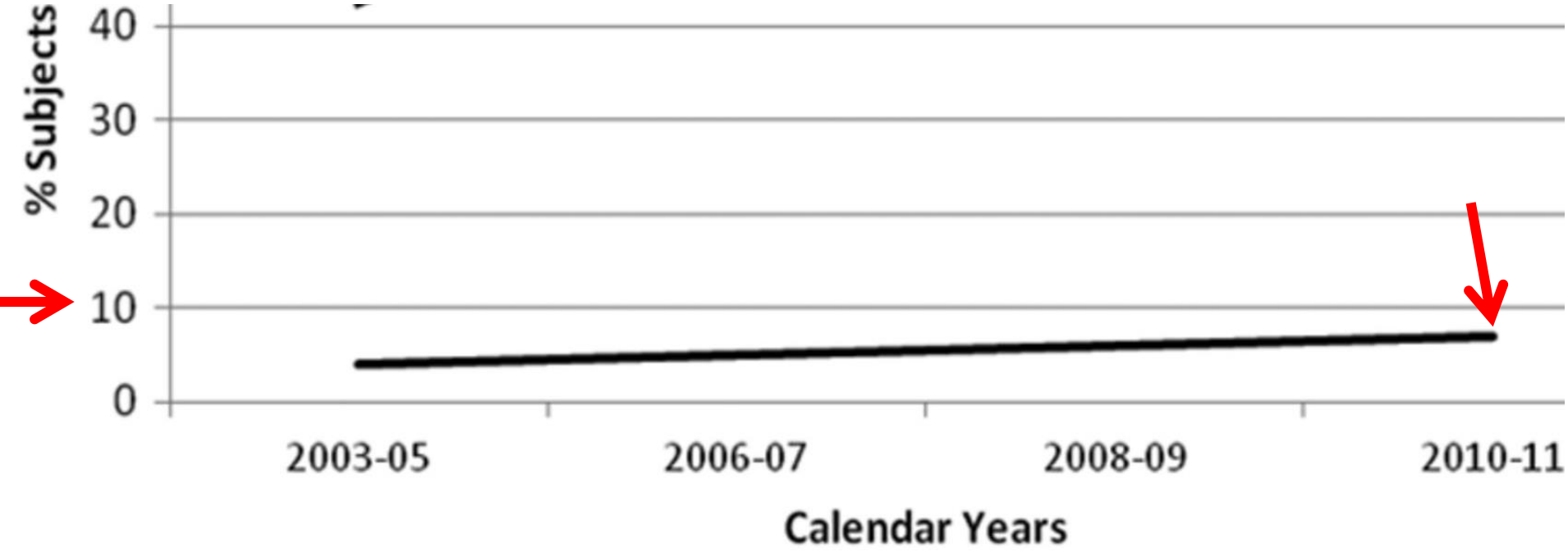
Lysis…….

Within 3 hours

between 3-4,5 hours



Lysis in acute stroke: USA



Lee H. Schwamm et al. Circ Cardiovasc Qual Outcomes. 2013;6:543-549

iv. Thrombolysis in Debrecen

2011	2012	2013	2014	2015	2016.12.12.
133	142	113	131	147	216
(%) 20	20	17	18	19%	23%

More than 1300 iv. lysis!

Debrecen stroke care

stroke

The strength of the chain depends on the weakest element!

Ambulance!

CT-!
CT+CT angio!

Stroke unit

Personalised:

Iv?
intraarterial?
iv+ia?
mechanical thrombectomy

Time window?
depends on the vessel and time elapsed after stroke?

Within 4,5 hours (some subgroups 3 hours) **Iv.** lysis if small vessel occlusion

6-8 hours IF ICA or MCA occlusion:intraarterial or mechanical thrombectomy (MET) BUT start with iv.
In case of specific constellation of MRI(!) and age and infarct volume → the time window could be 24 h!!!

In basilar artery the time window could be **12 h (iv or ia. lysis)**

time

If out of time window?

- 100-300 mg aspirin
- Monitoring of BP and ECG
- Do NOT decrease BP till 220/110 Hgmm!
- Pulsoximetry, 2-4 lit oxigen, if less 94%
- Normoglycemia
- LMWH or heparin to prevent DVT deep venous thromb.
- Nasogastric tube if dysphagia
- Antipyretic ther.
- If seizure antiepilept.
- antibiotics

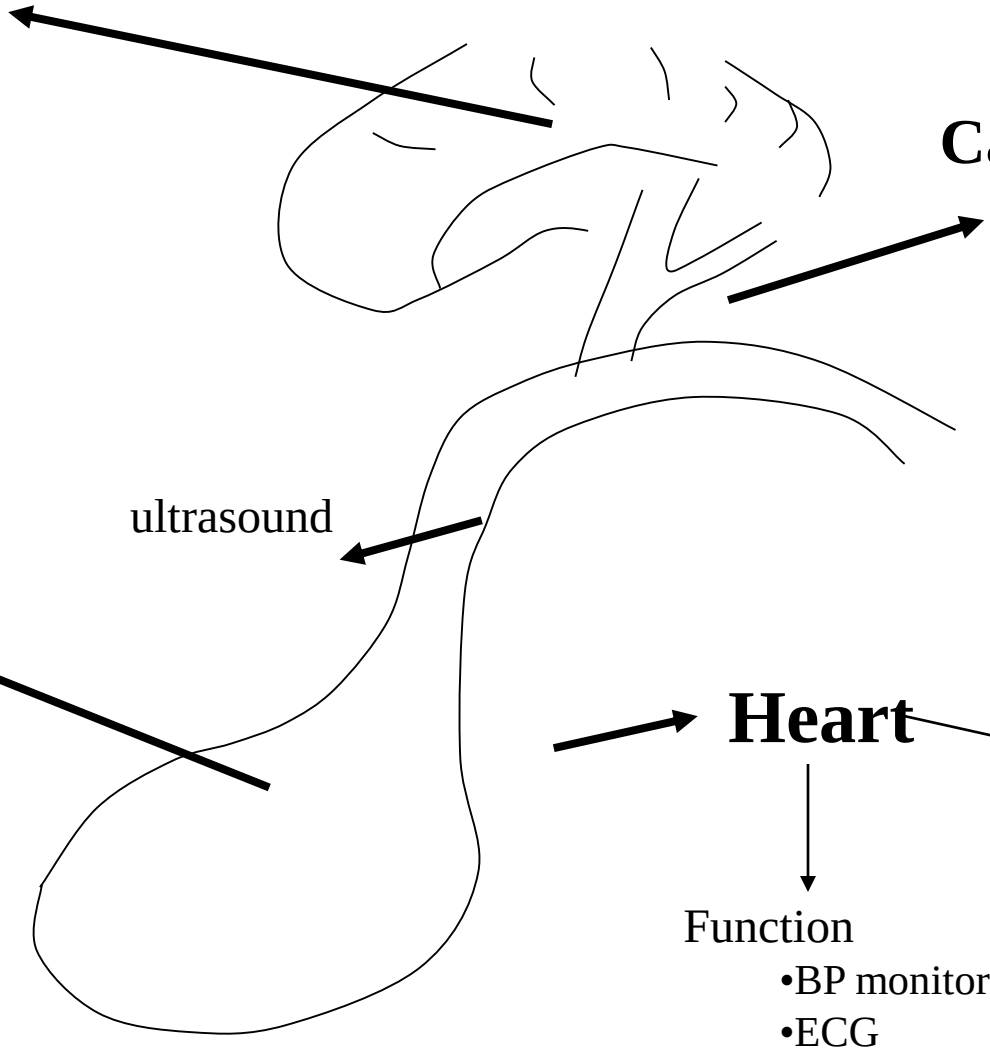
After desoblieration therapy, the next question:

cause of stroke?

Stroke: diagnosis

Imaging

•CT
•MRI
  •Diff. WI
  •TCD
•DSA angiogr.



Carotid, vertebr-

•ultrasound
•CT AG
•MRA
•DSA

ultrasound

Blood

•Blood counts
•Glucose, ions
•Coagulation?
•lipids
•Immunol. tests

Heart

morphology
•TTE
•TEE

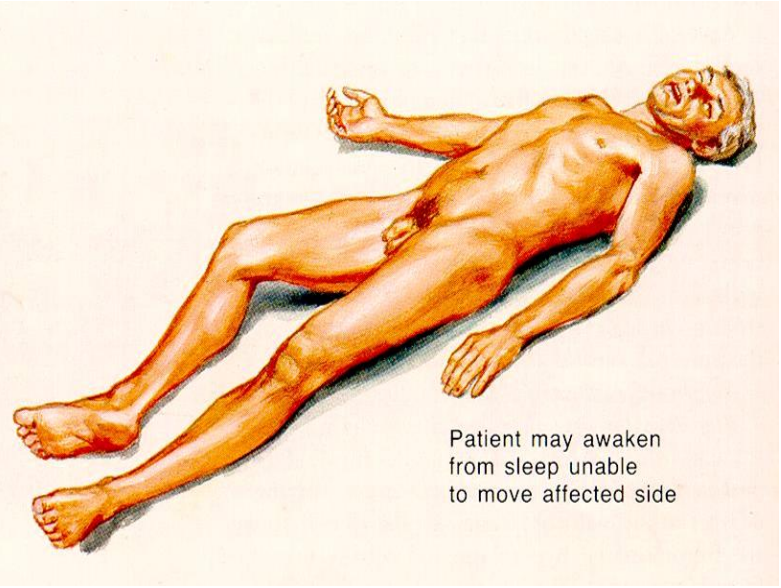
Function
•BP monitoring
•ECG
•Holter ECG

How to prevent the 2^{nd} stroke?

Recurrent stroke

- 1 mo 30%
- 1 year 10%
- then 6-8%

How to prevent the 2nd stroke



1. Antiplatelet therapy
   - asp+DP>aspirin mono
   - clopidogrel 75 mg
   - triflusal
2. Antihipertensive th.
   - ACE inhib+diureticum
   - E.g.perindopril+indapamide
3. Statin
4. AF or cardiogenic source of emboli→ AC INR 2-3
5. Carotid stenosis stent or CEA in 2 weeks

   70-99% stenosis

   Only if TIA and minor stroke

   Occlusion NO!! Plegic?NO!!!!!

1. Antiplatelet

- 2x aspirin (25mg)+ ER dipyridamol (200 mg) prevents 1 pts/100 pts in 1 year

- aspirin + Clopidogrel? No!

How to prevent the 2nd stroke

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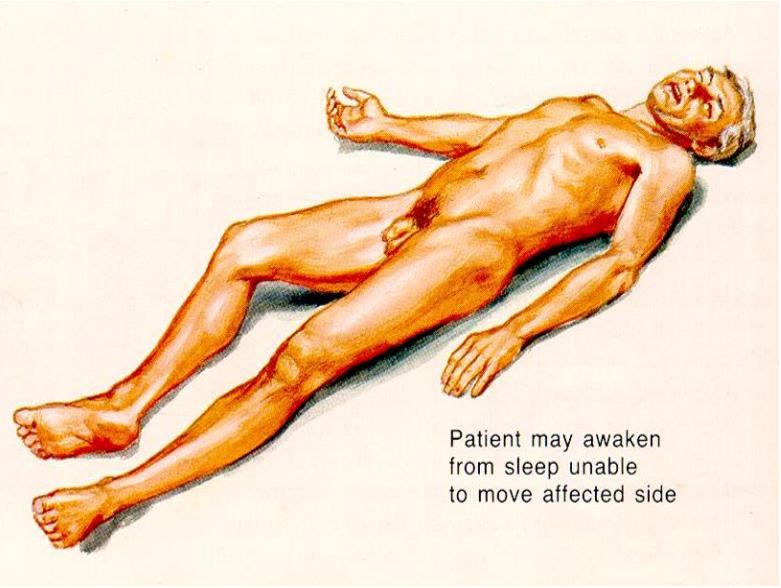
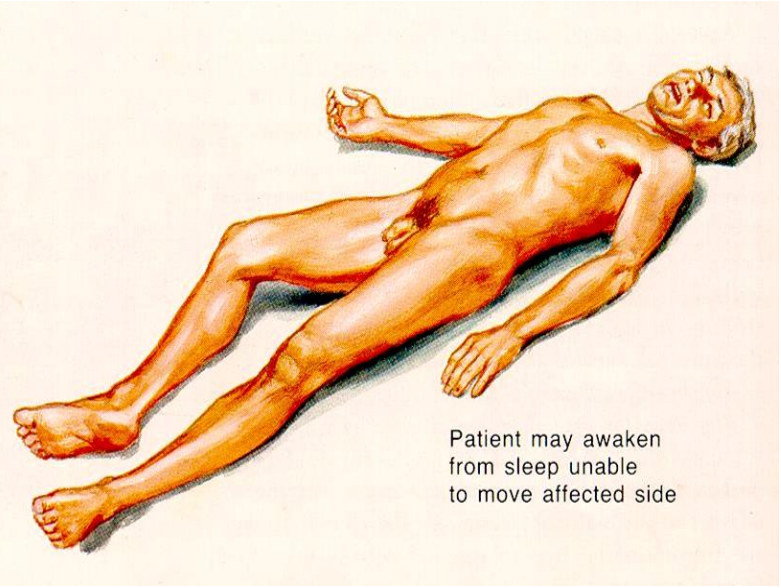


2. BP

- stroke risk ↑if more than 115 Hgmm
- ↓ If 10/5 Hgmm BP stroke risk ↓ 30-40%
- Target? – 120/80 or↓
- Diuretics alone +ACE inhib

How to prevent the 2nd stroke



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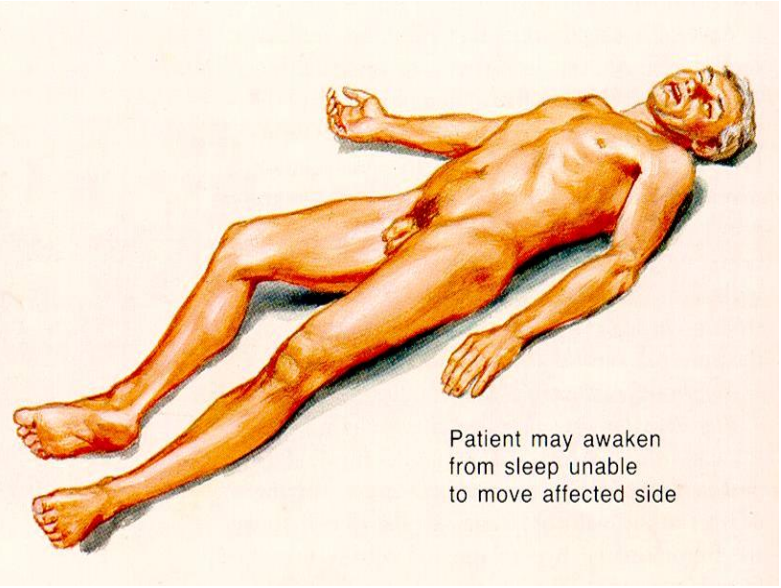
   70-99% stenosis

   TIA and minor stroke

3.Statin

- Statin if ≥LDL 2.6 mmol/l or signs of atherosclerosis
- target: LDL < 1.8 mmol/l
- Niacin or gemfibrosil if HDL-C low

How to prevent the 2nd stroke



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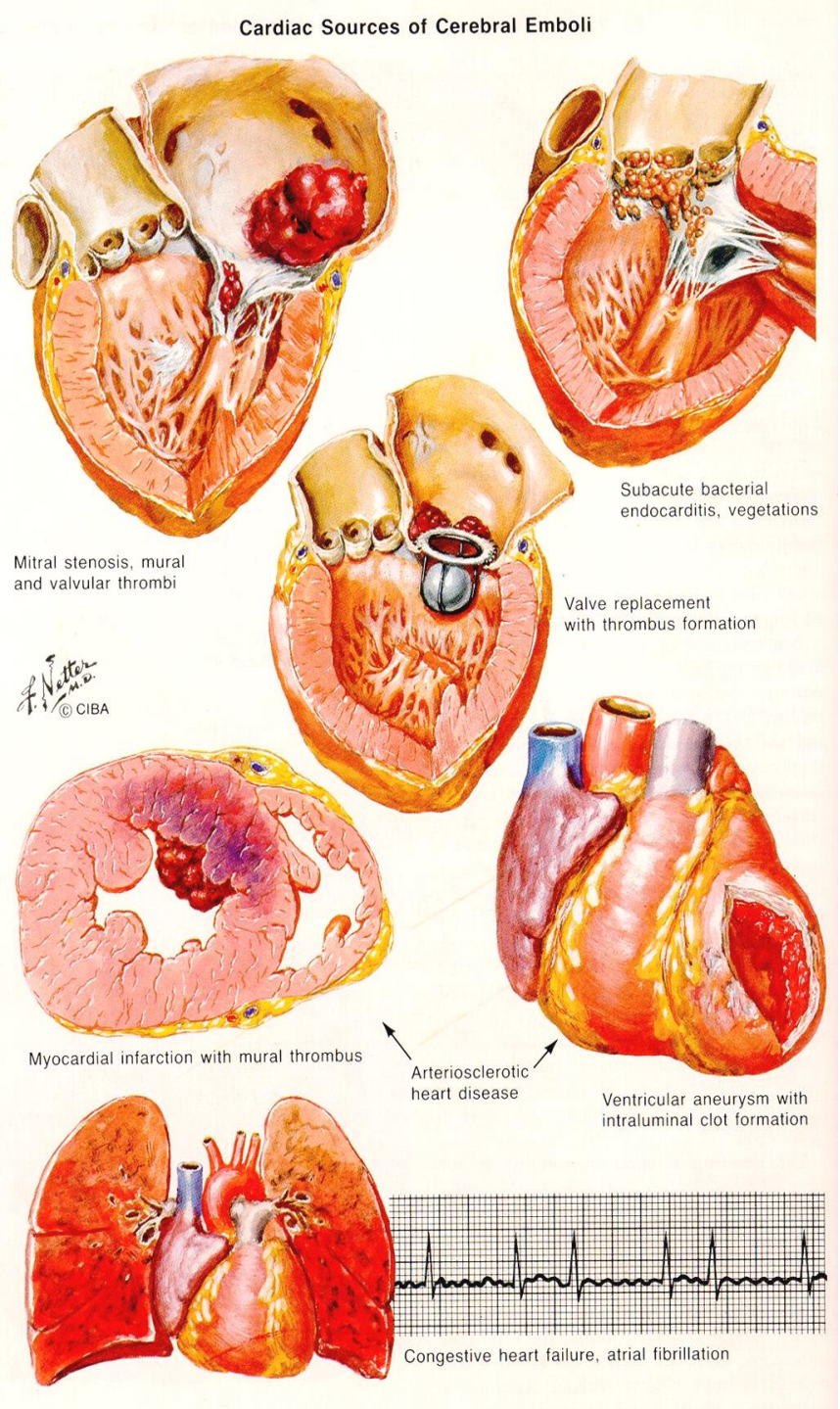
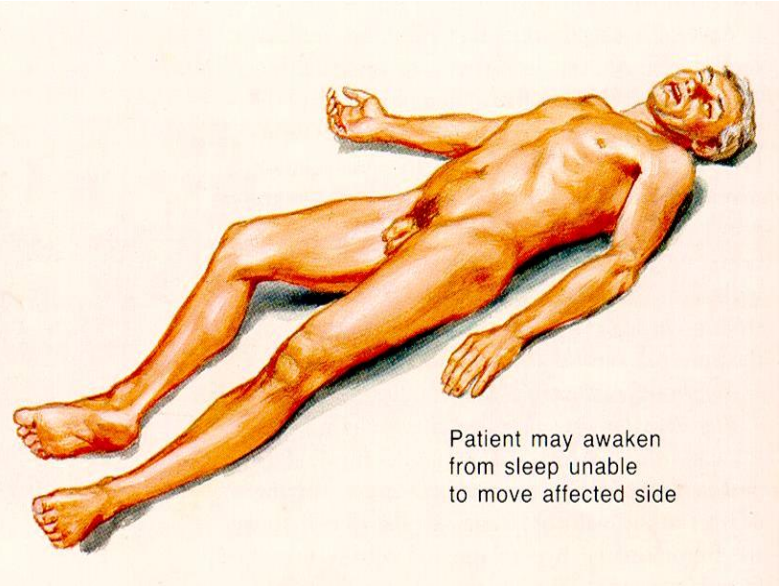

Cardiac Sources of Cerebral Emboli
Mitral stenosis, mural and valvular thrombi
Subacute bacterial endocarditis, vegetations
Valve replacement with thrombus formation
F. Netter M.D.
© CIBA
Myocardial infarction with mural thrombus
Arteriosclerotic heart disease
Ventricular aneurysm with intraluminal clot formation
Congestive heart failure, atrial fibrillation

Netter, 1986

4. If AF post-stroke

- Aspirin is NOT enough---if possible -----AC with
  - warfarin
  - or NOAC (dabigatran, rivaroxaban, apixaban, edoxaban----all as good as warfarin but less bleeding and no need for INR control)

How to prevent the 2nd stroke



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   70-99% stenosis

   TIA and minor stroke

Carotid Stent or CEA after TIA or minor stroke

If
Periprocedural
stroke+mort.
<6%

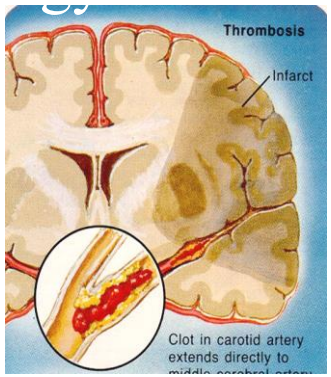
>70 y CEA>CAS

<70 év

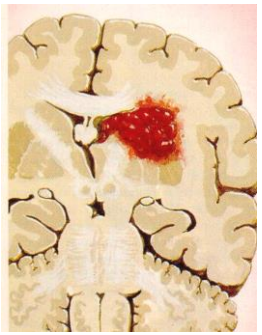
BUT! Analyse the carotid plaque!!!!

Differential diagnosis---CT/MRI!!!

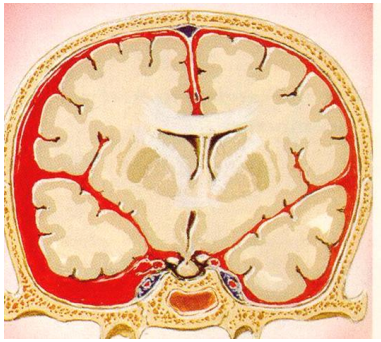
ischemia 80%



sp. Hemorrhage 10-15%

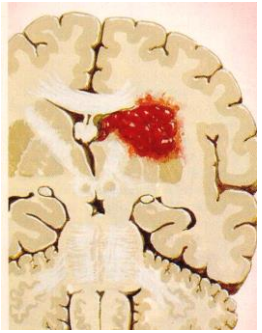


Subarachnoidal bleeding

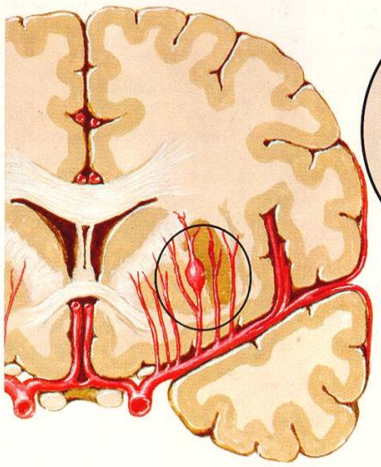


Hemorrhage

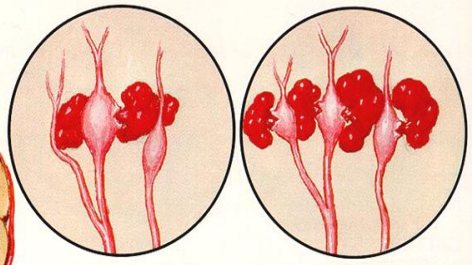
sp. Hemorrhage 10-15%



Intracerebral Hemorrhage (Hypertensive): Pathogenesis

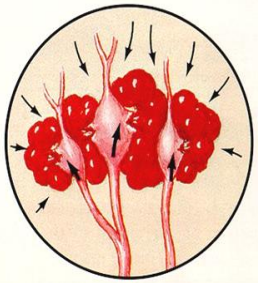


A. Microaneurysm formed in parenchymal artery of brain as result of hypertension. Lenticulostriate vessels (shown) most commonly involved, but similar process may occur in other parts of brain, especially lobar white matter, thalamus, pons and cerebellum



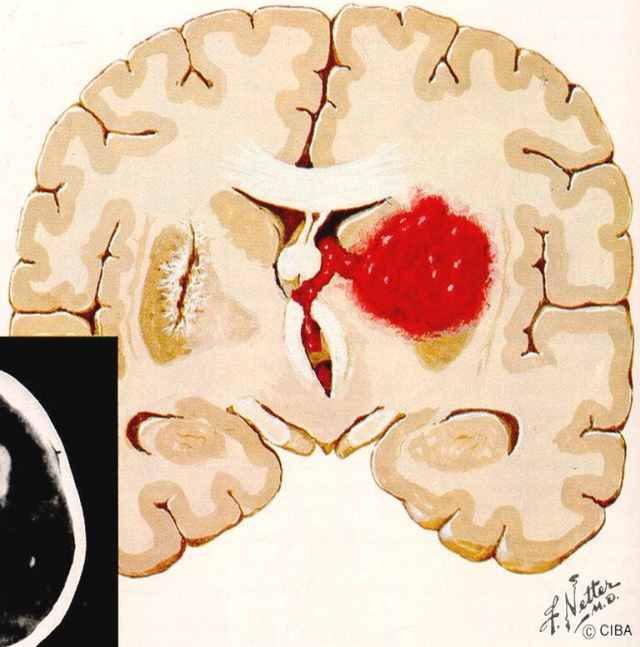
B. Microaneurysm ruptures, causing pressure on adjacent (satellite) vessels

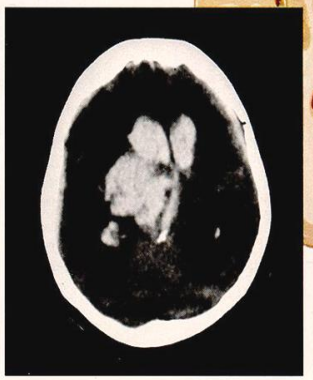
C. Satellite vessels rupture



D. Amount of blood extravasated into brain tissue depends on tissue turgor opposed to intravascular blood pressure

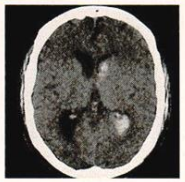
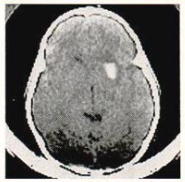
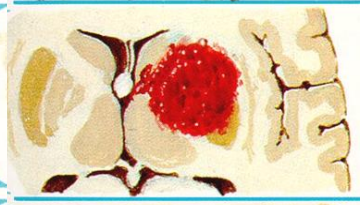
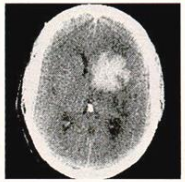
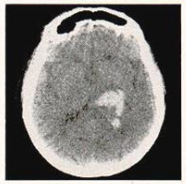
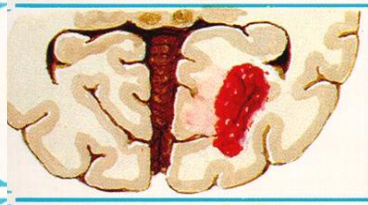
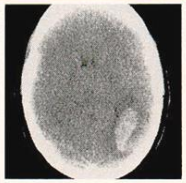
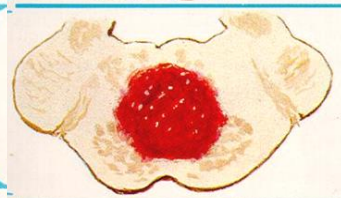
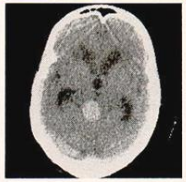
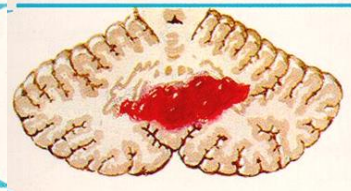
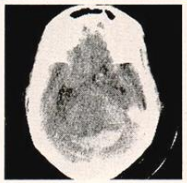
Moderate-sized intracerebral hemorrhage involving left putamen, with rupture into lateral ventricle. Brain distorted to opposite side. Scar of healed hemorrhage on right side





CT scan showing large putaminal hemorrhage with blood in ventricles

Netter, 1986

Pathology	CT scan	Pupils	Eye movements	Motor and sensory deficits	Other
Caudate nucleus (blood in ventricle)			Sometimes ipsilaterally constricted	Conjugate deviation to side of lesion. Slight ptosis	Contralateral hemiparesis, often transient	Headache, confusion
Putamen (small hemorrhage)			Normal	Conjugate deviation to side of lesion	Contralateral hemiparesis and hemisensory loss	Aphasia (if lesion on left side)
Putamen (large hemorrhage)			In presence of herniation, pupil dilated on side of lesion	Conjugate deviation to side of lesion	Contralateral hemiparesis and hemisensory loss	Decreased consciousness
Thalamus			Constricted, poorly reactive to light bilaterally	Both lids retracted. Eyes positioned downward and medially. Cannot look upward	Slight contralateral hemiparesis, but greater hemisensory loss	Aphasia (if lesion on left side)
Occipital lobar white matter			Normal	Normal	Mild, transient hemiparesis	Contralateral hemianopsia
Pons			Constricted, reactive to light	No horizontal movements. Vertical movements preserved	Quadriplegia	Coma
Cerebellum			Slight constriction on side of lesion	Slight deviation to opposite side. Movements toward side of lesion impaired, or sixth cranial nerve palsy	Ipsilateral limb ataxia. No hemiparesis	Gait ataxia, vomiting

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ICH

- neuroimaging with CT or MRI
- CT angiography and contrast CT may be considered to identify patients at risk for hematoma expansion
  - structural lesions?
  - vascular malformations?
  - tumors?

VII

- FVIIa can limit the extent of hematoma expansion
- increase in thromboembolic risk with rFVIIa
- rFVIIa is not recommended
- intermittent pneumatic compression +elastic stockings
- *After cessation of bleeding, low-dose sc. LMW heparin or unfractionated heparin may be considered for prevention of DVT with lack of mobility after 1 to 4 days from onset!!!!*

Guidelines for the Management of Spontaneous Intracerebral Hemorrhage ASA/AHA 2015

Blood Pressure	For ICH patients presenting with SBP between 150 and 220 mm Hg and without contraindication to acute BP treatment, acute lowering of SBP to 140 mm Hg is safe *(Class I; Level of Evidence A)* and can be effective for improving functional outcome *(Class IIa; Level of Evidence B)*. (Revised from the previous guideline)
General Monitoring and Nursing Care	Initial monitoring and management of ICH patients should take place in an intensive care unit or dedicated stroke unit with physician and nursing neuroscience acute care expertise *(Class I; Level of Evidence B)*. (Revised from the previous guideline)

(*Stroke*. 2015;46:2032-2060. DOI: 10.1161/STR.0000000000000069.)

Intravenous Medications That May Be Considered for Control of Elevated Blood Pressure in Patients with ICH

Drug	Intravenous Bolus Dose	Continuous Infusion Rate
Labetalol	5 to 20 mg every 15 min	2 mg/min (maximum 300 mg/d)
Nicardipine	NA	5 to 15 mg/h
Esmolol	250 μg/kg IVP loading dose	25 to 300 $\mu g \cdot kg^{-1} \cdot min^{-1}$
Enalapril	1.25 to 5 mg IVP every 6 h*	NA
Hydralazine	5 to 20 mg IVP every 30 min	1.5 to 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$
Nipride	NA	0.1 to 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$
Nitroglycerin	NA	20 to 400 μg/min

Prevention of Secondary Brain Injury

- Glucose should be monitored and normoglycemia (range 4.4 to 6,1 mmol/L)
- seizures should be treated with antiepileptic drugs
- Prophylactic anticonvulsant medication should not be used

Surgery

- If Glasgow Coma Scale of 8 +clinical evidence of transtentorial herniation,
- or significant intraventric. Hemorrhage (IVH)
- or hydrocephalus
- ventricular drainage in patients with decreased level of consciousness

Surgery 2

- uncertain
- exceptions deteriorating cerebellar hemorrhage
  - with brainstem compression
  - and/or hydrocephalus ASAP!

Guidelines for the Management of Spontaneous Intracerebral Hemorrhage ASA/AHA 2015

Management of Medical Complications	A formal screening procedure for dysphagia should be performed in all patients before the initiation of oral intake to reduce the risk of pneumonia *(Class I; Level of Evidence B)*. (New recommendation)
Surgical Treatment of ICH	Patients with cerebellar hemorrhage who are deteriorating neurologically or who have brainstem compression and/or hydrocephalus from ventricular obstruction should undergo surgical removal of the hemorrhage as soon as possible *(Class I; Level of Evidence B)*. (Unchanged from the previous guideline)

(*Stroke*. 2015;46:2032-2060. DOI: 10.1161/STR.0000000000000069.)

Clot removal

- lobar clots >30 ml and within1 cm of the surface, by standard craniotomy might be considered
- The effectiveness
  - of minimally invasive clot evacuation
    - stereotactic
    - or endoscopic aspiration with or without thrombolytic usage is uncertain
  - no clear evidence

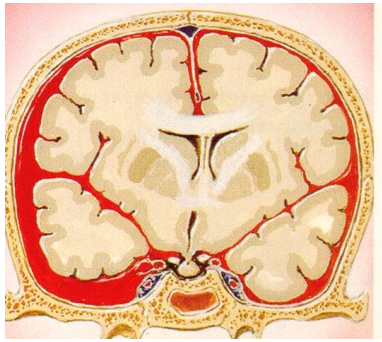
Prevention of Recurrent ICH

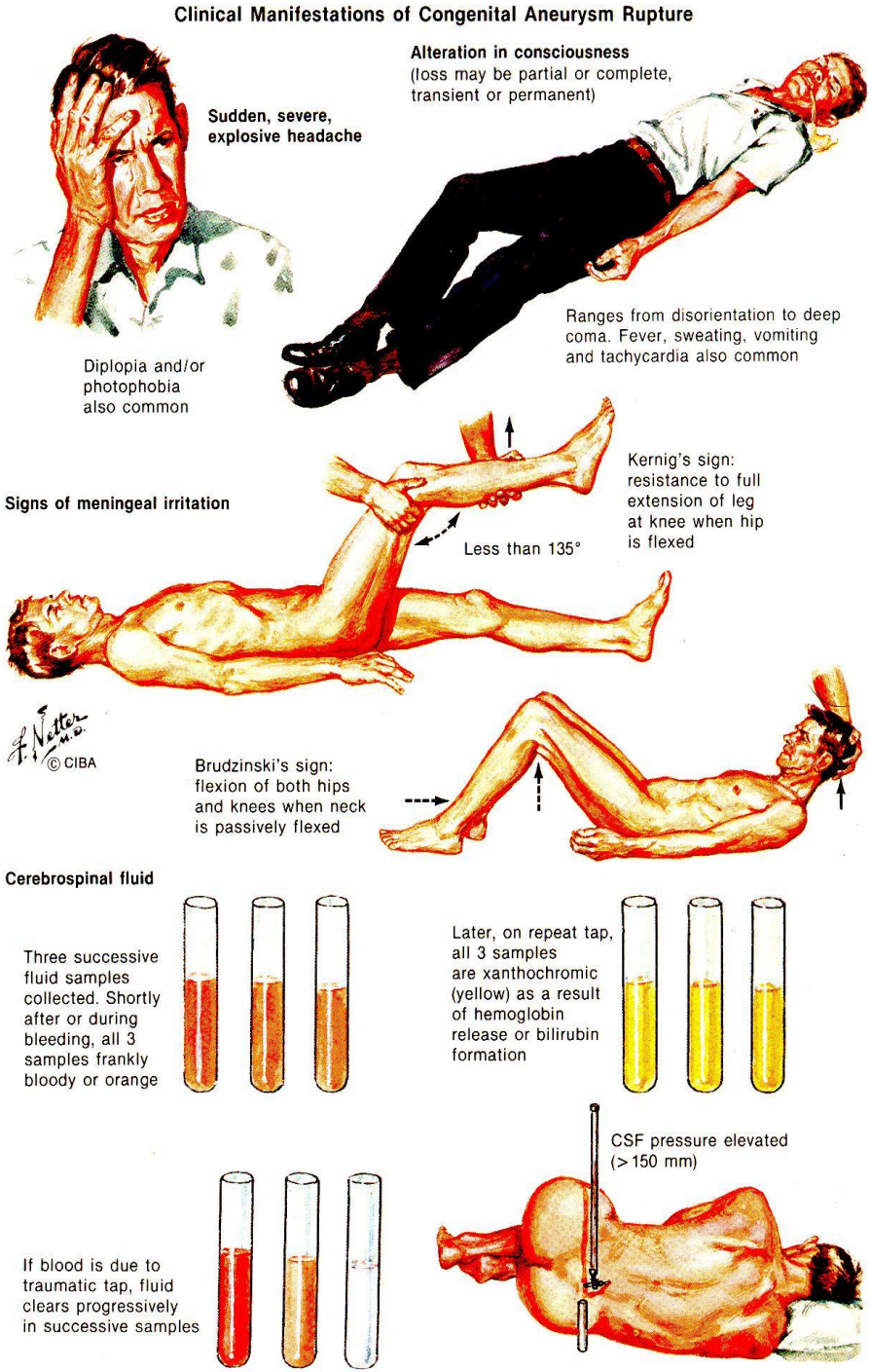
- risk factors for recurrence:
  - Lobar location
  - older age,
  - ongoing anticoagulation,
  - presence of the apolipoprotein E ε2 or ε4 alleles,
  - and greater number of microbleeds on MRI!!
- After the acute ICH period BP <140/90 <130/80 if diabetes or chronic kidney disease, reasonable

What to do if concomittant risk for cardiogenic stroke exists after ICH

- Anticoagulation after lobar ICH NO (but antiplatelet) due to high risk of recurrence
- Anticoagulation YES after non lobar ICH (e.g. basal ganglia)
- No heavy alcohol use
- insufficient data
  - use of statin?
  - physical or sexual activity

Subarachnoidal bleeding





SAH

10-20/100 000

- Thunderclap headache
- Vomitus, photophobia
- During physical exercise

But not always!

- Neck rigidity
- sometimes paresis

ACT sometimes negative!! Lp!
aneurysma multiplex?
Vasospasm 4-11 day

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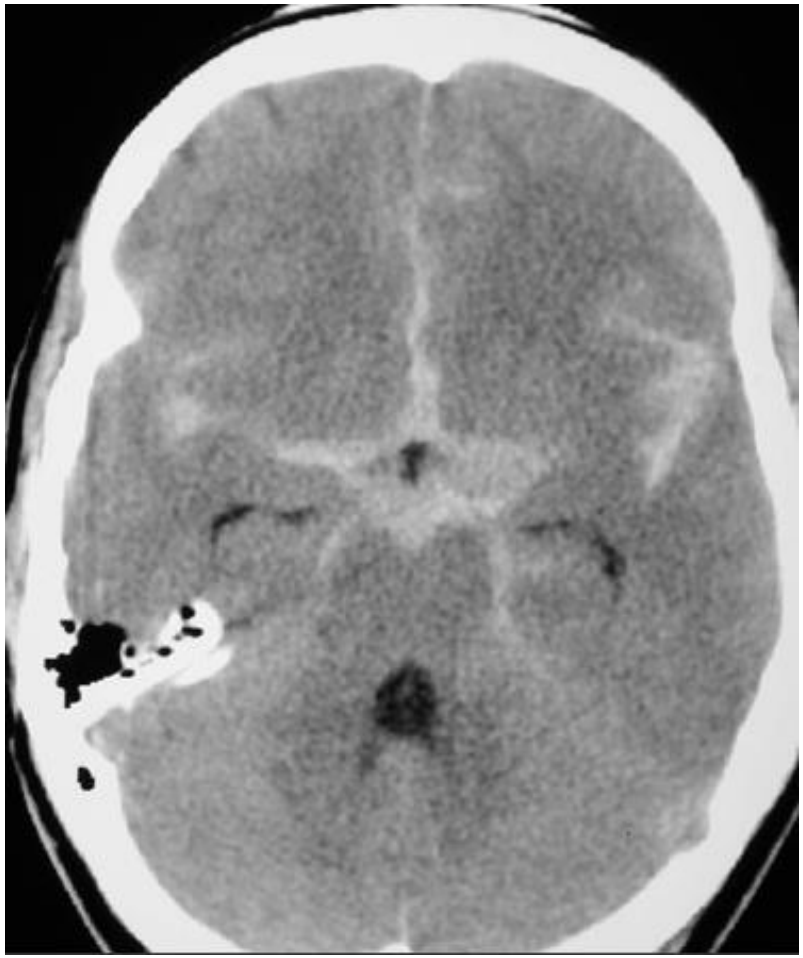
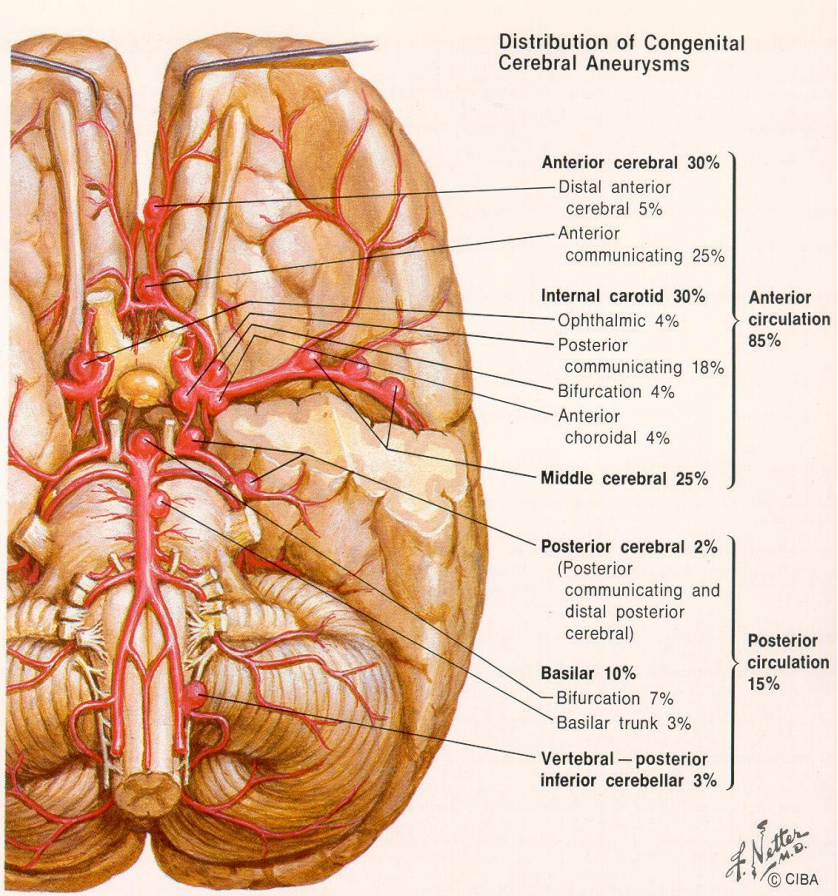
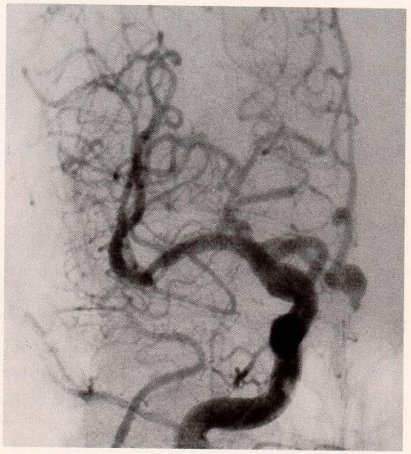
The classic presentation of SAH can include the following:

- Sudden onset of severe headache (the classic feature)
- Accompanying nausea or vomiting
- Symptoms of meningeal irritation
- Photophobia and visual changes
- Focal neurologic deficits
- Sudden loss of consciousness at the ictus
- Seizures during the acute phase

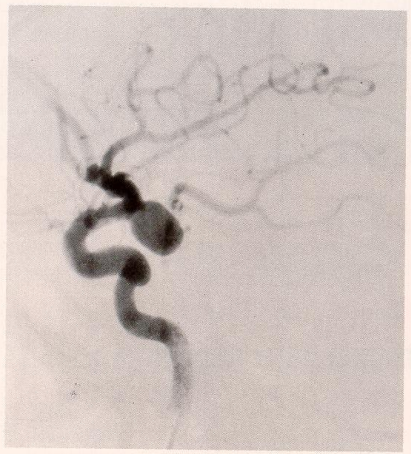
SAH







Frontal carotid arteriogram disclosing bilobate aneurysm of anterior communicating artery



Different patient: lateral view showing large aneurysm of internal carotid artery at origin of posterior communicating artery

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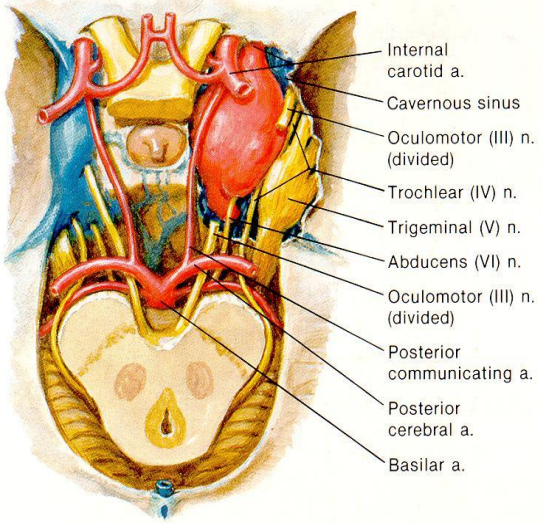
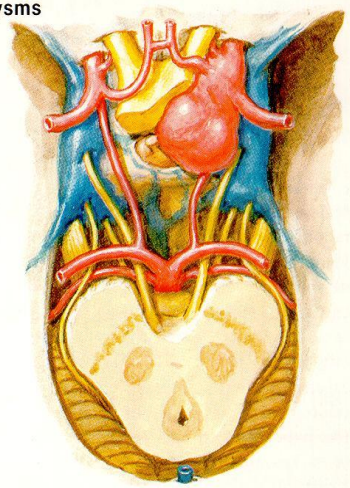
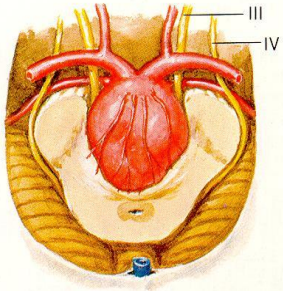
Giant Congenital Aneurysms



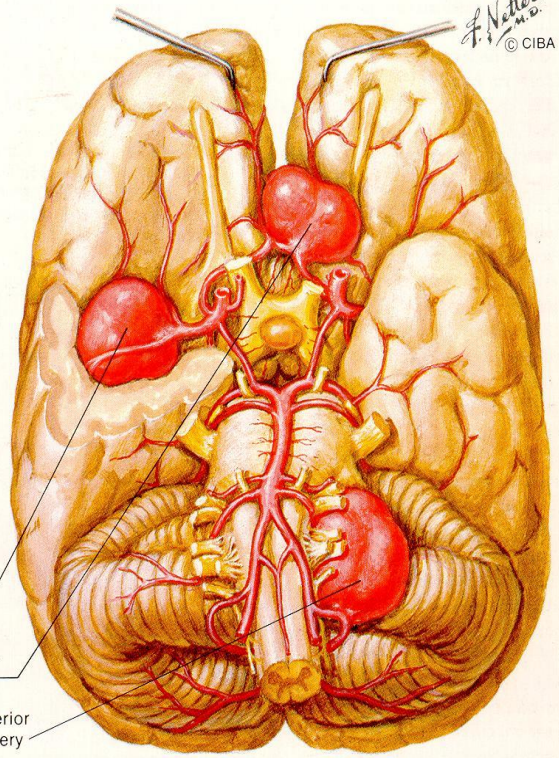
A. Intracavernous (infraclinoid) internal carotid aneurysm compressing abducens (VI) nerve. Oculomotor (III), trochlear (IV) and trigeminal (V) nerves may also be affected. Trigeminal involvement may cause facial pain



B. Aneurysm of supraclinoid segment of internal carotid artery elevating optic chiasm, distorting infundibulum and compressing oculomotor (III) nerve



C. Aneurysm of basilar bifurcation projecting posteriorly, invading peduncles and compressing cerebral aqueduct. Corticospinal tracts may be affected, resulting in paralysis or paresis



D. Aneurysm of middle cerebral artery

E. Aneurysm of anterior cerebral–anterior communicating arteries

F. Aneurysm of posterior inferior cerebellar artery

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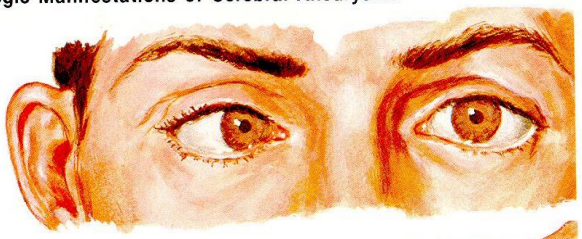
Ophthalmologic Manifestations of Cerebral Aneurysms

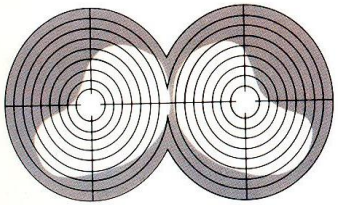
A. Neuromuscular disorders

Abducens nerve palsy: affected eye turns medially. May be first manifestation of intracavernous carotid aneurysm. Pain above eye or on side of face may be secondary to trigeminal (V) nerve involvement

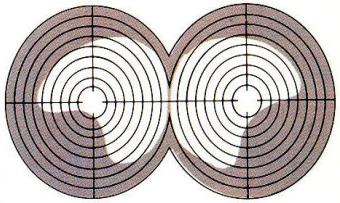


Oculomotor nerve palsy: ptosis, eye turns laterally and inferiorly, pupil dilated. Common finding with cerebral aneurysms, especially carotid-posterior communicating aneurysms

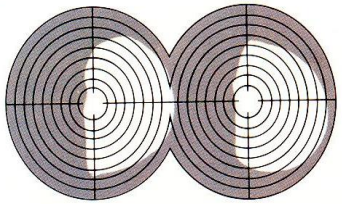
B. Visual field disturbances



Superior bitemporal quadrantanopia caused by supraclinoid carotid aneurysm compressing optic chiasm from below



Inferior bitemporal quadrantanopia caused by compression of optic chiasm from above



Right (or left) homonymous hemianopsia caused by compression of optic tract. Unilateral amaurosis may occur if optic (II) nerve is compressed

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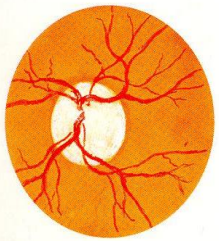
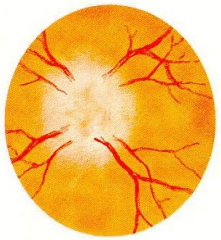
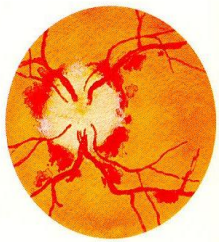
C. Retinal changes



Optic atrophy may develop as result of pressure on optic (II) nerve from a supraclinoid carotid, ophthalmic or anterior cerebral aneurysm



Papilledema may be caused by increased intracranial pressure secondary to rupture of cerebral aneurysm



Hemorrhage into optic (II) nerve sheath after rupture of aneurysm may result in subhyaloid hemorrhage, with blood around disc

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Signs present before SAH include the following:

- Sensory or motor disturbance (6%)
- Seizures (4%)
- Ptosis (3%)
- Bruits (3%)
- Dysphasia (2%)

Complications of SAH include the following:

- Hydrocephalus
- Rebleeding
- Vasospasm
- Seizures
- Cardiac dysfunction

Diagnosis

- clinical suspicion combined with
- noncontrast CT,
- followed by lumbar puncture
- or CT angiography of the brain.
- further imaging to characterize the source of the hemorrhage.

Laboratory studies

- Complete blood count
- Prothrombin time (PT)/activated partial thromboplastin time (aPTT)
- Blood typing/screening
- Cardiac enzymes
- Arterial blood gas (ABG) determination

Imaging studies

- CT (noncontrast, contrast, or infusion)
- Digital subtraction cerebral angiography
- Multidetector CT angiography
- MRI (if no lesion is found on angiography)
- Magnetic resonance angiography (MRA; investigational for SAH)

Other diagnostic studies that may be warranted are as follows:

- Baseline chest radiograph
- ECG on admission
- Lumbar puncture and CSF analysis

HUNT AND HESS SCALE.

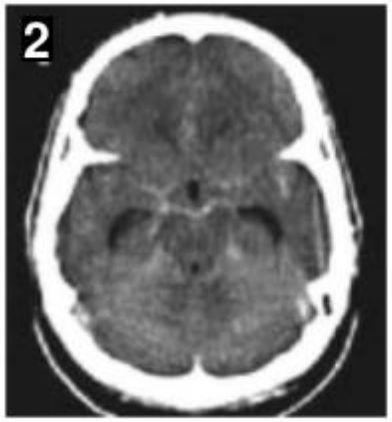
I - Asymptomatic or with mild headache

II-Moderate or severe headache, nuchal rigidity

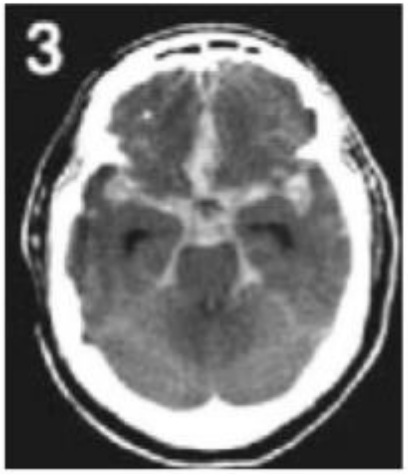
III-Confusion, drowsiness, or mild focal deficit (discounting third nerve palsy)

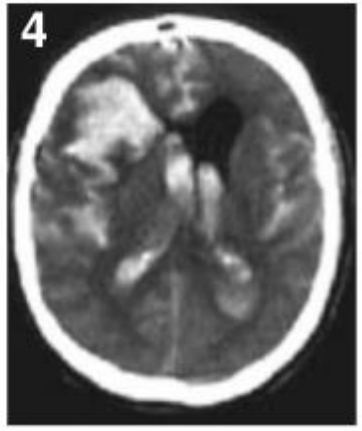
IV-Stupor or hemiparesis, early decerebrate rigidity

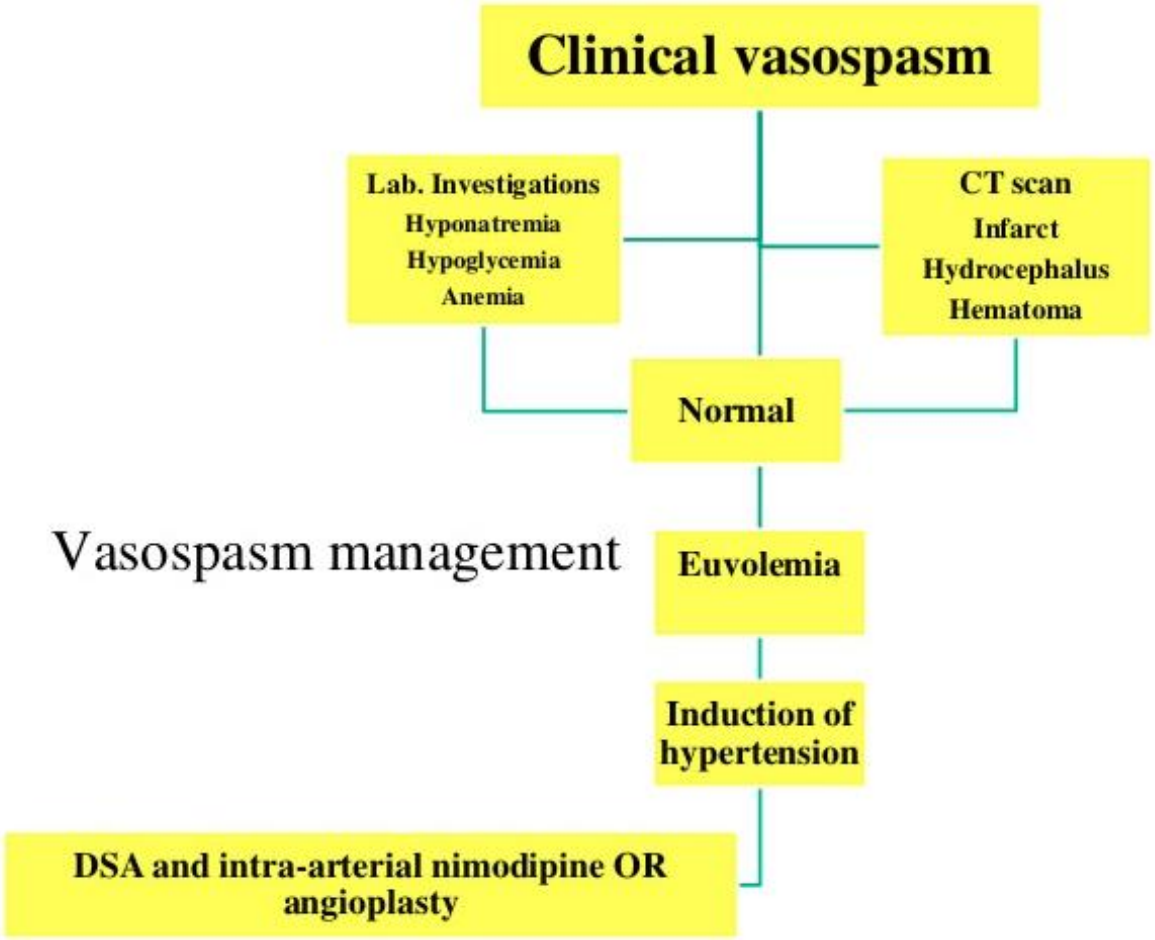
V-Deep coma, extensor posturing

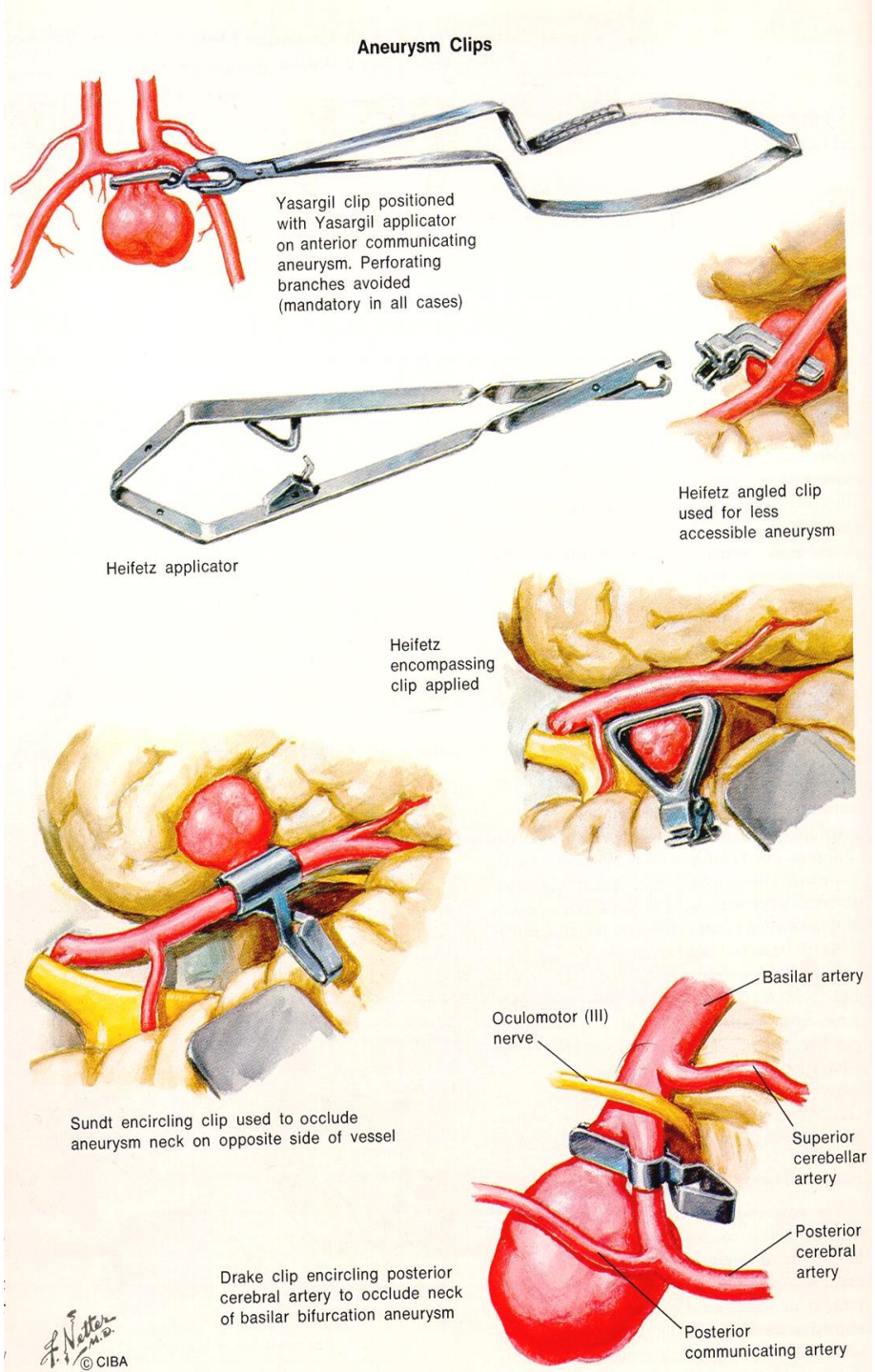


Fisher's Grade






Clinical vasospasm
Lab. Investigations
Hyponatremia
Hypoglycemia
Anemia
CT scan
Infarct
Hydrocephalus
Hematoma
Normal
Vasospasm management
Euvolemia
Induction of hypertension
DSA and intra-arterial nimodipine OR angioplasty


Aneurysm Clips
Yasargil clip positioned with Yasargil applicator on anterior communicating aneurysm. Perforating branches avoided (mandatory in all cases)
Heifetz angled clip used for less accessible aneurysm
Heifetz applicator
Heifetz encompassing clip applied
Sundt encircling clip used to occlude aneurysm neck on opposite side of vessel
Basilar artery
Oculomotor (III) nerve
Superior cerebellar artery
Posterior cerebral artery
Drake clip encircling posterior cerebral artery to occlude neck of basilar bifurcation aneurysm
Posterior communicating artery
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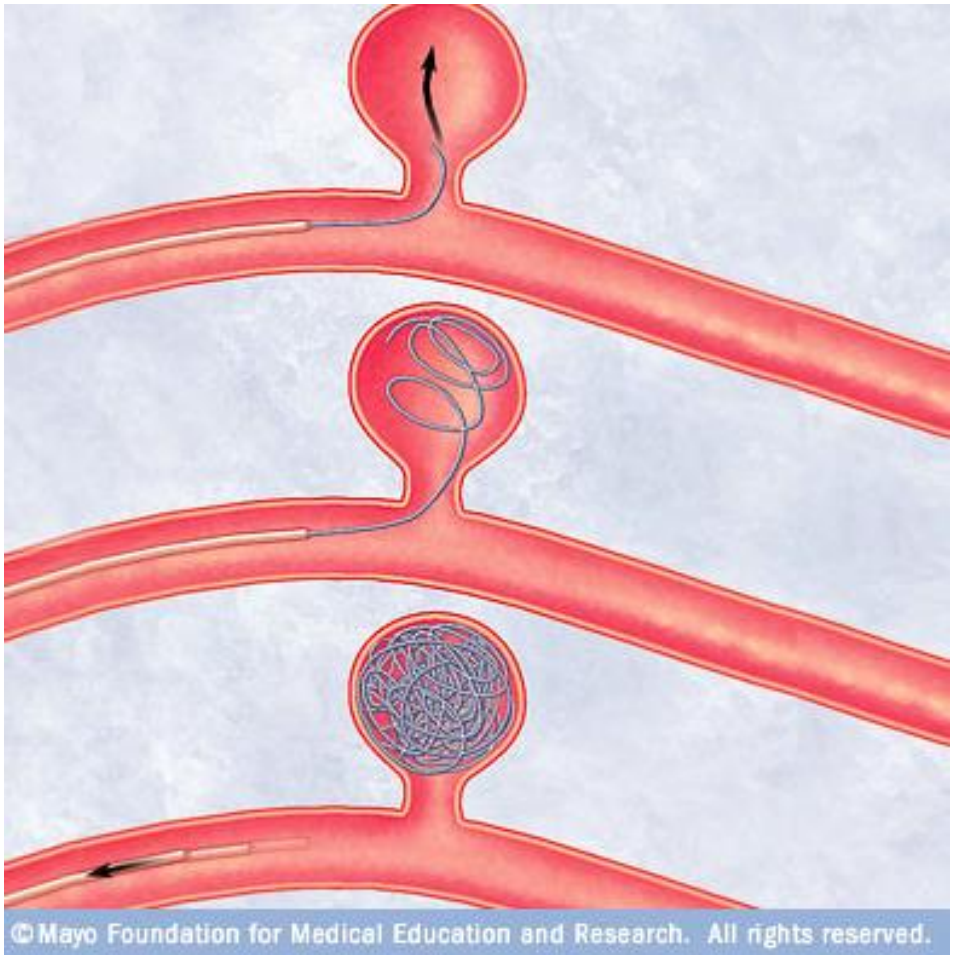
Aneurysma sac is filled



Therapy

- Anti-HT (eg, IV beta block.) if mean BP more than 130
- No Nitrite!
- Hydralazine and calcium calcium blocker
- ACE
- Intubation hiperventilation
- Mannisol
- furosemide
- IV steroid (contradict.)

Complications

- Re-bleeding
- Vasospasm (4-11. nap, Transcran. Doppler) nimodipine
- Hydrocephalus
- Hyponatremia
- Seizures
- Lung edema, MI

Others

- Clipping the ruptured aneurysm
- Endovascular treatment
- The choice between coiling and clipping usually depends on the location of the lesion, the neck of the aneurysm, and the availability and experience of hospital staff.
- Screening is not recommended in the general population.
- However, it can lower cost and improve quality of life in patients at relatively high risk for aneurysm formation and rupture.
- Vasospasm between 4-11th after rupture! Transcranial Doppler