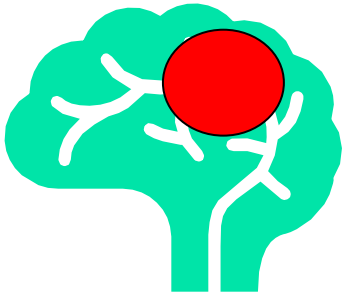
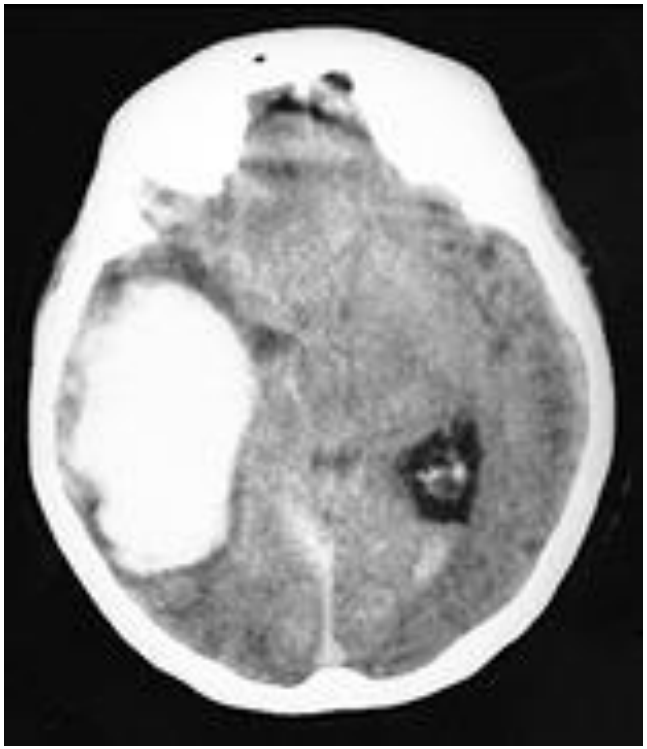
Intracerebral hemorrhage (ICH)

=Hemorrhagic stroke
Non-traumatic intracerebral haemorrhage
Spontaneous bleeding into the brain tissue



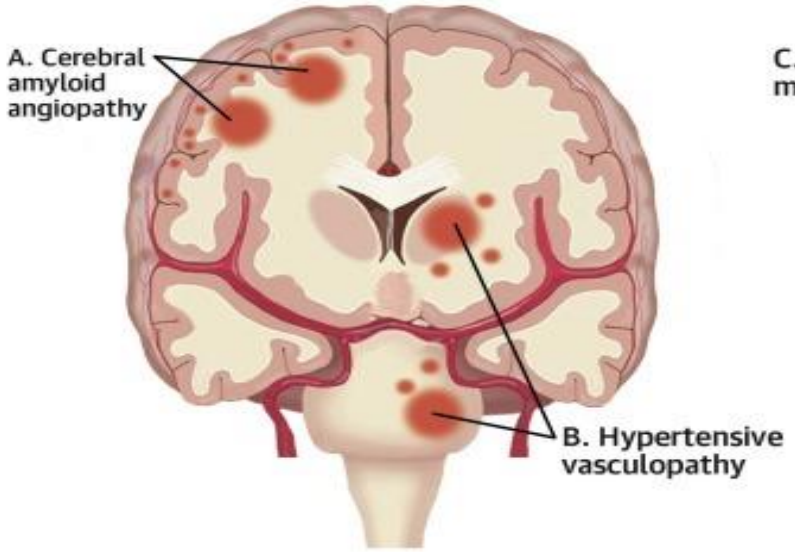


ICH: CT

HIGHLIGHTS

- ICH, although less common than ischemic stroke, is the major cause of stroke mortality and there is not yet a definitive therapy beyond supportive care.
- Early stabilization of acute ICH involves identifying and treating the cause(s) of decreased alertness, aggressively correcting blood pressure, and reversing any coagulopathy.
- Long-term management of ICH requires correctly identifying the underlying etiology and, when possible, correcting it.

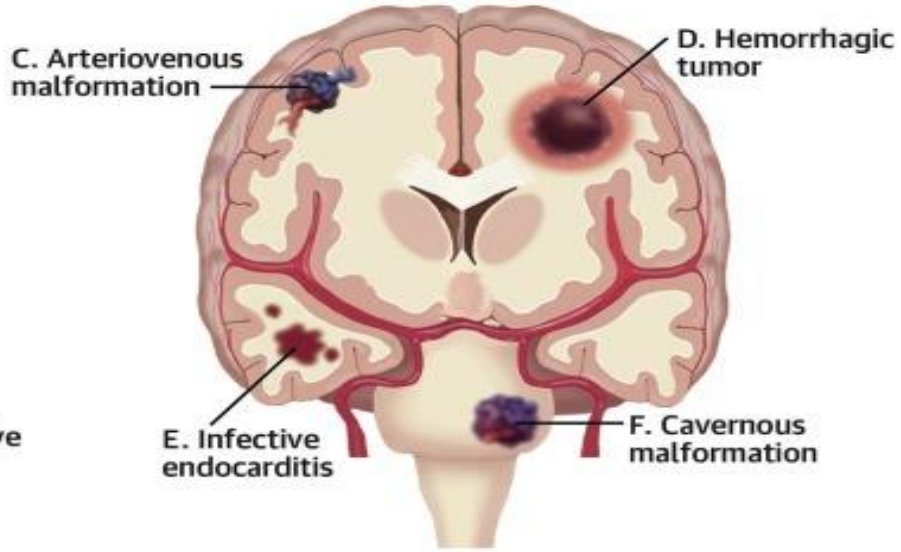
Primary Intracerebral Hemorrhage



Treatment Strategy

- Exclude secondary hemorrhage
- Control blood pressure and other risk factors
- Reduce or eliminate anticoagulant and/or antiplatelet therapies, depending on risk of re-occurence

Secondary Intracerebral Hemorrhage



Treatment Strategy

- Identify and treat underlying mechanism
- Magnetic resonance imaging with contrast and CT angiography for most
- Conventional angiogram when vascular malformation suspected, transesophageal echocardiography when endocarditis suspected

Schrag, M. et al. J Am Coll Cardiol. 2020;75(15):1819–31.

Causes

Spontaneous intracerebral bleeds

The second most common cause of stroke

Accounting for 20% of hospital admissions for stroke

High blood pressure raises the risk of spontaneous intracerebral hemorrhage by two to six times

Other causes

Head trauma

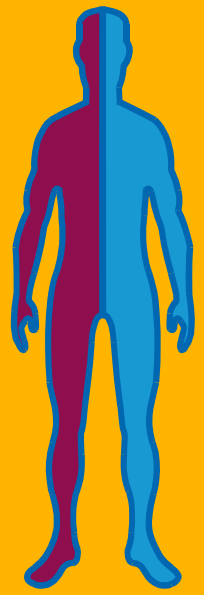
- Penetrating head trauma
- Depressed skull fractures
- Acceleration-deceleration trauma

Rupture of an aneurysm or arteriovenous malformation (AVM)

Bleeding within a tumor

A very small proportion is due to cerebral venous sinus thrombosis.

Signs and symptoms



Weakness, sensory disorder
Contralaterally

Blurred visoin

Speech disorders

Acute headache

Vertigo, dizziness

ICH

10-20% of strokes

High blood pressure 72- 81%

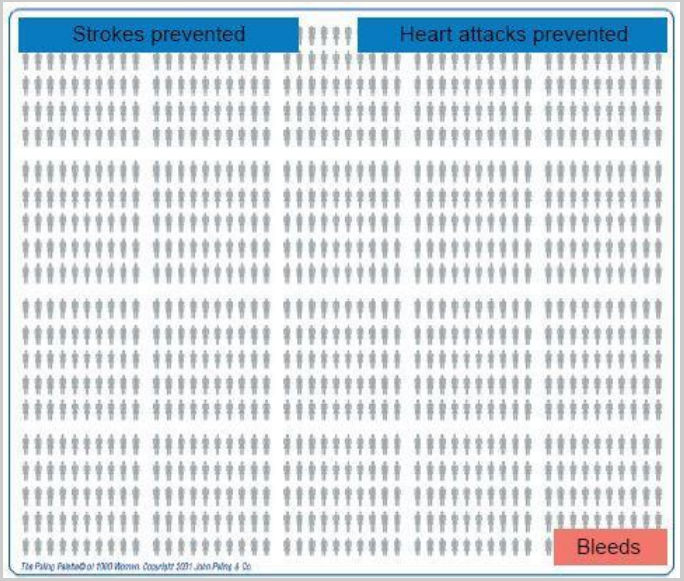
- Reduction of DBP (5, 7,5, 10 mm Hg) results to reduction of ICH (34%, 46%, 56%)
- Treatment of systolic HT in elderly is connected with 36% reduction of ICH.

Risk factors

Study ARIC 15 792 pts	• TKS 160 mm Hg or TKD 110 mm Hg • RR 5.55 (95% CI 3.07 to 10.0)
Other risk possible factors	• gender, smoking, drinking, BMI, waist cirkumference, diabetes
Swedish study 2006	• Smoking • Risk factor for lobar ICH only • Diabetes

Risk factors

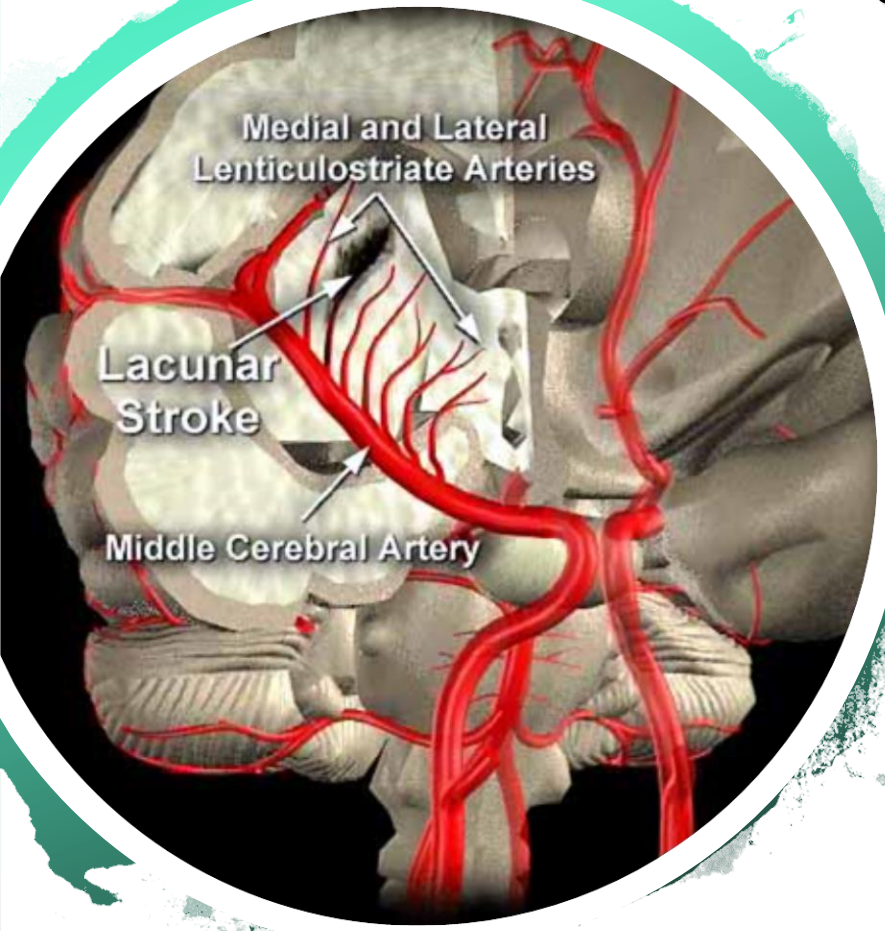
- ASA
  - Epistaxis
- Anticoagulation
  - Warfarin / DOAC
  - 2% of treated patiens
  - Risk of bleeding 8-11x Hypolipidemics
- Thrombolysis
  - 6,4%



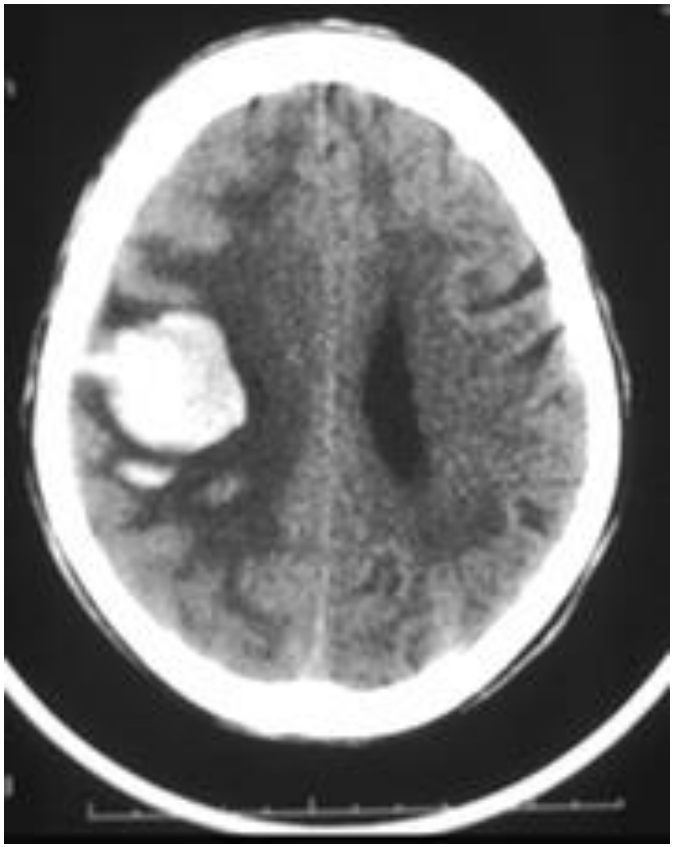
Pathophysiology

- Chronic hypertension produces a small vessel vasculopathy characterized by lipohyalinosis, fibrinoid necrosis, and development of Charcot-Bouchard aneurysms, affecting penetrating arteries

Localisation of ICH among pts with HBP

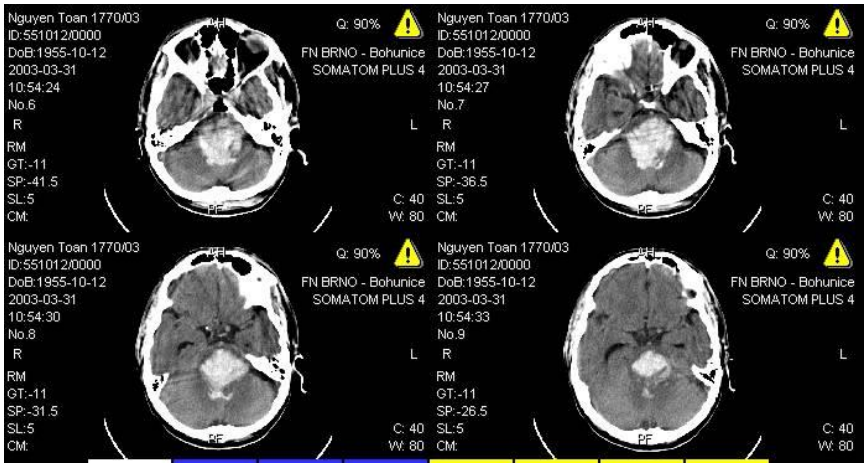


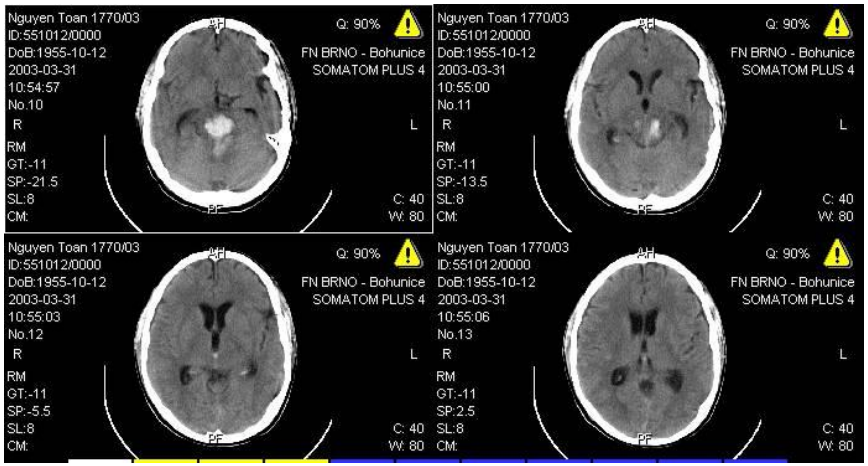
- 40- 50% Putamen
- 20% Lobar
- 15% Thalamus
- 8% Cerebellum
- 8% Pons
- 8% Nucleus caudatus



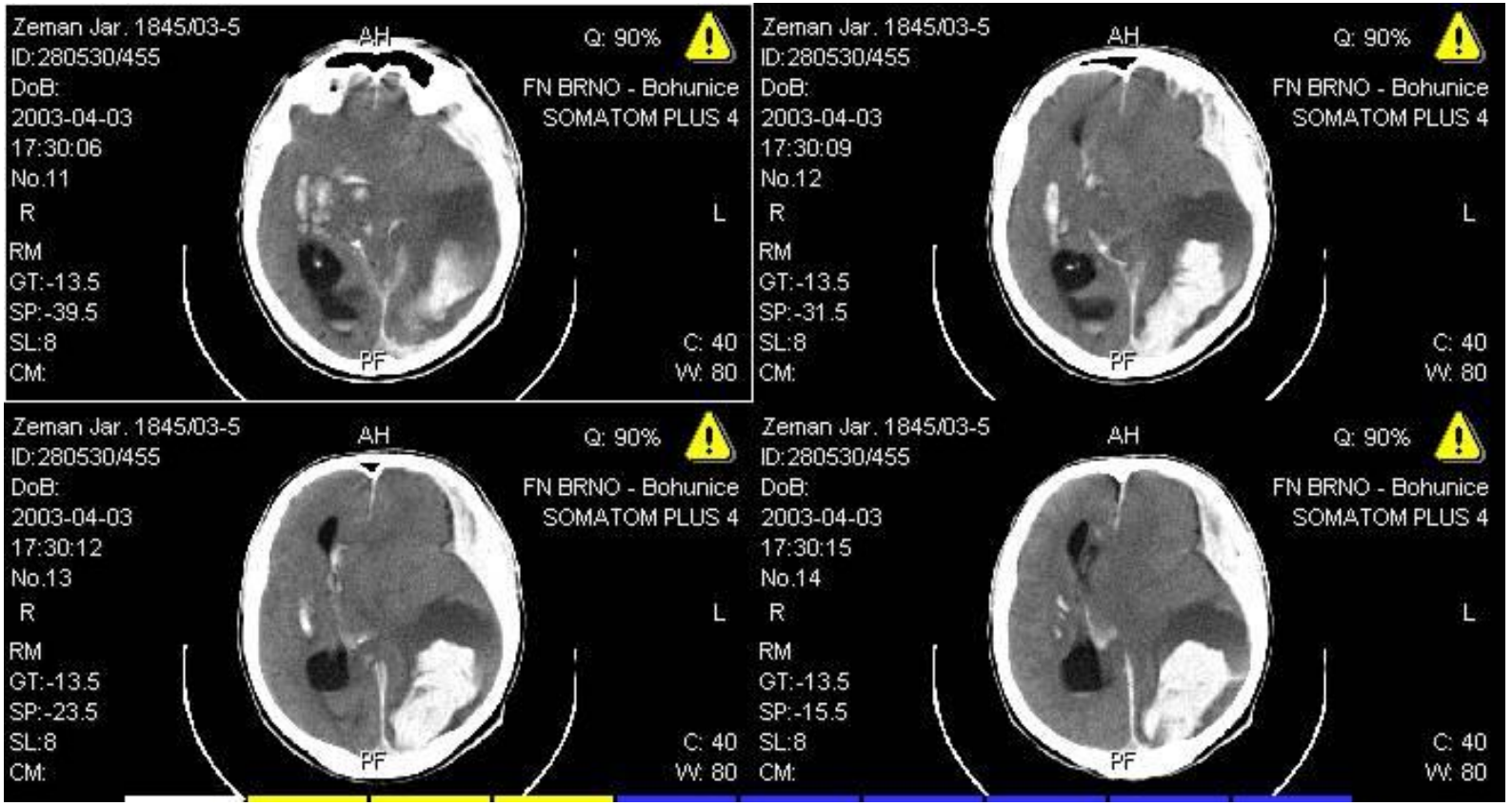
ICH: CT

Brain-stem haemorrhage

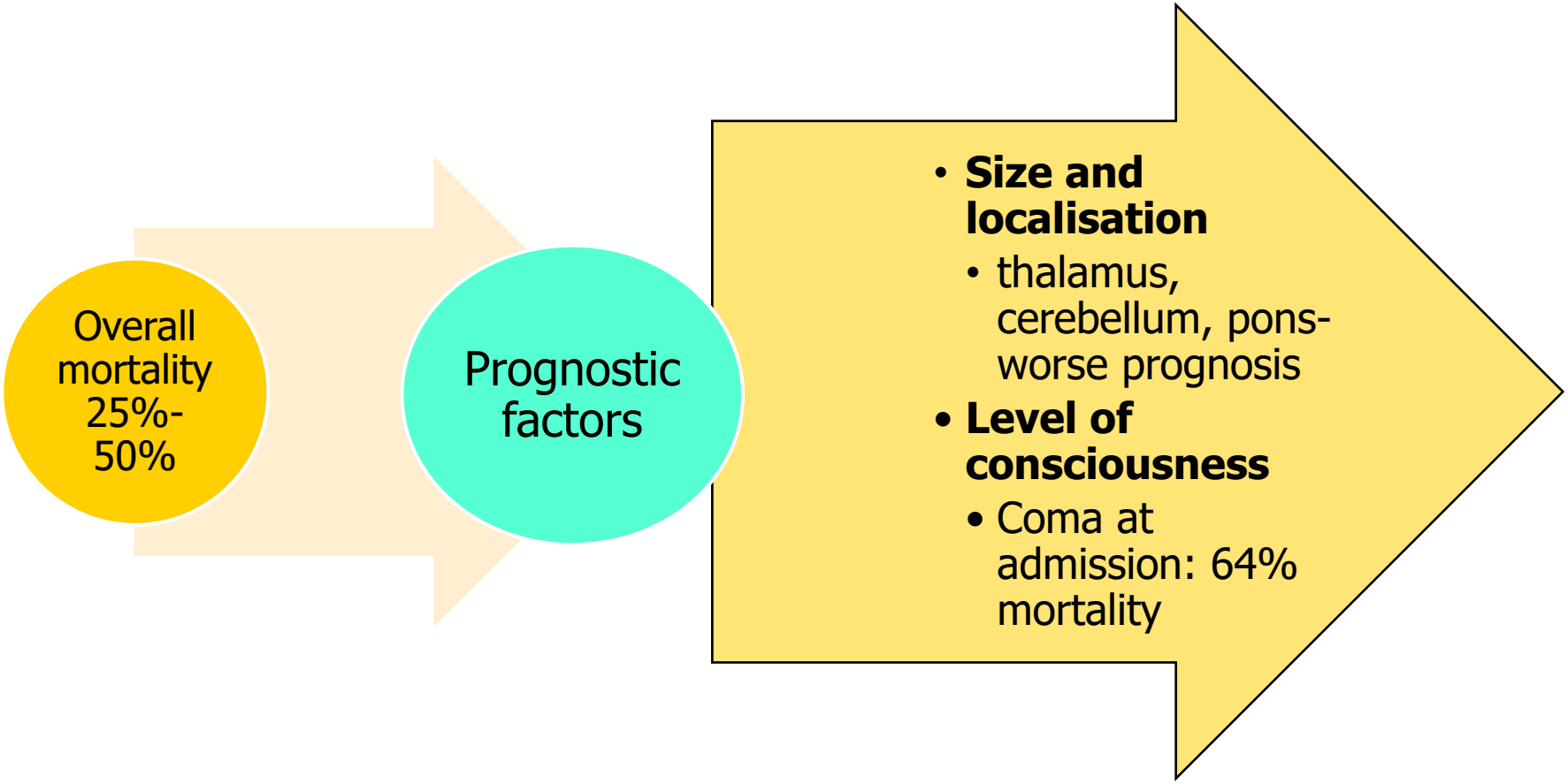




A-V Malformation



ICH: mortality and prognosis



ICH: Indication for surgery

- **Haematoma < 20 ml**: good prognosis regardless of therapy.
- **Haematoma > 60 ml**: bad prognosis regardless of therapy.
- **Surgical treatment**
  - Cerebellar ICH.
  - Gradual decrease of the level of the consciousness (haematoma > 20 ml).

Subarachnoideal haemorage

SAH

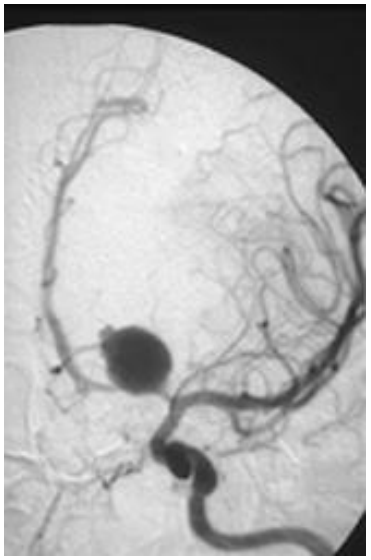
Incidence of aneurysmatic SAH

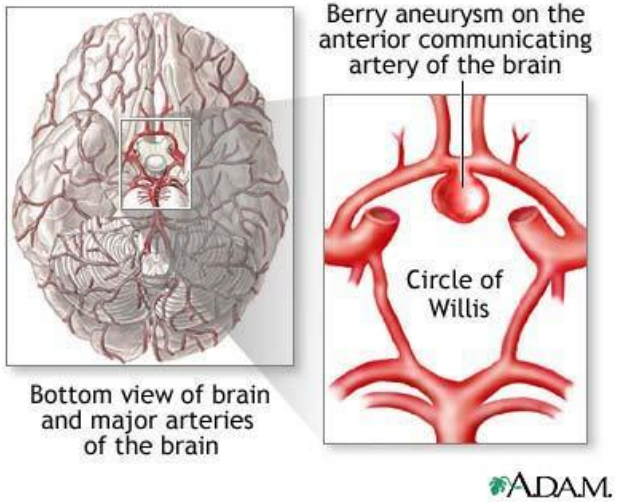
6/100 000/year

Etiology

85% spontaneous bleeding from the ruptured aneurysma

SAH: aneurysm A1





SAH-diagnosis

- **Acute headache**
  - Instantaneous severe headache, development during 1 min. lasting at least 1 hour. History of unusually severe headache that started suddenly.
- Absence of the neck stiffness doesn't exclude SAH !
  - Takes hours to develop and in some cases it is not present during whole course
- Loss of consciousness 50% pts.
- 40% headache and meningism only.

- SAH can be present in patient without
  - Meningeal signs
  - Focal signs and symptoms
  - Loss of consciousness

- **Should be SAH considered (and excluded) in all patients with acute (explosive) headache ?**
  - **YES**

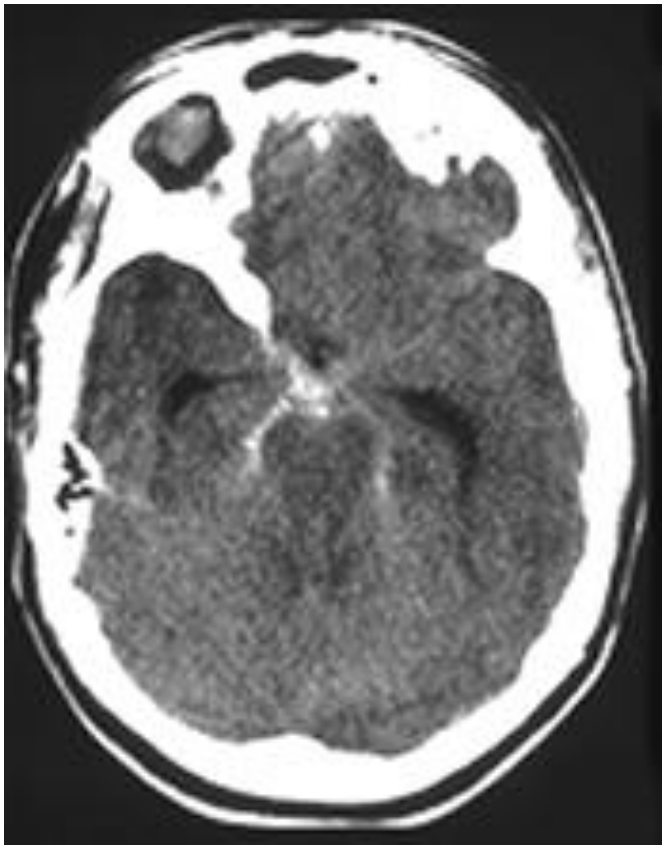
SAH- diagnosis

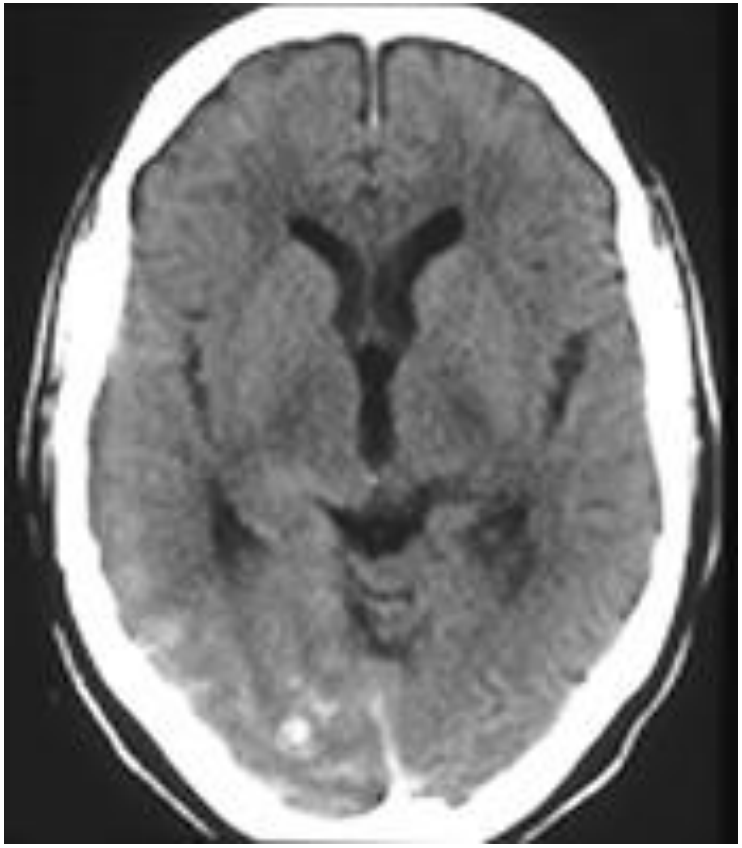
- **Thunderclap headache only**
  - **SAH 12%**
  - **Risk of ruptured aneurysm 6%.**
- **CT+CSF negative**
  - **SAH excluded and <u>angiography not indicated</u>**
- **Distribution of blood on CT scan can predict the absence of the aneurysm and vice versa**

CT imaging: metod of first choice

- 199 pts with the ruptured aneurysm- CT during first 12 h. negative only in 2 persons.
  - During first 6 h. (0.2)
  - Delayed examination – decreased sensitivity
- **If CT negative, LP indicated**
  - Spinal tap should be postpone at least 12 h. (xanthochromia= hemoglobin degradation products)
  - Discrimination of the artificial bleeding and hemorrhage due to SAH is visually not possible.
  - The reliable method is spectrophotometry
  - Test of three tubes doesn't exclude SAH
  - Finding of erytrophages confirms SAH
  - 12 h. after bleeding is xanthochromia present in all cases and lasts 2 weeks.

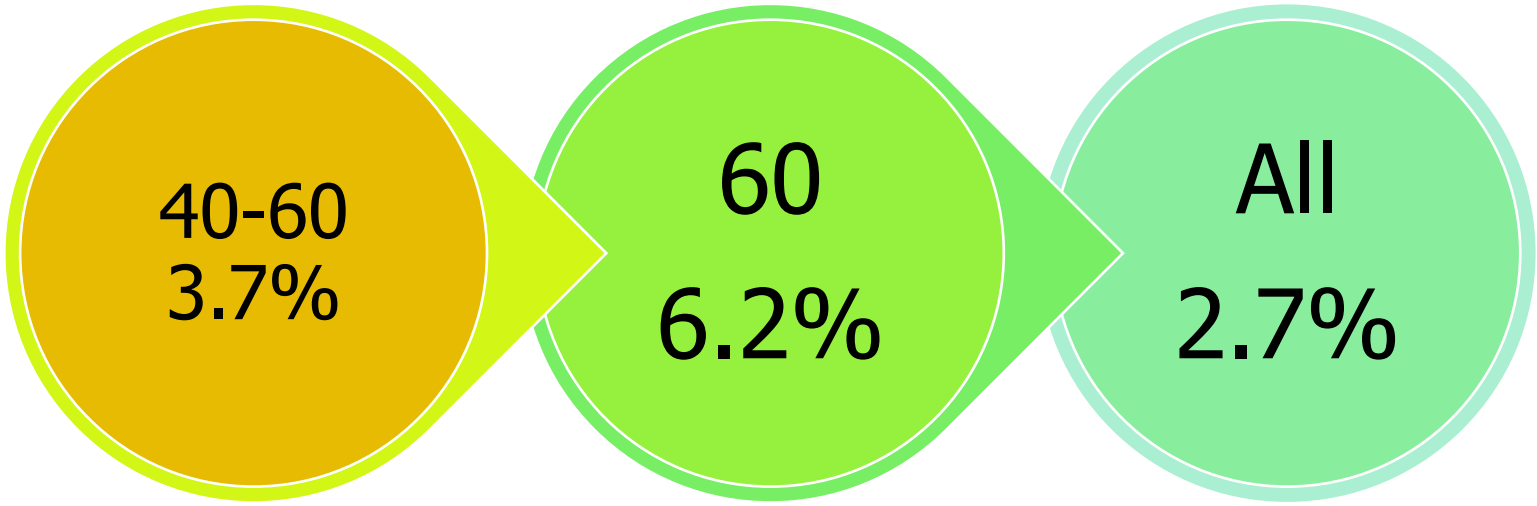
SAH: CT





85%	• Bleeding due to ruptured aneurysm
10%	• Non-aneurysmatic perimesencefalic bleeding
5%	• Other
Aneurysm is not inborn	• Among newborn never found, very rare in childhood • Hereditary predisposition exists: pts with positive familial history are younger, have more frequently multiple aneurysms or aneurysms on ACM

How many people has aneurysm?



Size of the aneurysm- annual risk of bleeding

10-25 mm

- 3-4%

< 10 mm

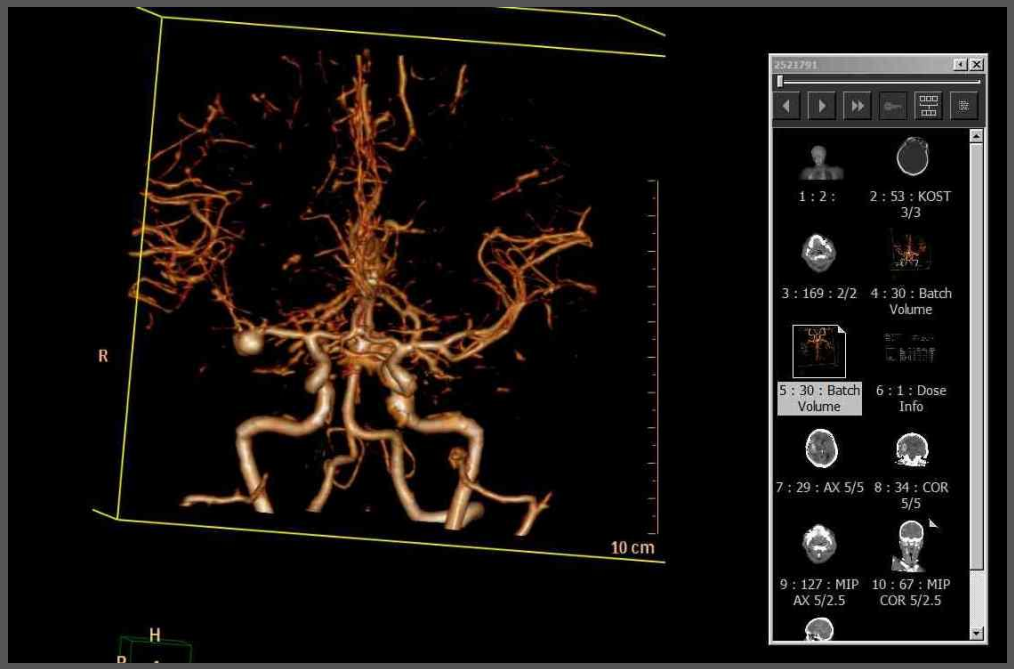
- 0,5% and less

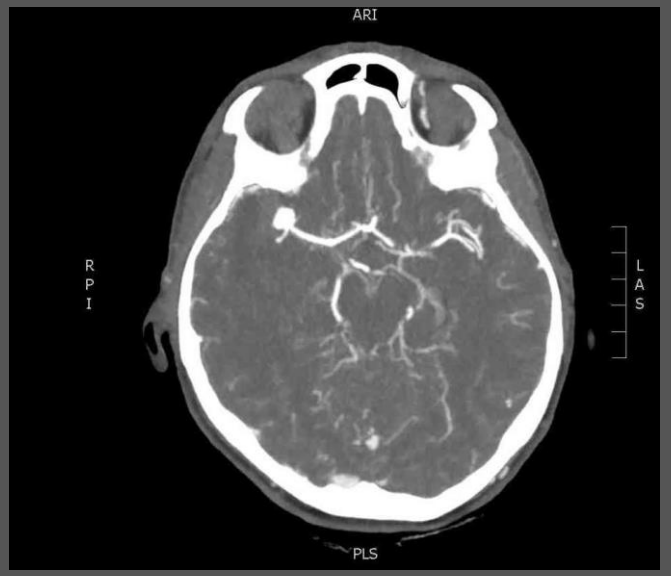
Localisation of ruptured aneurysms

AcoA	41%
ACI	31%
ACM	18%
Post.	10%

Severity scale Hunt and Hess

Grade	Signs and symptoms	Survival
1	Asymptomatic or minimal headache and slight neck stiffness	70%
2	Moderate to severe headache; neck stiffness; no neurologic deficit except cranial nerve palsy	60%
3	Drowsy; minimal neurologic deficit	50%
4	Stuporous; moderate to severe hemiparesis; possibly early decerebrate rigidity and vegetative disturbances	20%
5	Deep coma; decerebrate rigidity; moribund	10%


R
10 cm
H
1 : 2 :
2 : 53 : KOST 3/3
3 : 169 : 2/2
4 : 30 : Batch Volume
5 : 30 : Batch Volume
6 : 1 : Dose Info
7 : 29 : AX 5/5
8 : 34 : COR 5/5
9 : 127 : MIP AX 5/2.5
10 : 67 : MIP COR 5/2.5


ARI
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SRP
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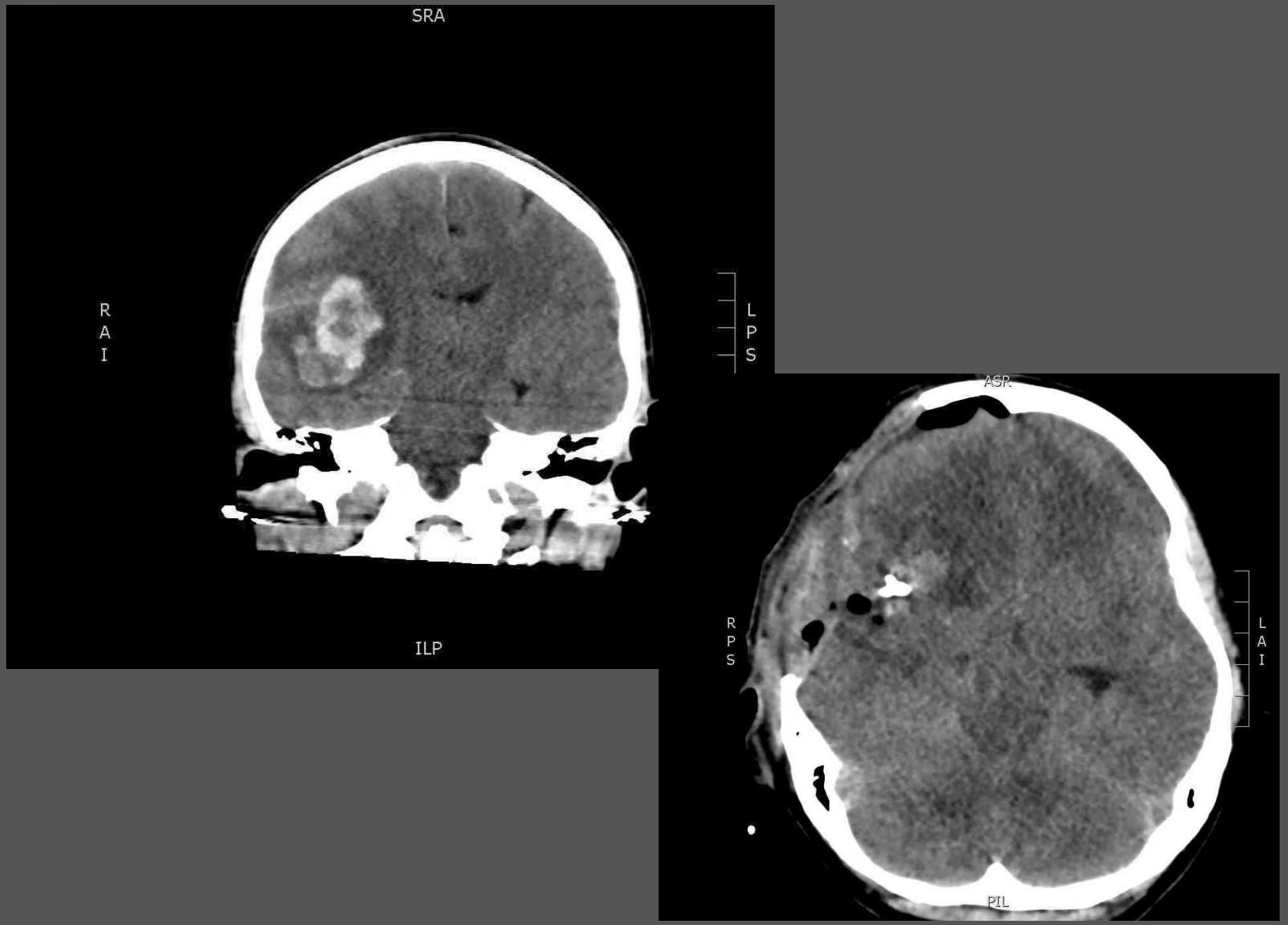
SRA
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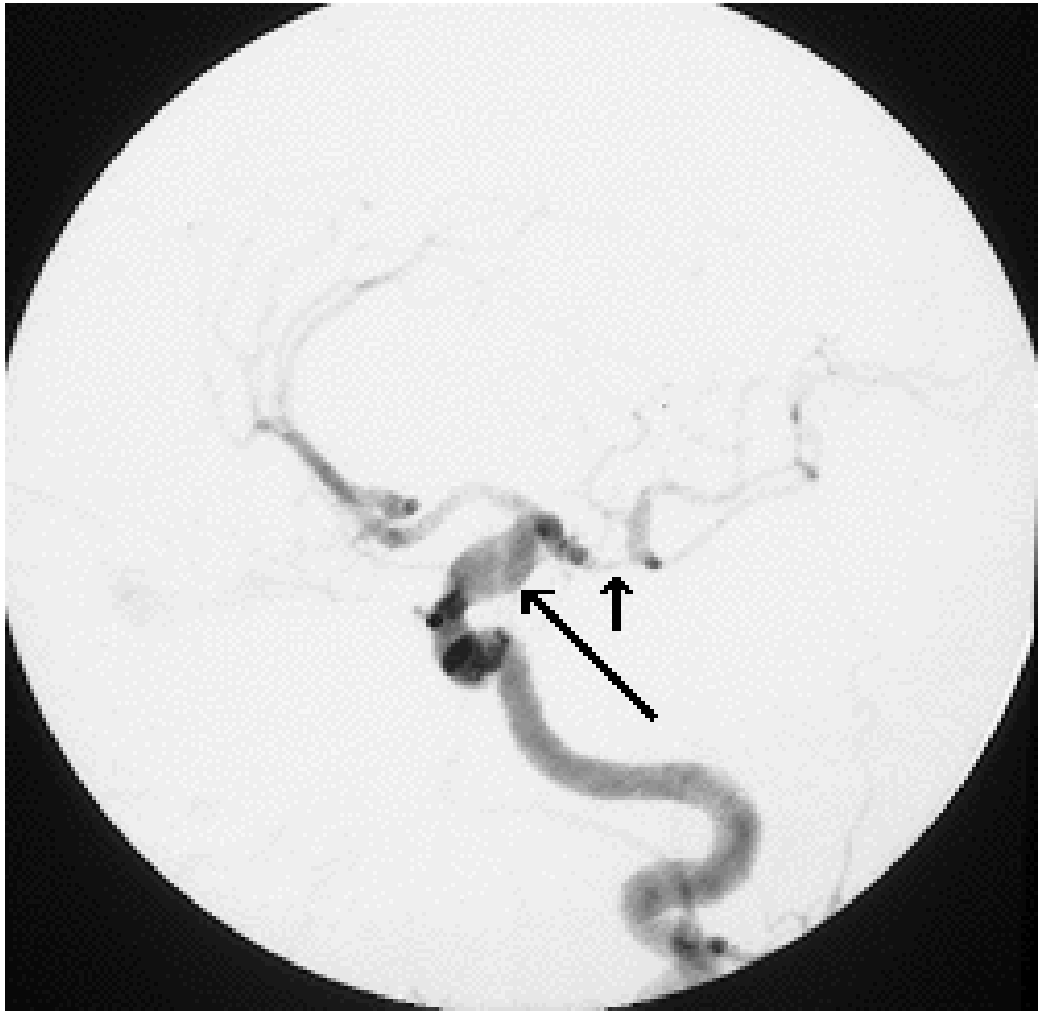
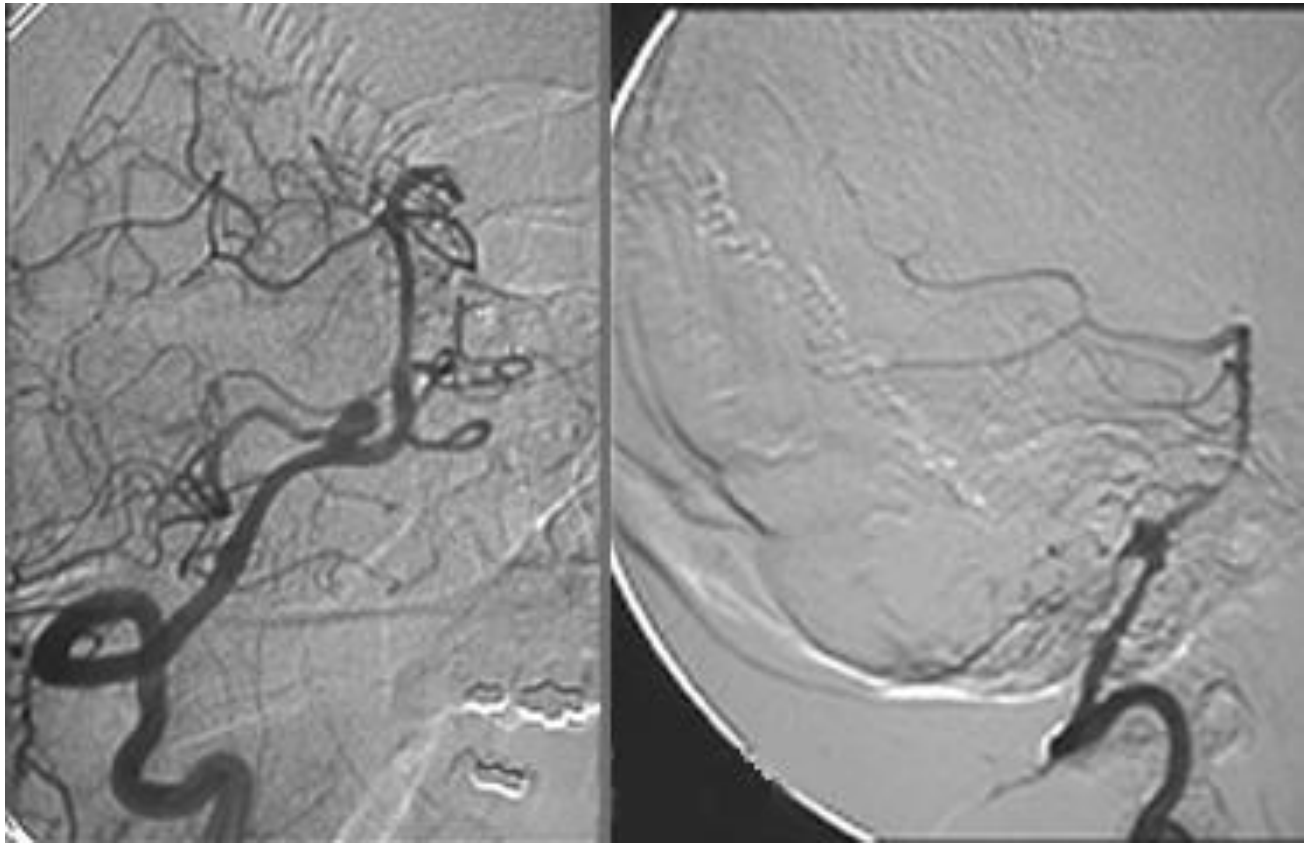
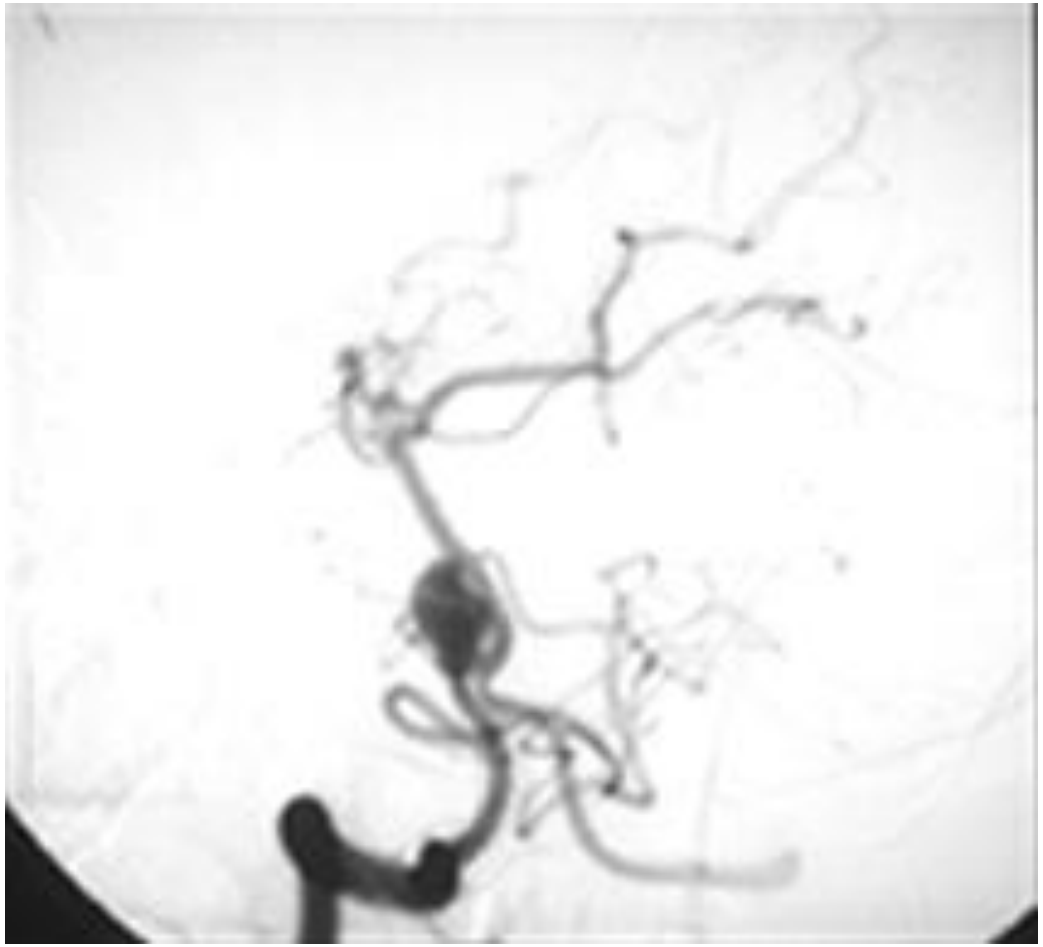
SAH: fusiform aneurysm



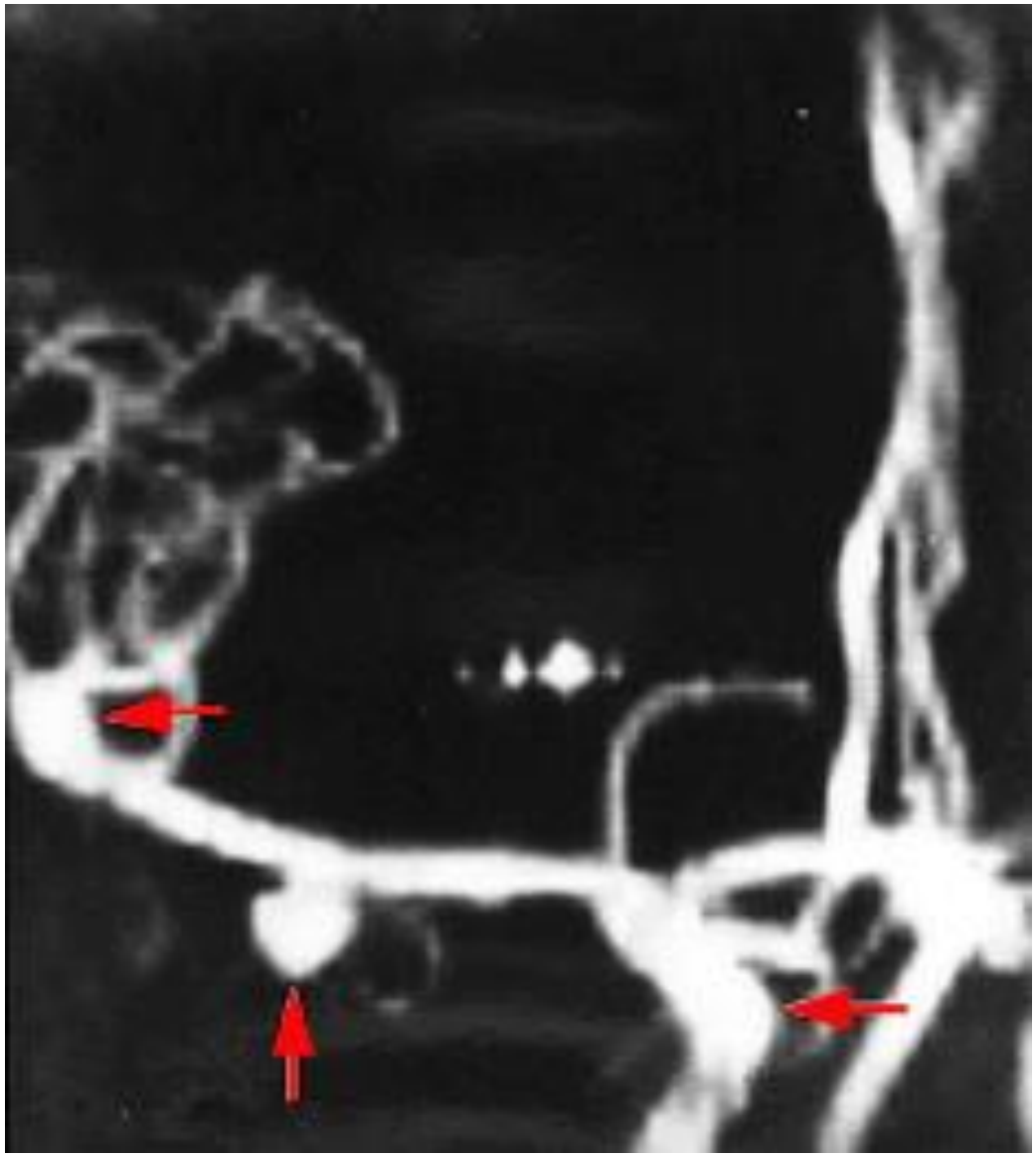
SAH: aneurysm PICA



SAH: aneurysm VB



SAH: 3 aneurysms (MRA)



Prognosis of aneurysmatic SAH

50% patients die

50% of the survivors have severe disability

8-17% die before admission to hospital

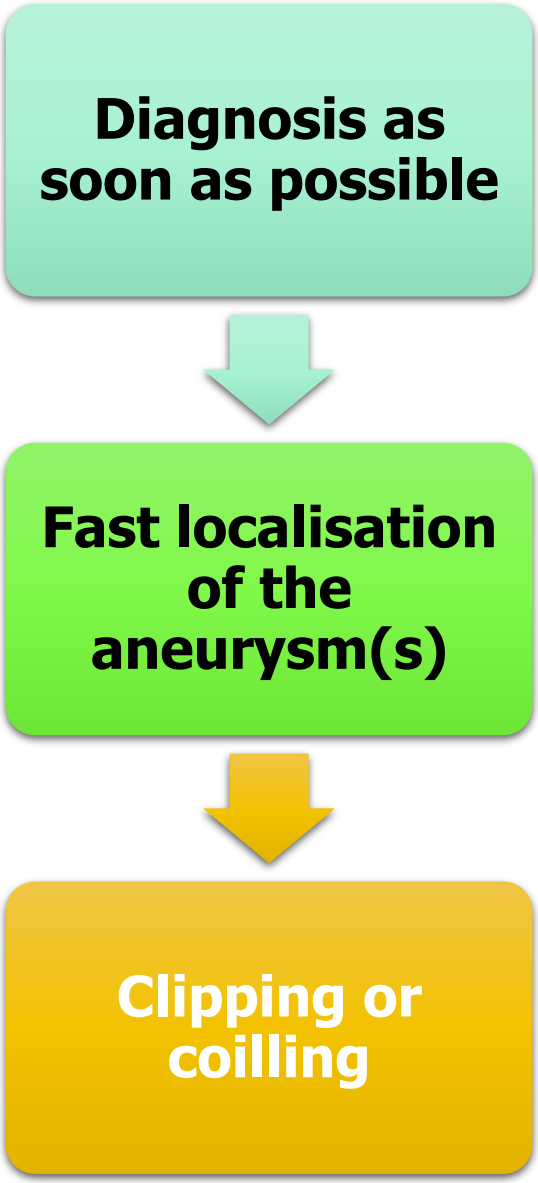
1. day	20%
1. m	40% from survivors
	50% cummulative risk
4 w- 6 m	gradual decrease from 1-2%/d to constant final 3%/y

Treatment

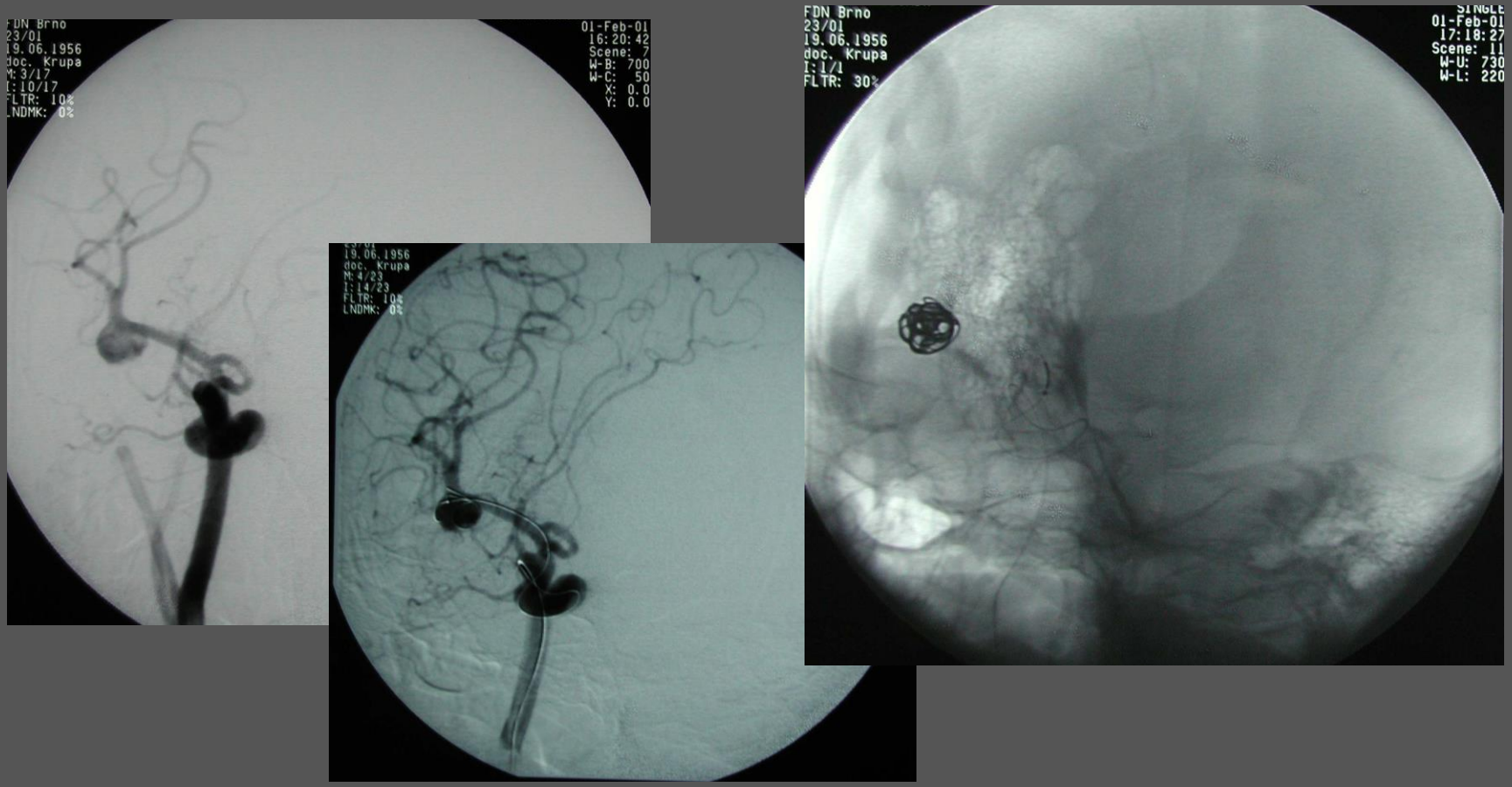
Timing of treatment

Risk of repeated bleeding

Treatment


Diagnosis as soon as possible
Fast localisation of the aneurysm(s)
Clipping or coilling

Coilling


FDN Brno
23/01
19.06.1956
doc. Krupa
M: 3/17
I: 10/17
FLTR: 10%
LNDMK: 0%
01-Feb-01
16:20:42
Scene: 7
W-B: 700
W-C: 50
X: 0.0
Y: 0.0
19.06.1956
doc. Krupa
M: 4/23
I: 14/23
FLTR: 10%
LNDMK: 0%
FDN Brno
23/01
19.06.1956
doc. Krupa
I: 1/1
FLTR: 30%
01-Feb-01
17:18:27
Scene: 11
W-U: 730
W-L: 220

Main complications

Acute hydrocephalus

- **Transient ventricular catheter**
- **Permanent VP shunt**

Cerebral ischaemia

- **Vasospasms**
  - **Nimodipin (prevention)**
  - **3H (therapy)**

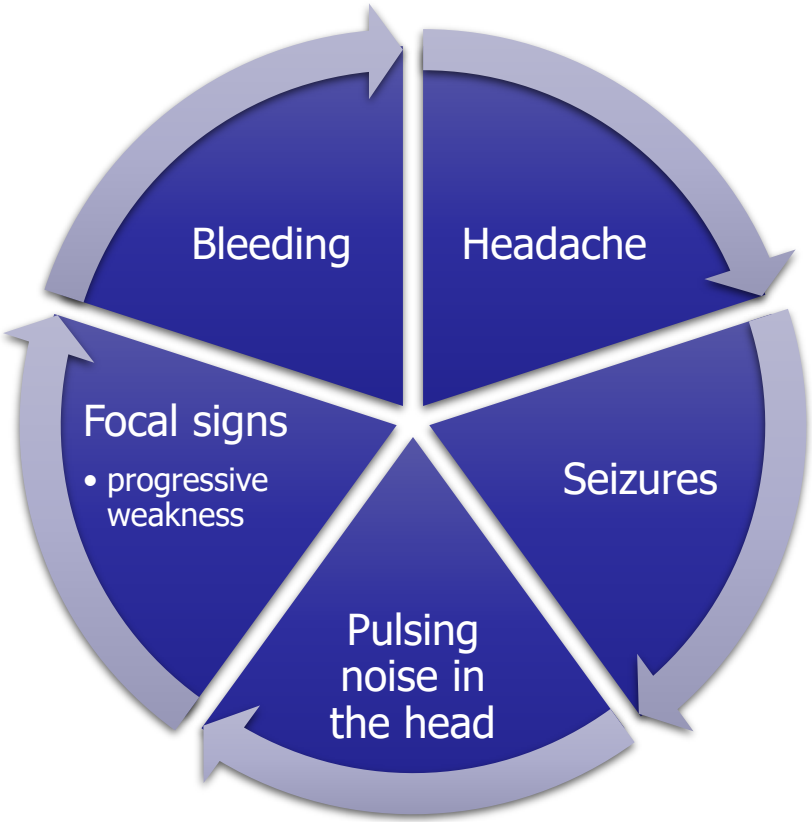
Abnormal connection between arteries and veins bypassing the capillary system

- **AVM is usually congenital**
  - Discovered by autopsy
  - During treatment of an unrelated disorder

How many of people affected with brain AVM are asymptomatic ???

- The annual detection of the symptomatic AVMs is approximately 1 per 100,000 / year (Netherlands, Minnesota)
- The prevalence in adults was approximately 1,4% (autoptic study)

AV malformation

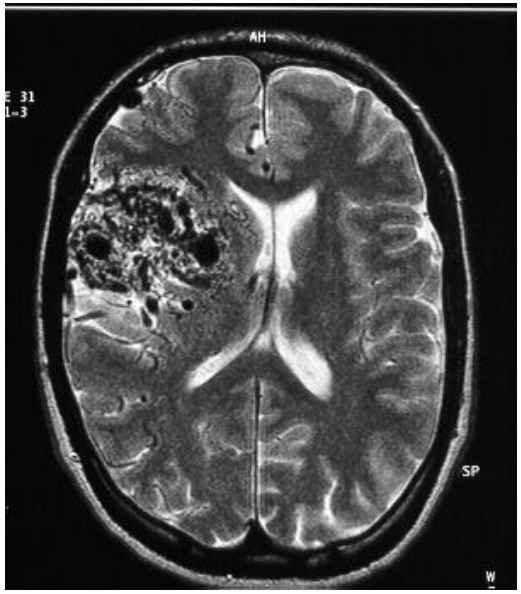


Signs and symptoms

AVM: clinical manifestation

- Bleeding (< 3 cm) **42%**
- **Annual risk of bleeding 2- 4%**
  - **Bleeding**
    - **18% mortality**
      - **60% of survivors have no or minimal deficit**

- Epileptic seazures (>3 cm) **25%**
- Focal deficit (ischaemia) **10%**



Diagnostic work-up

- CT
- MR
- Angiography
- X rays (calcifications)

Treatment

- Treatment depends on the location and size of the AVM and whether there is bleeding or not.
- **Sudden bleeding**
  - Restoration of vital functions
  - Anticonvulsant medications to control seizure
  - Medications or procedures to relieve intracranial pressure
- **Preventive treatment of as yet unruptured brain AVM**
  - Controversial results
    - *Several studies suggested favorable long-term outcome for unruptured AVM patients not undergoing intervention.*

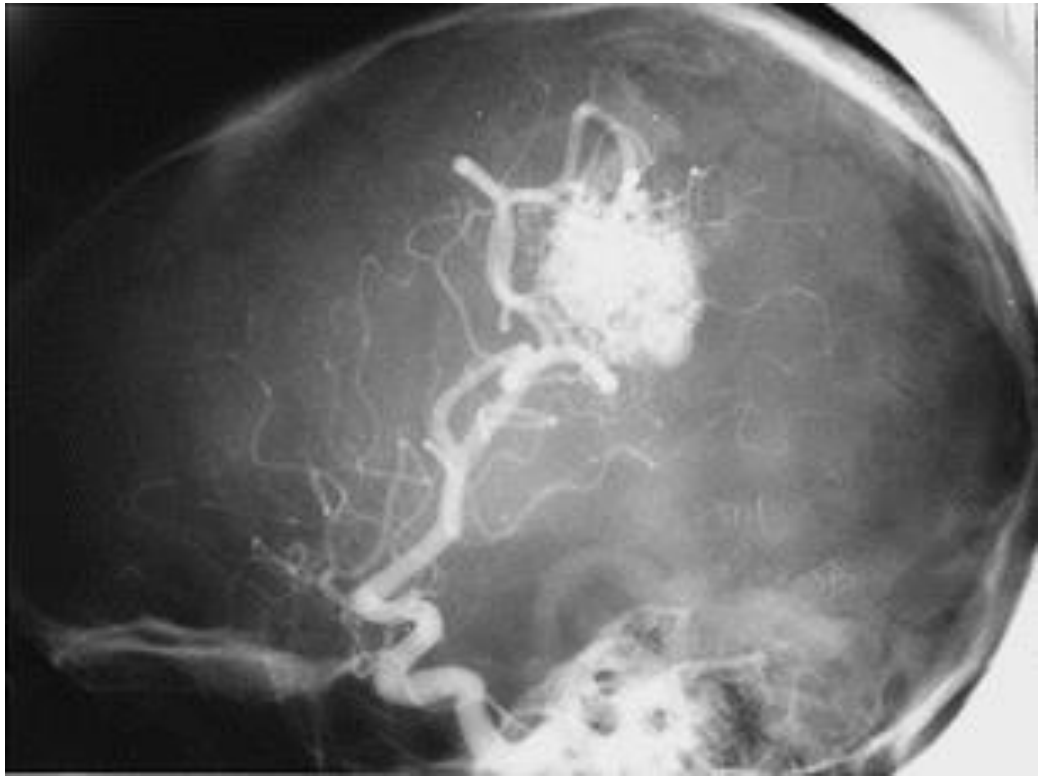
Spetzler-Martin grade

AVM size	Adjacent eloquent cortex	Draining veins
Under 3 cm = 1	Non-eloquent = 0	Superficial only = 0
3–6 cm = 2	Eloquent = 1	Deep veins = 1
Over 6 cm = 3		

Eloquent cortex = removed will result in loss of sensory processing or linguistic ability, minor paralysis, or paralysis.

The risk of post-surgical neurological deficit (difficulty with language, motor weakness, vision loss) increases with increasing Spetzler-Martin grade

AV malformation: DSA



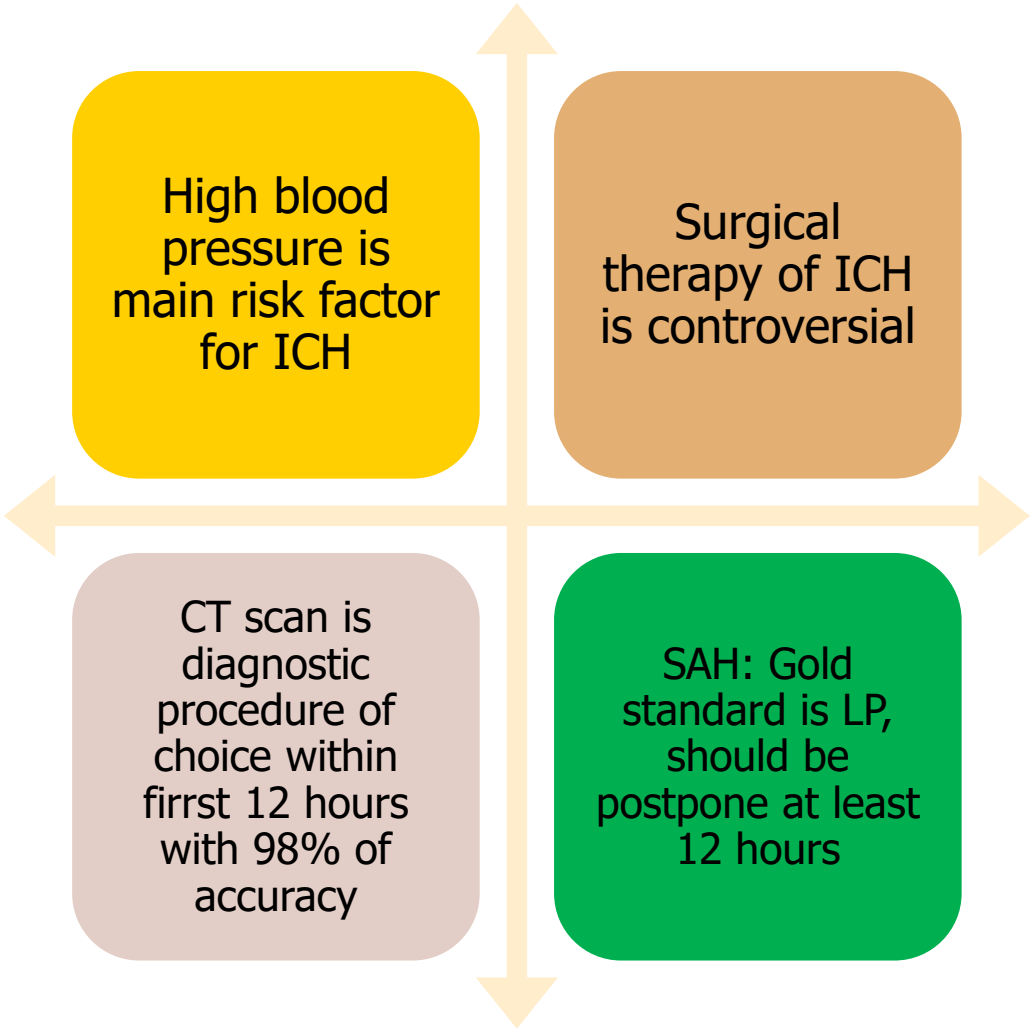
AV malformation: after operation

AVM: treatment possibilities

- Resection
  - Craniotomy
- Embolisation
  - Radiologically guided catheter
- Radiation surgery
  - Gamma knife

The goal: Total occlusion of AVM

Take home message



Take home message 2

Sudden headache, SAH should be considered and excluded

Non-ruptured aneurysm- risk of bleeding depends on size

Silent AVM- therapy is controversial