



UNIVERSITATEA DE STAT DE MEDICINĂ ȘI FARMACIE
"NICOLAE TESTEMIȚANU" DIN REPUBLICA MOLDOVA

ISCHEMIC STROKE

Mihail Gavriiuc



Ischemic stroke

INTRODUCTION



STROKE

Since ancient times **Cerebral Vascular Accident**

is taught of "mystery and inevitability", and he also has a special term - "**apoplexy**" (ἀποπληξία – shock, stroke).





STROKE

The prototype: a short-necked man with a red face without any predictors falls to the ground, lose consciousness and dies in a few hours.



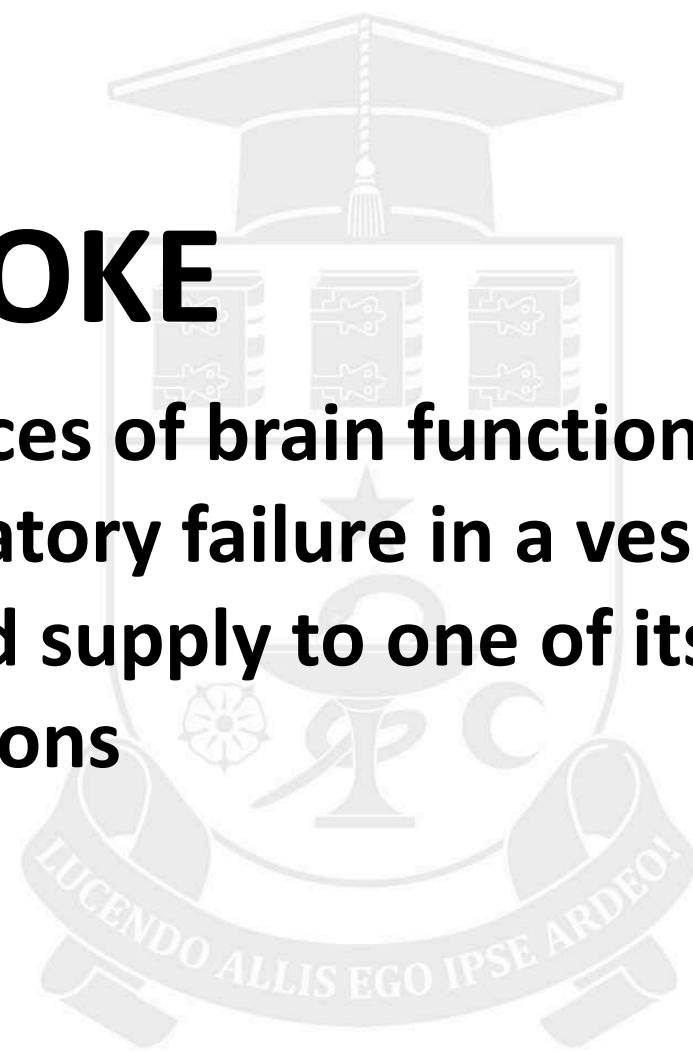
BACH!



STROKE

STROKE

is an acute disturbances of brain functions caused by blood circulatory failure in a vessel responsible for blood supply to one of its regions



STROKE is one medical emergency!

Myocardial infarction

Visceral colic

Acute abdomen

Asthmatic status

Acute intoxication

etc.

Emergency Care



Trauma



ALGORITHM

of diagnosis and treatment of the patient with ischemic stroke

Recognition + Call emergency service



Prehospital Support + Transportation + Phone Advert



STROKE Unite



Imagistical Examination(CT, MRI, Angio-CT/MRI)



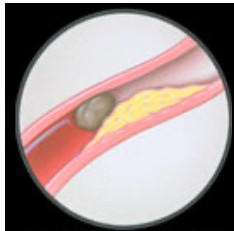
TREATAMENT:
general + recanalization + rehabilitation + secondary profilaxy



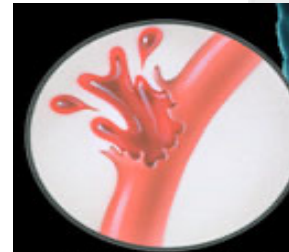
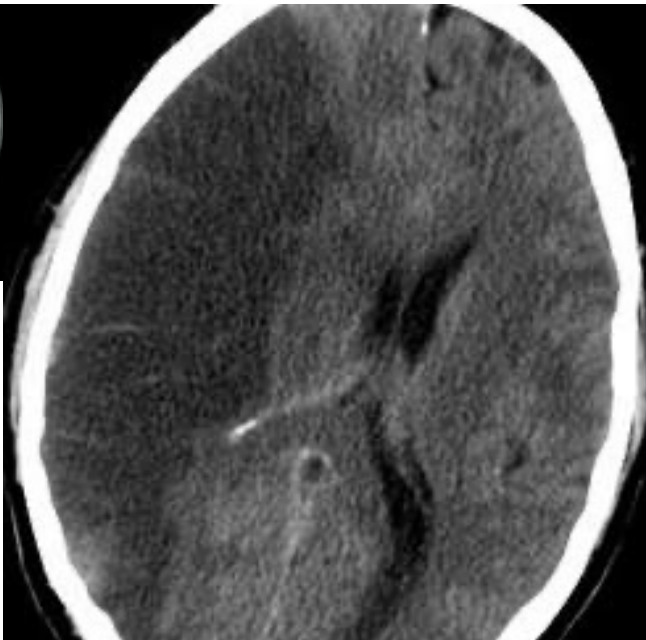
STROKE

ISCHEMIC (80-85%)

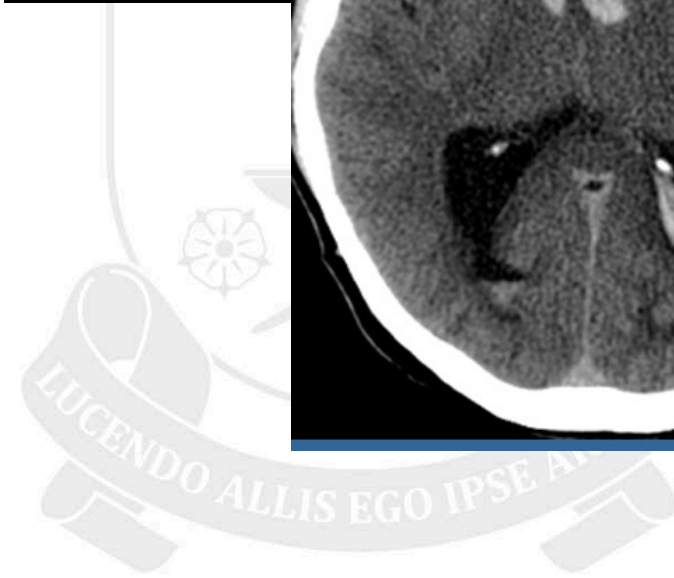
HEMORRHAGIC (15-20%)



Ischemic stroke



Hemorrhagic stroke



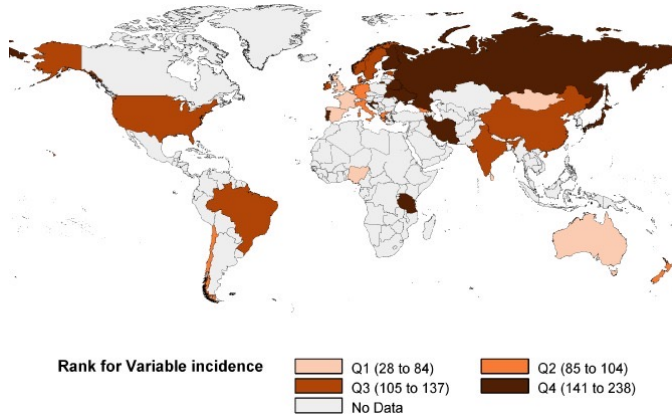


STROKE

1. EPIDEMIOLOGY



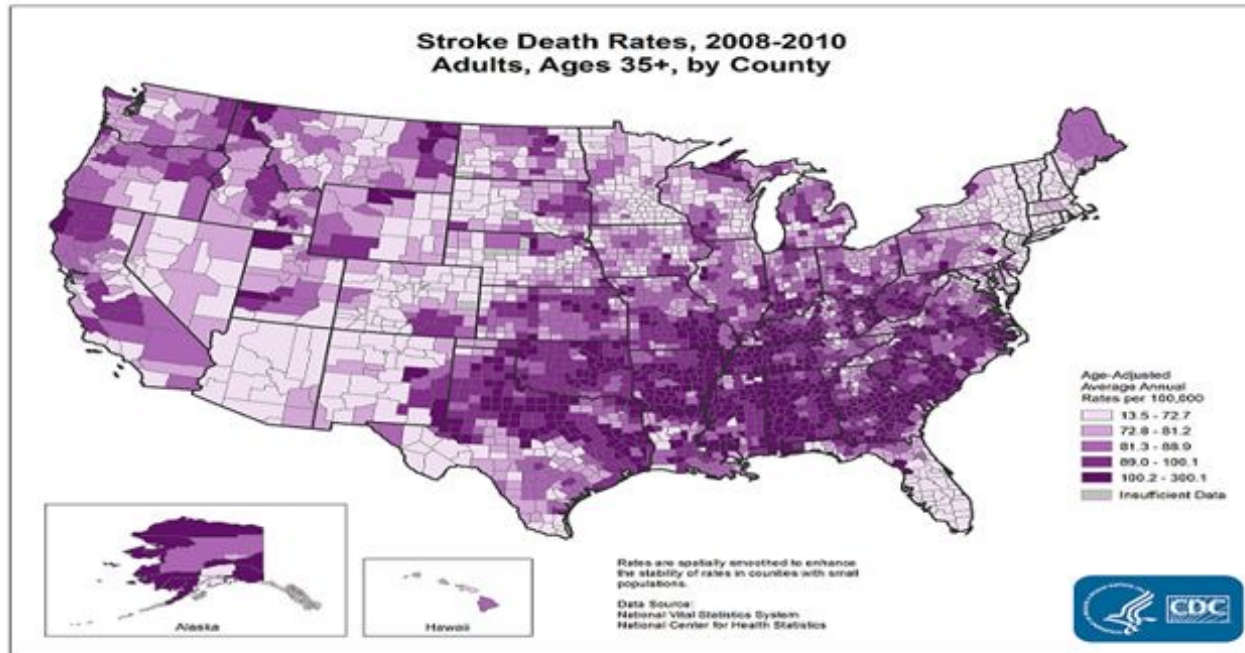
STROKE incidence



- ✓ the third cause of death
- ✓ worldwide six people die from a stroke every 60 seconds
- ✓ every two seconds, someone, somewhere in the world is having a stroke
- ✓ the second cause of long-term disability



STROKE facts



Centers for Disease and Control and Prevention. Stroke Facts. accessed on Feb. 25, 2015 from: <http://www.cdc.gov/stroke/facts.htm>

- Stroke is the fifth leading cause of death in the **United States**, killing more than 130,000 Americans each year —that's 1 of every 20 deaths.



STROKE incidence

- in Germany the incidence is 200-250 cases per 100,000 population
- incidence in Eastern European countries reaches 400 cases per 100,000 population
- 80% of all people who have suffered from a stroke now live in low and mid-income countries





STROKE incidence

IJMR Indian Journal of Medical Research

Home
Current issue
Instructions
Submit article

[Indian J Med Res. 2017 Aug; 146\(2\): 175–185.](#)

doi: [10.4103/ijmr.IJMR_516_15](#)

PMCID: PMC5761027

PMID: [29265018](#)

Incidence & prevalence of stroke in India: A systematic review

[Sureshkumar Kamalakannan](#),¹ [Aashrai S. V. Gudlavalleti](#),² [Venkata S. Murthy Gudlavalleti](#),¹ [Shifalika Goenka](#),³
and [Hannah Kuper](#)¹

- **INDIA.** The cumulative incidence of stroke ranged from 105 to 152/100,000 persons per year, and the crude prevalence of stroke ranged from 44.29 to 559/100,000 persons in different parts of the country during the past decade. These values were higher than those of high-income countries.



STROKE facts



- ❖ Stroke is the third most common cause of death in **Israel**.
- ❖ The annual incidence of hospitalized acute cerebrovascular events is ~ 13,000 and rates, particularly in women, seem even higher than those observed for acute myocardial infarction. (*Tanne D, Goldbourt, U. Harefuah. 2008 Nov;147(11):869-70, 942, 941*)



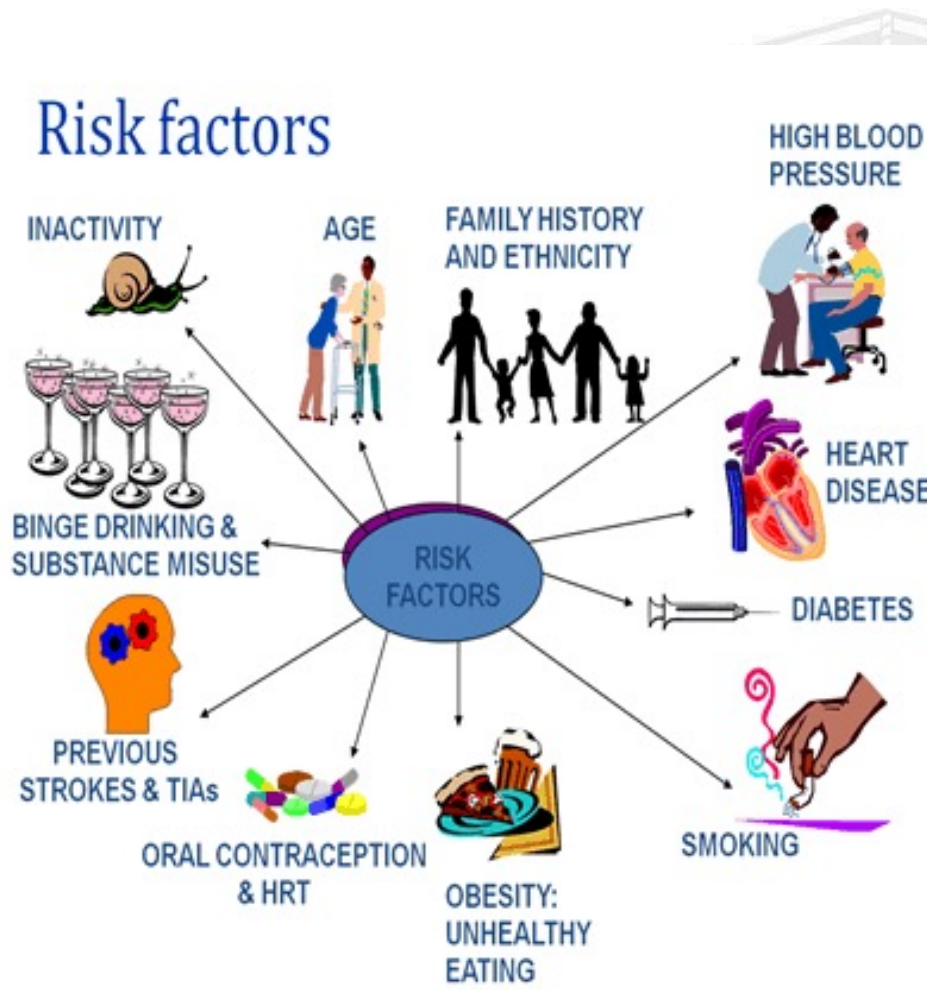
STROKE

2. RISK FACTORS



STROKE

Unmodifiable :



Modifiable :



Risk factors

Unmodifiable:

- **Age**
- **Previous Stroke or Transient Ischemic Attack**
- **Gender**
- **Heredity**
- **Ethnicity**

Modifiable:

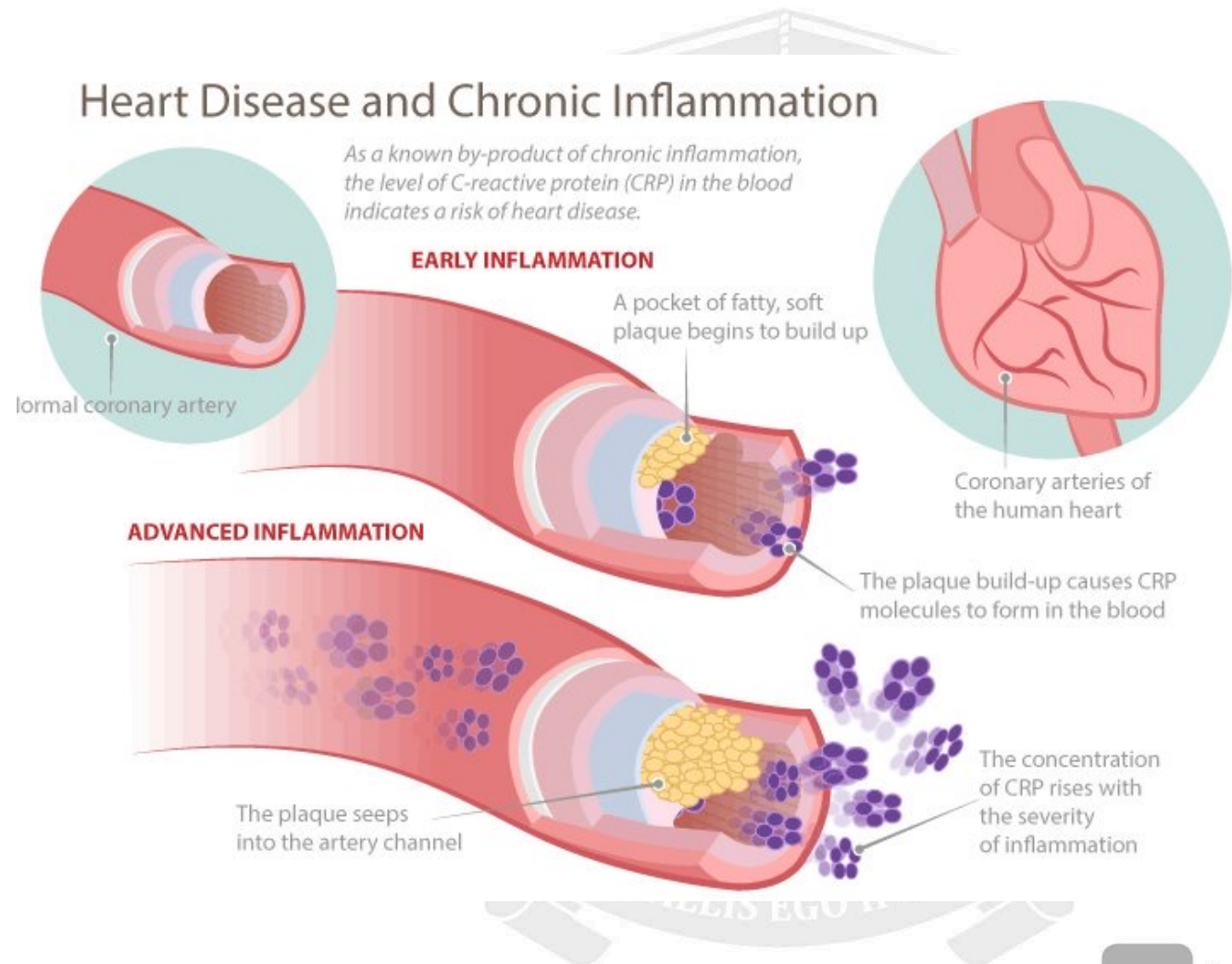
- **Hypertension**
- **Cardiac disease**
- **Diabetes**
- **Hypercholesterolemia**
- **Cigarette Smoking**
- **Alcohol consumption**
- **Illicit Drug Use**
- **Lifestyle Factors**



Risk Factors

high sensitivity C-reactive protein

- ✓ Chronic inflammation of the arterial wall contributes to the formation of atherosclerosis plaques.
- ✓ Hs-CRP is considered a cardiovascular risk marker.





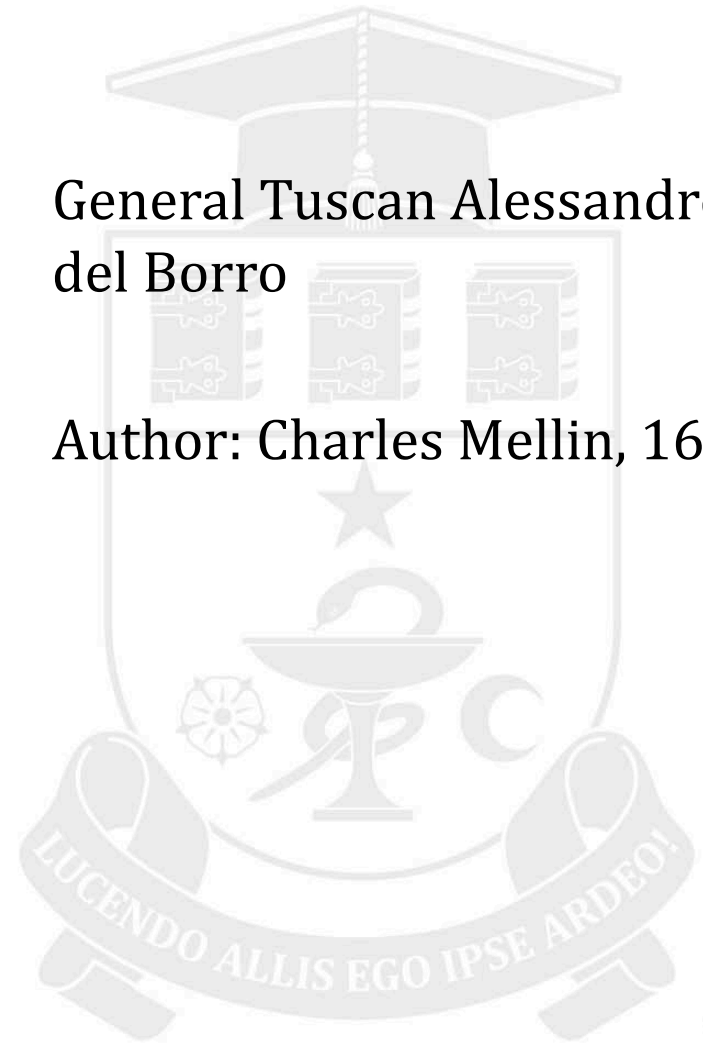
Stroke risk ractors

obesity



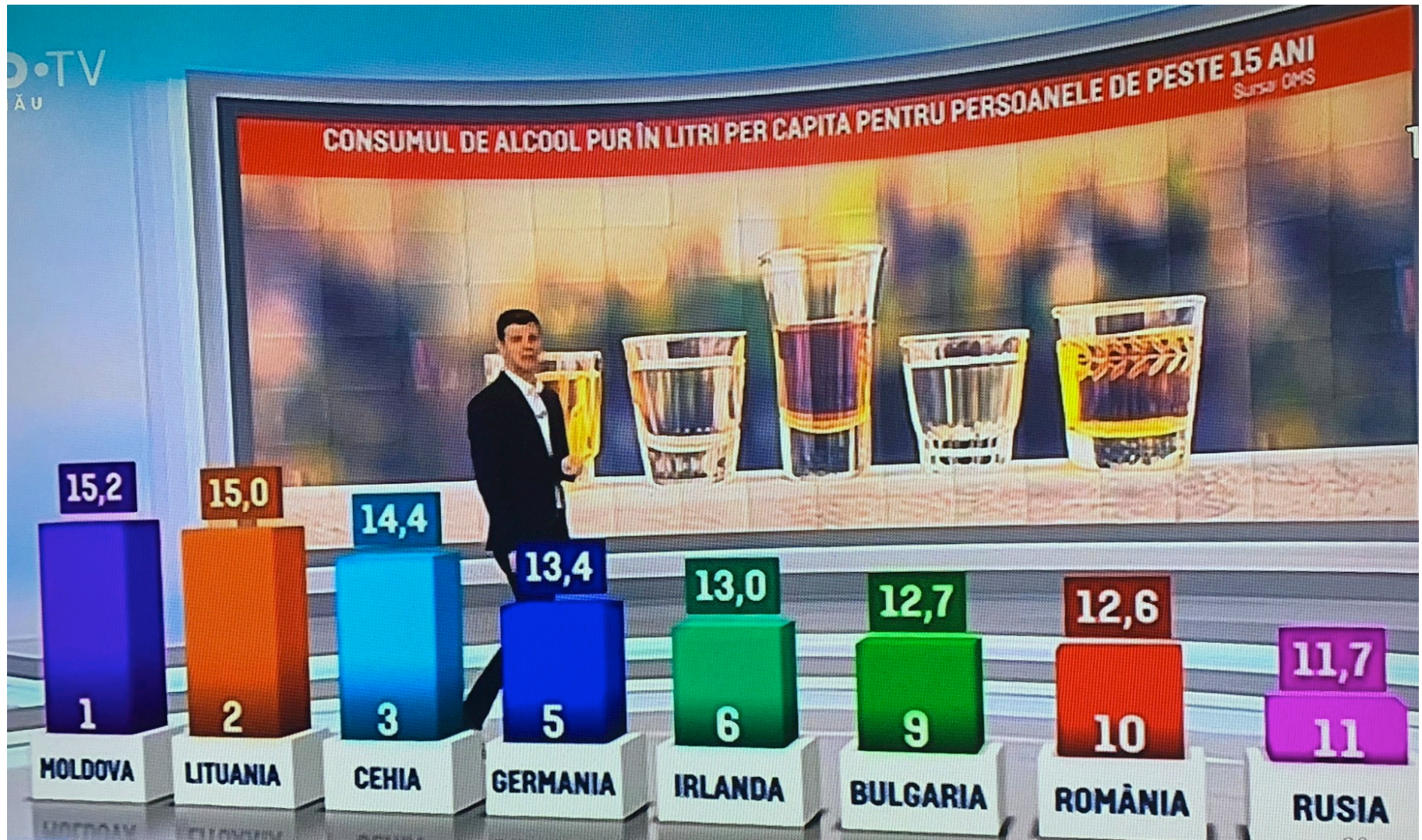
General Tuscan Alessandro
del Borro

Author: Charles Mellin, 1645





WHO: alcohol consumption 2018



Alcohol use and burden for 195 countries and territories, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016



GBD 2016 Alcohol Collaborators*



Methods Using 694 data sources of individual and population-level alcohol consumption, along with 592 prospective and retrospective studies on the risk of alcohol use, we produced estimates of the prevalence of current drinking, abstinence, the distribution of alcohol consumption among current drinkers in standard drinks daily (defined as 10 g of pure ethyl alcohol), and alcohol-attributable deaths and DALYs. We made several methodological improvements compared with previous estimates: first, we adjusted alcohol sales estimates to take into account tourist and unrecorded consumption; second, we did a new meta-analysis of relative risks for 23 health outcomes associated with alcohol use; and third, we developed a new method to quantify the level of alcohol consumption that minimises the overall risk to individual health.

Interpretation Alcohol use is a leading risk factor for global disease burden and causes substantial health loss. We found that the risk of all-cause mortality, and of cancers specifically, rises with increasing levels of consumption, and the level of consumption that minimises health loss is zero. These results suggest that alcohol control policies might need to be revised worldwide, refocusing on efforts to lower overall population-level consumption.

Funding Bill & Melinda Gates Foundation.

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Lancet 2018; 392: 1015–35

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[S0140-6736\(18\)31310-2](http://dx.doi.org/10.1016/S0140-6736(18)31310-2)



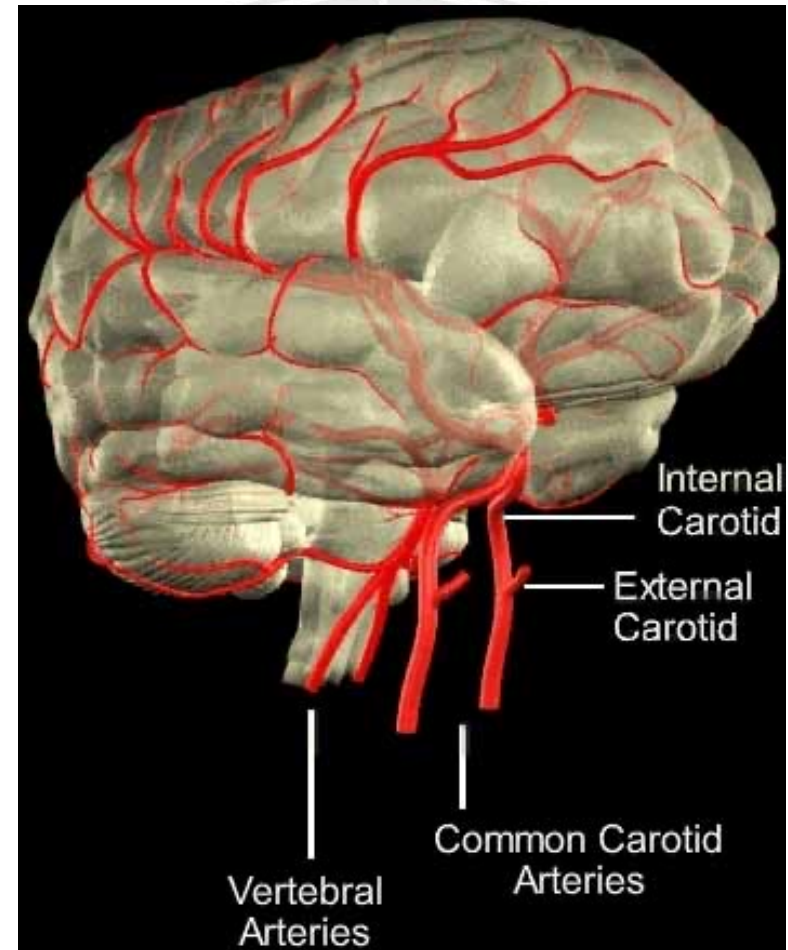
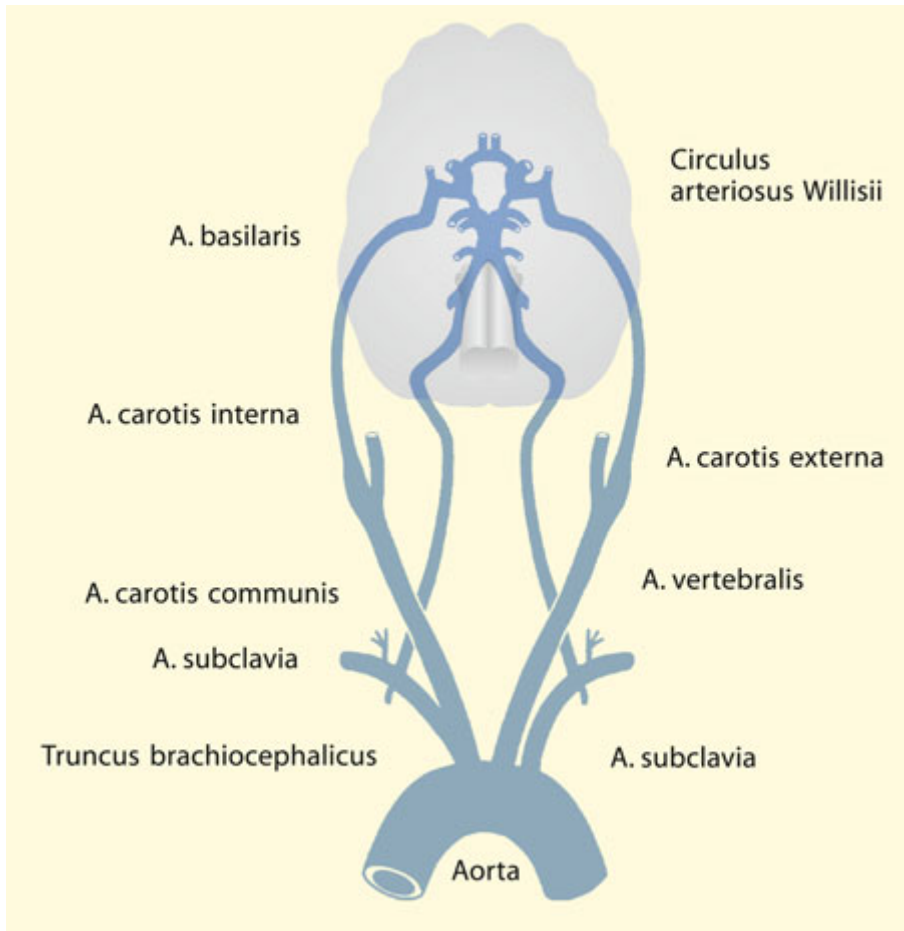
STROKE

3.

ANATOMICAL, PHYSIOLOGICAL AND PHYSIOPATHOLOGICAL PARTICULARITIES OF CEREBRAL BLOOD FLOW



Particularity of anatomy: vascularization

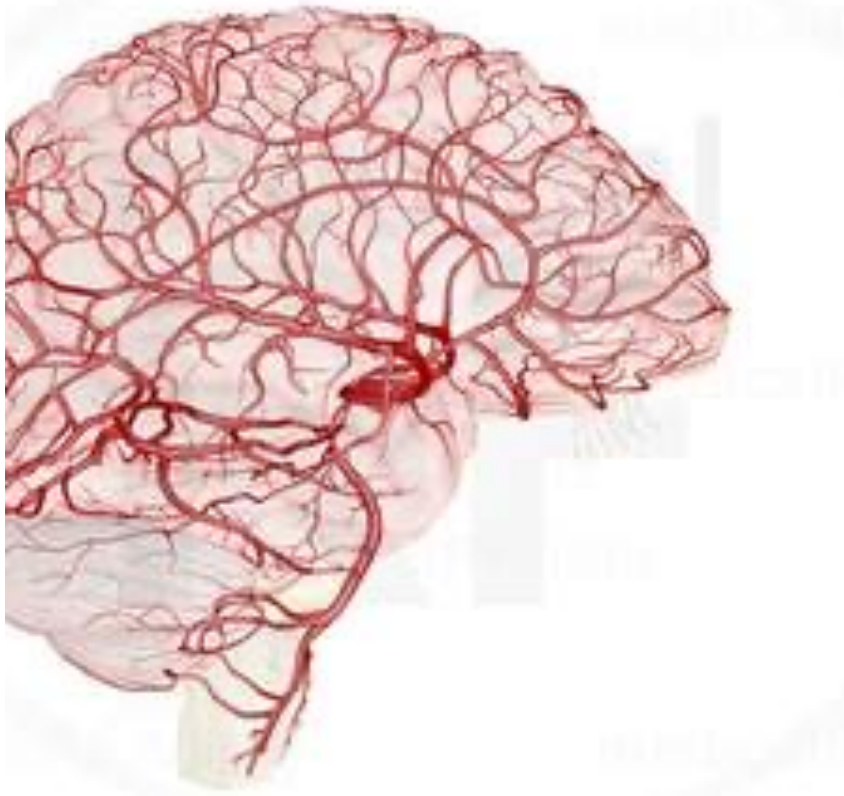


(Dorndorf 1983)

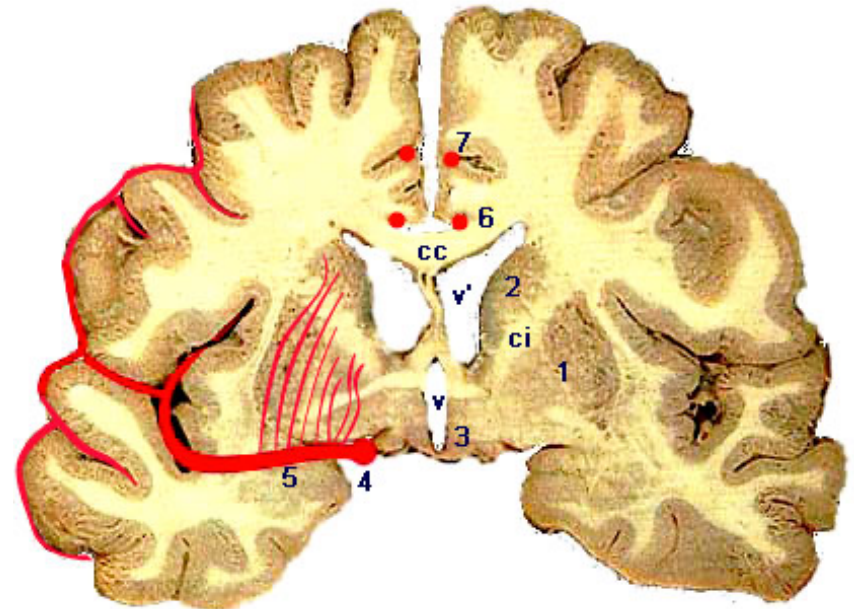


Functional Classification of Cerebral Arteries

Surface arteries (convex)



Perforating arteries (penetrating)





Middle cerebral artery



1. The internal carotid



Posterior communicating artery

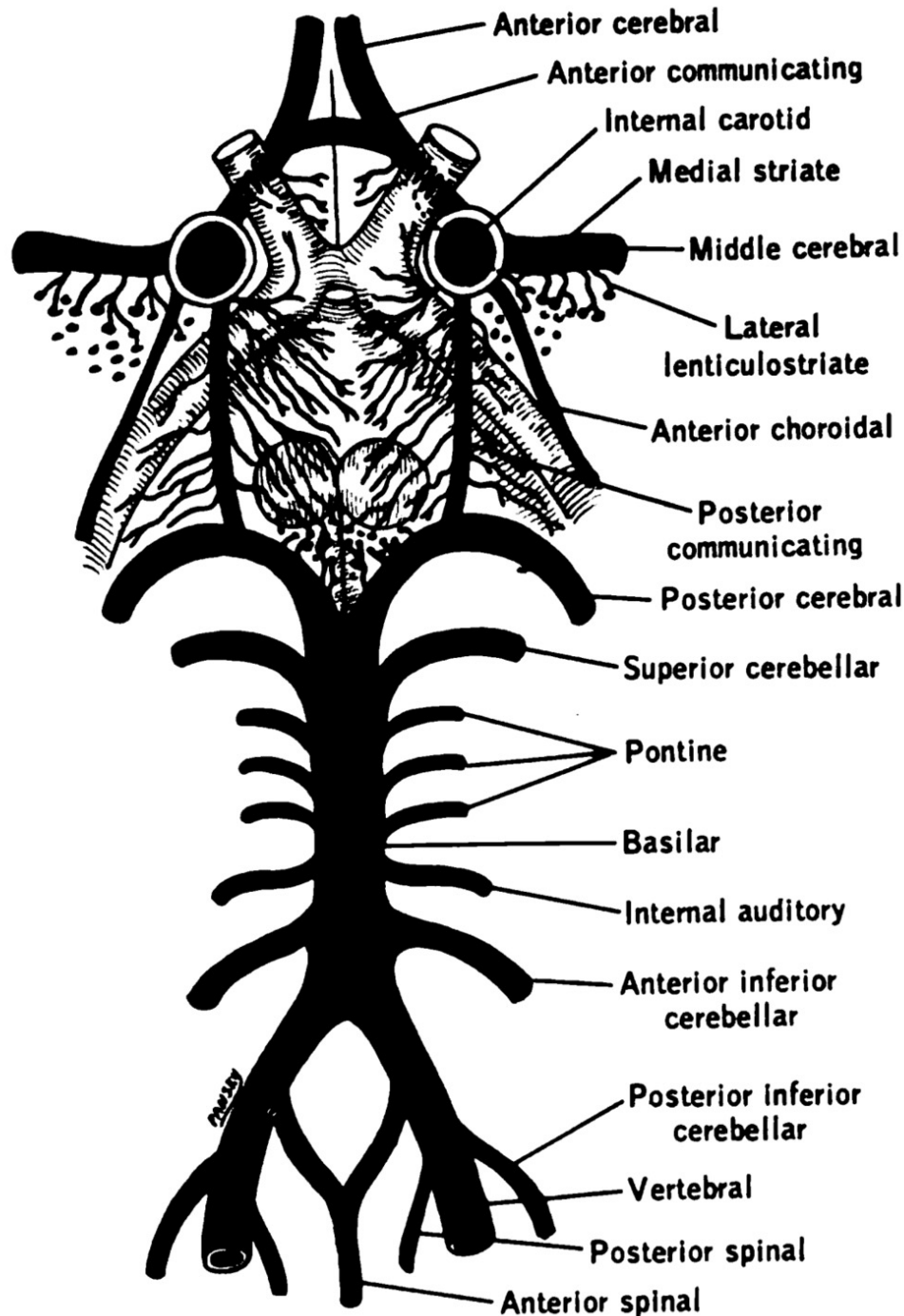
The external carotid artery

2. The anterior communicating artery WILISII'S POLYGON

4. The anterior communicating artery (carotid system)

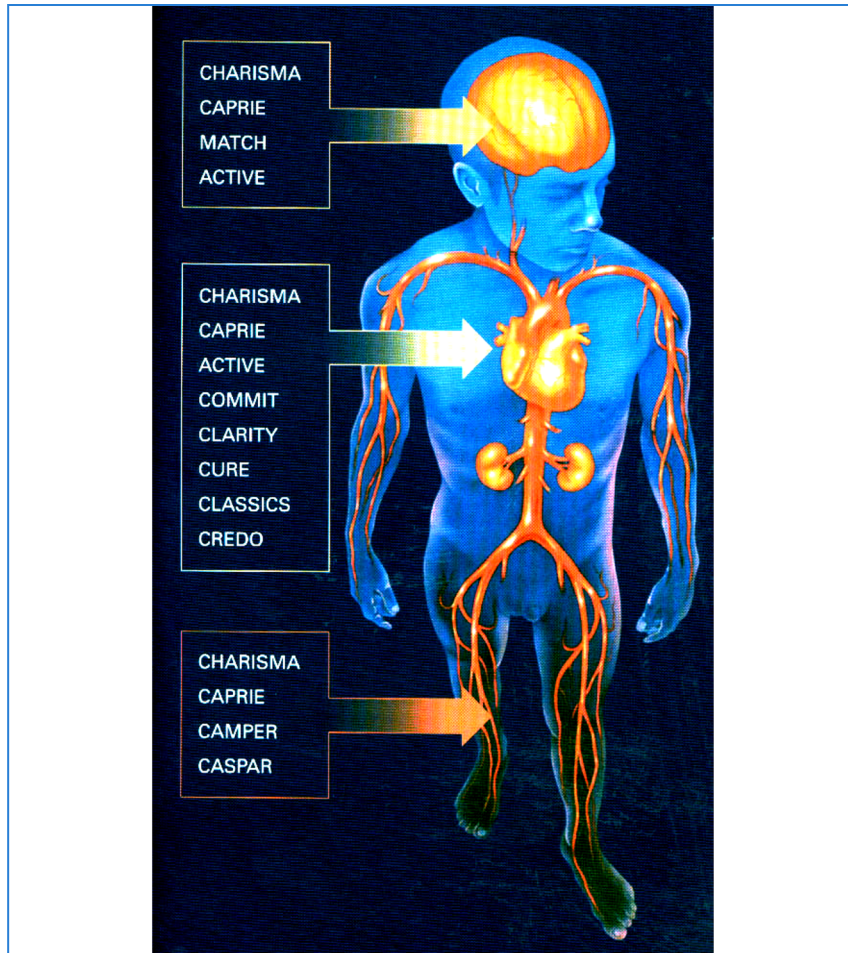
5. The posterior communicating artery WILISII'S POLYGON

6. Leptomeningeal





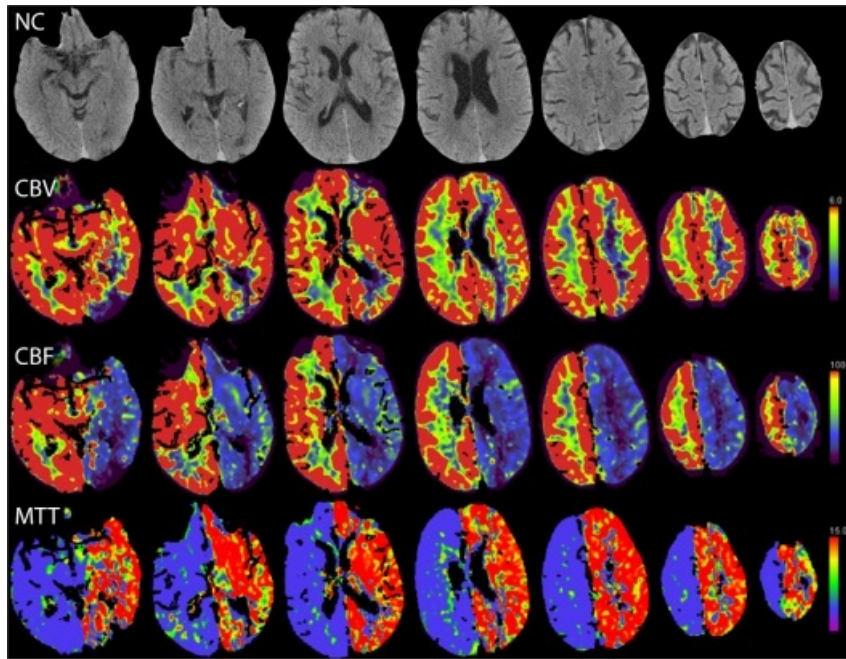
physiology: weight-to-volume correlation



Brain accounts only 2% of the body's mass but receives almost 15% of the blood volume per minute (~ 1.2 l).



perfusion



NC – noncontrast
CBV – cerebral blood volume
CBF – cerebral blood flow
MTT – mean transit time

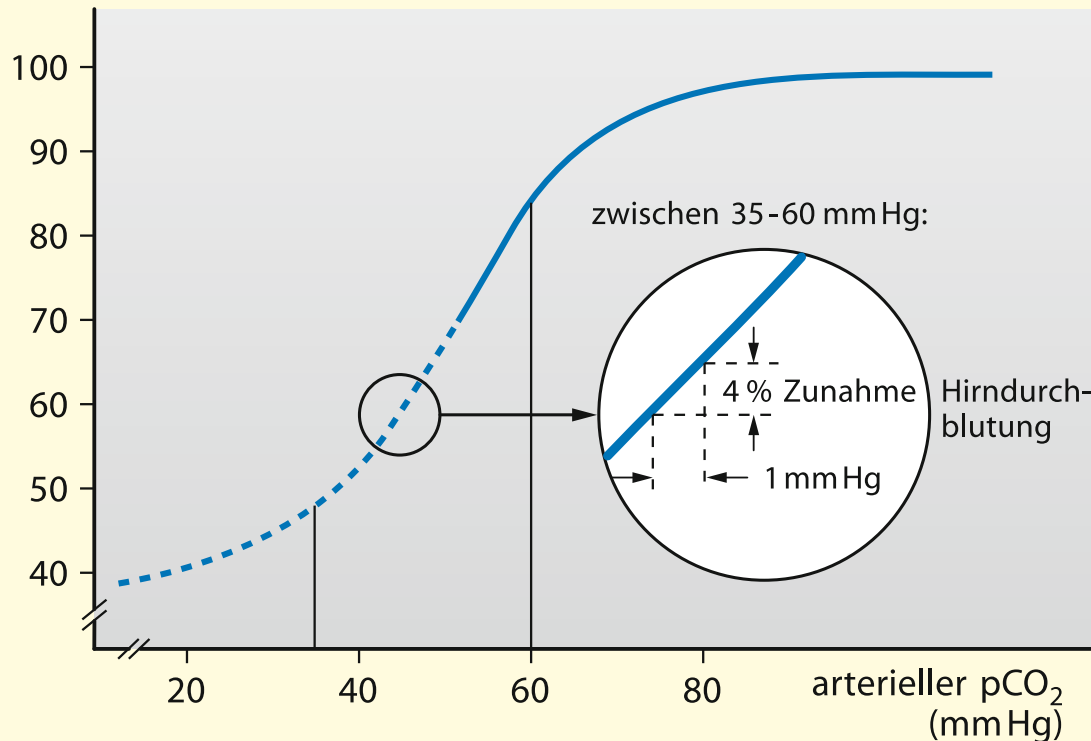
**N: 60-80 ml of blood for
100 g of cerebral tissue
in one minute**

**Dysfunction of nervous
tissue will occur when
blood flow decreases by
1/3 - 1/4 of its normal (~
20ml / 100g / min)**



self-regulation of intracerebral blood flow

Hirndurchblutung
(ml/min/100 g)



- **Bayliss effect**
- **Partial carbon dioxide pressure (pCO₂)**

Cerebral blood flow reactivity at partial pressure of carbon dioxide (pCO₂)

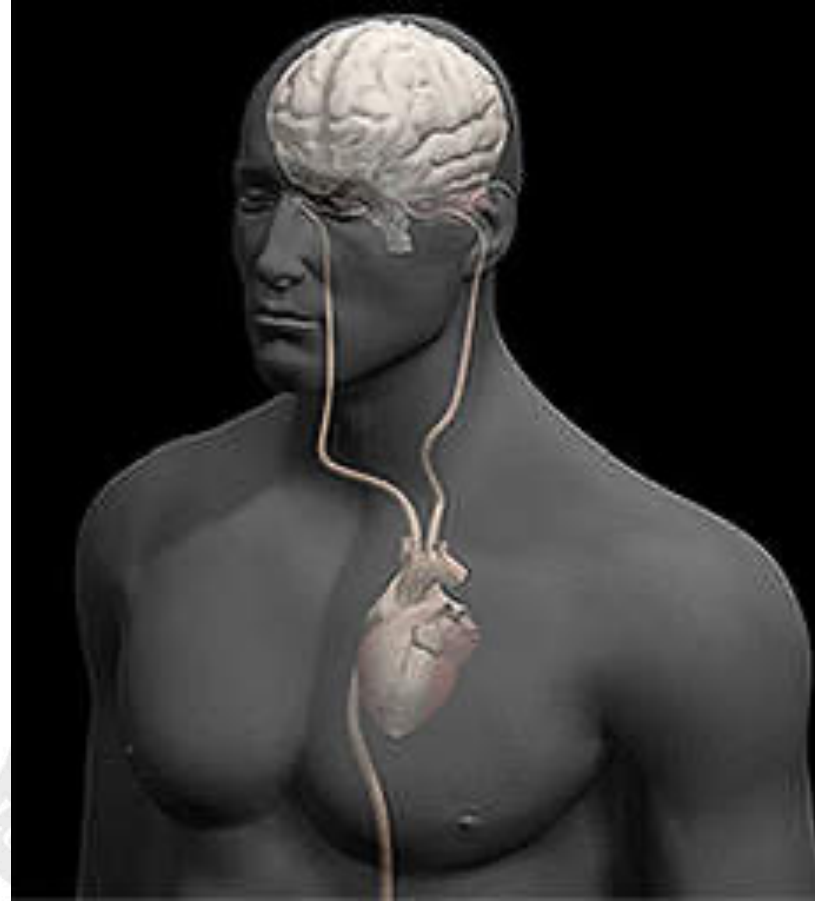
(Hacke 1991)



cerebral perfusion pressure

is determined by:

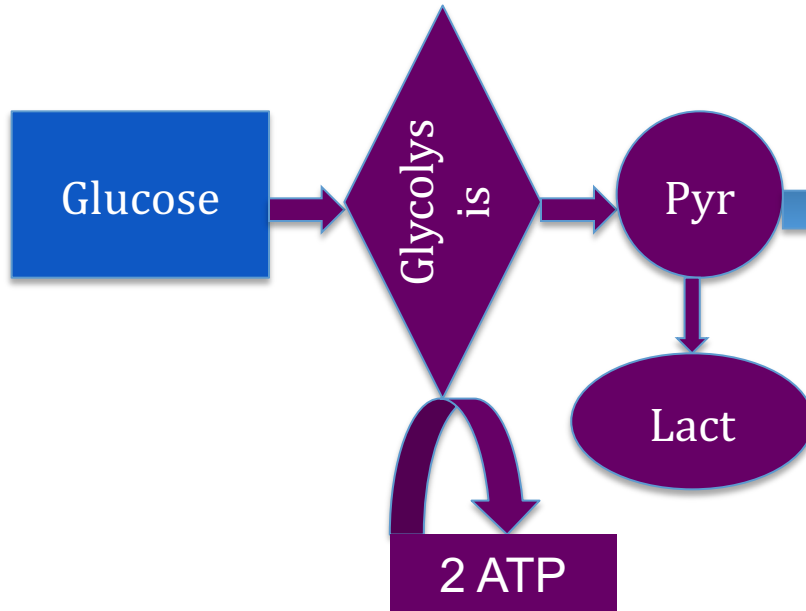
- 1) cardiac activity,**
- 2) the resistance of peripheral vessels,**
- 3) intracranial pressure**



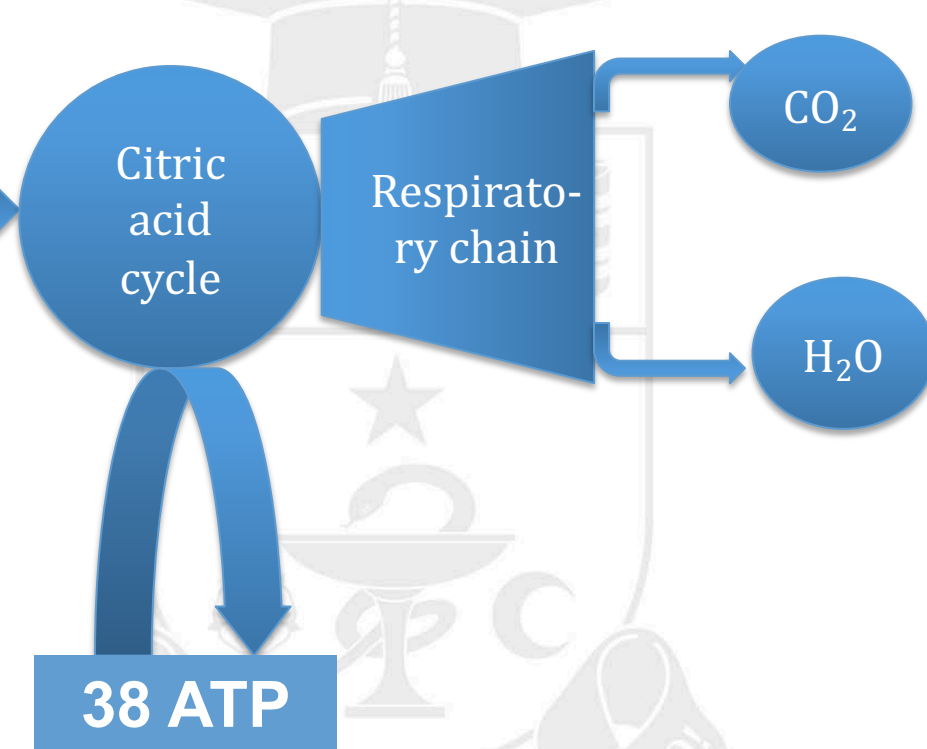


energy insurance

ANAEROB METABOLISM



AEROB METABOLISM



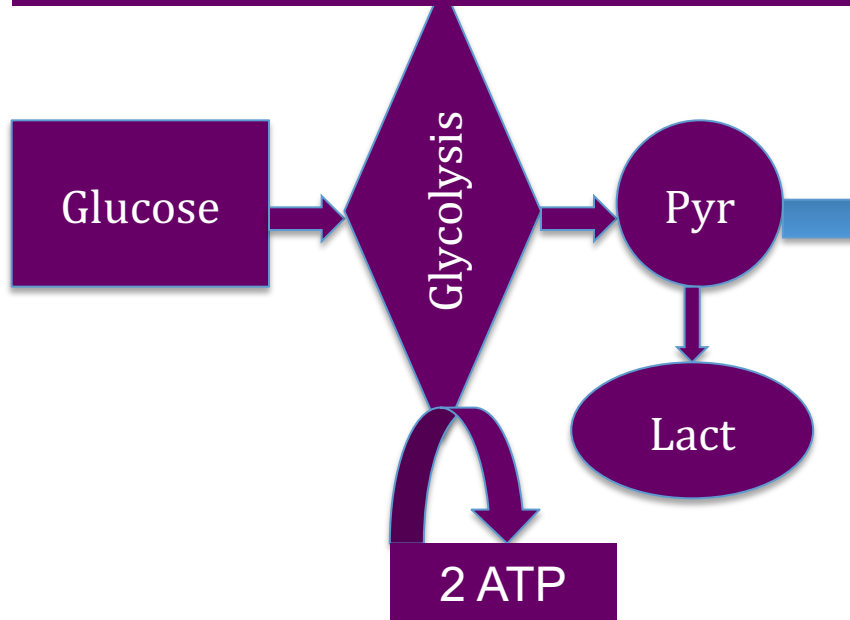
Anaerobic metabolism in glycolysis and oxidative transformation of glucose into the respiratory chain. ATP Adenosine triphosphate (Hacke 1991)



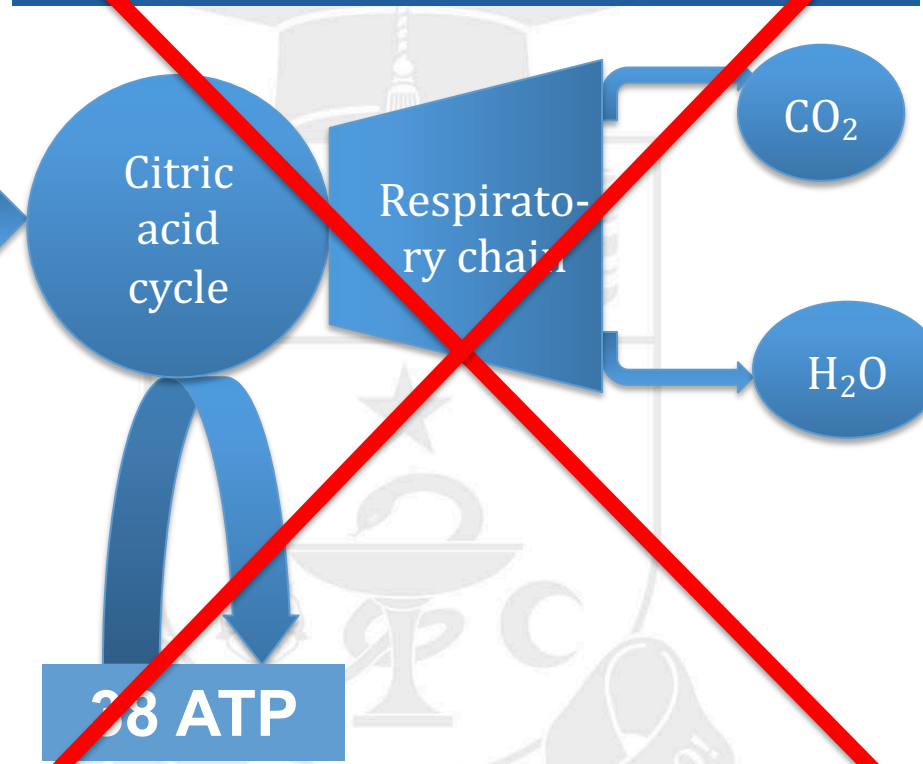
physiological – pathophysiological features:

ISCHEMIA !!!

ANAEROB METABOLISM



AEROB METABOLISM



Anaerobic metabolism in glycolysis and oxidative transformation of glucose into the respiratory chain. ATP Adenosine triphosphate

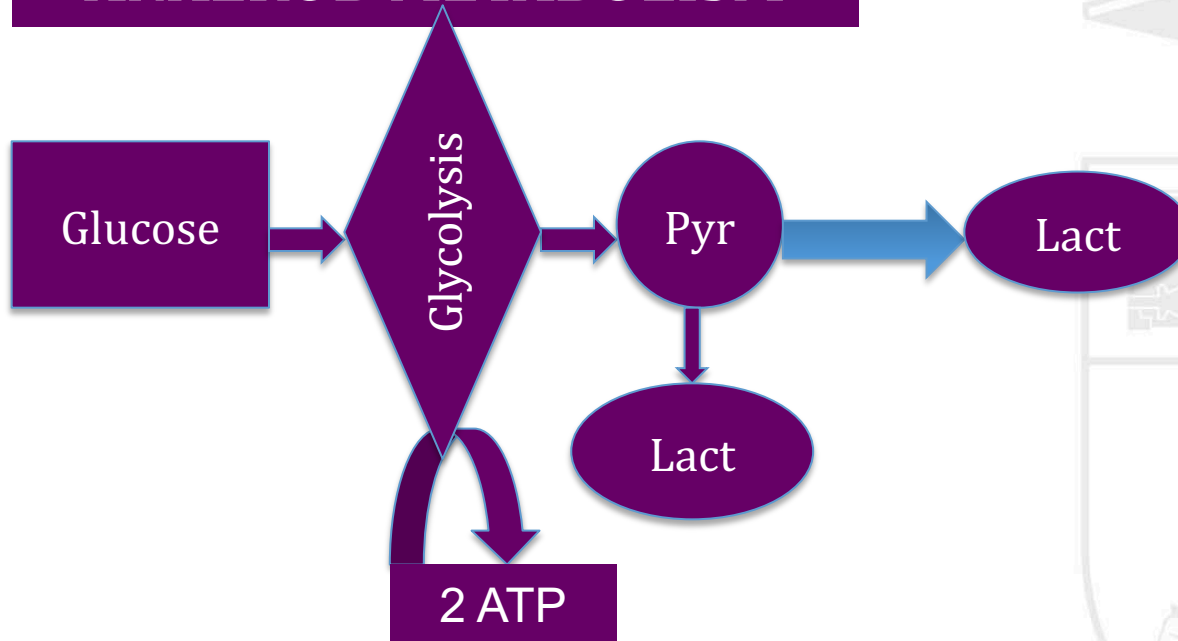
(Hacke 1991)



physiological – pathophysiological features:

ISCHEMIA !!!

ANAEROB METABOLISM

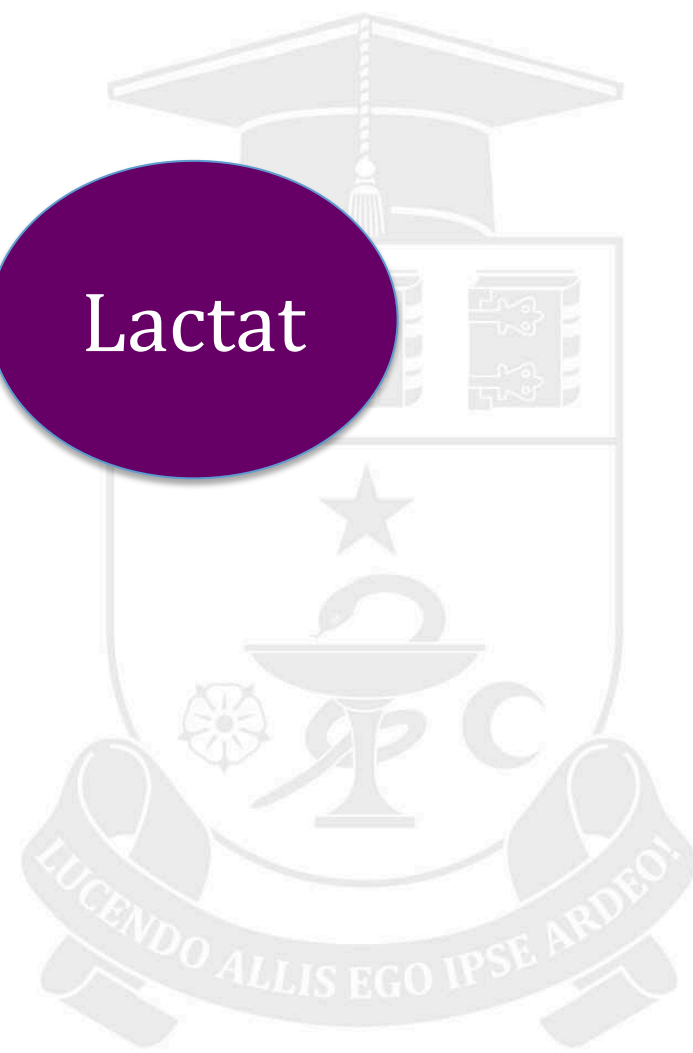
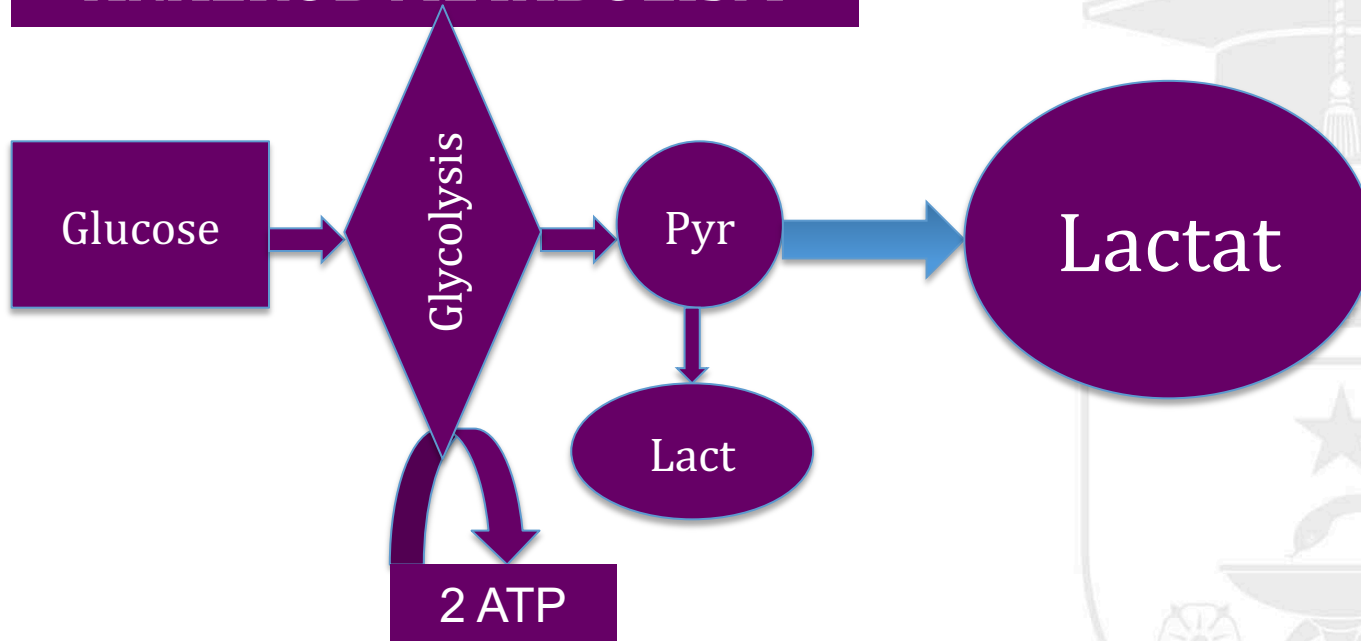




physiological – pathophysiological features:

ISCHEMIA !!!

ANAEROB METABOLISM

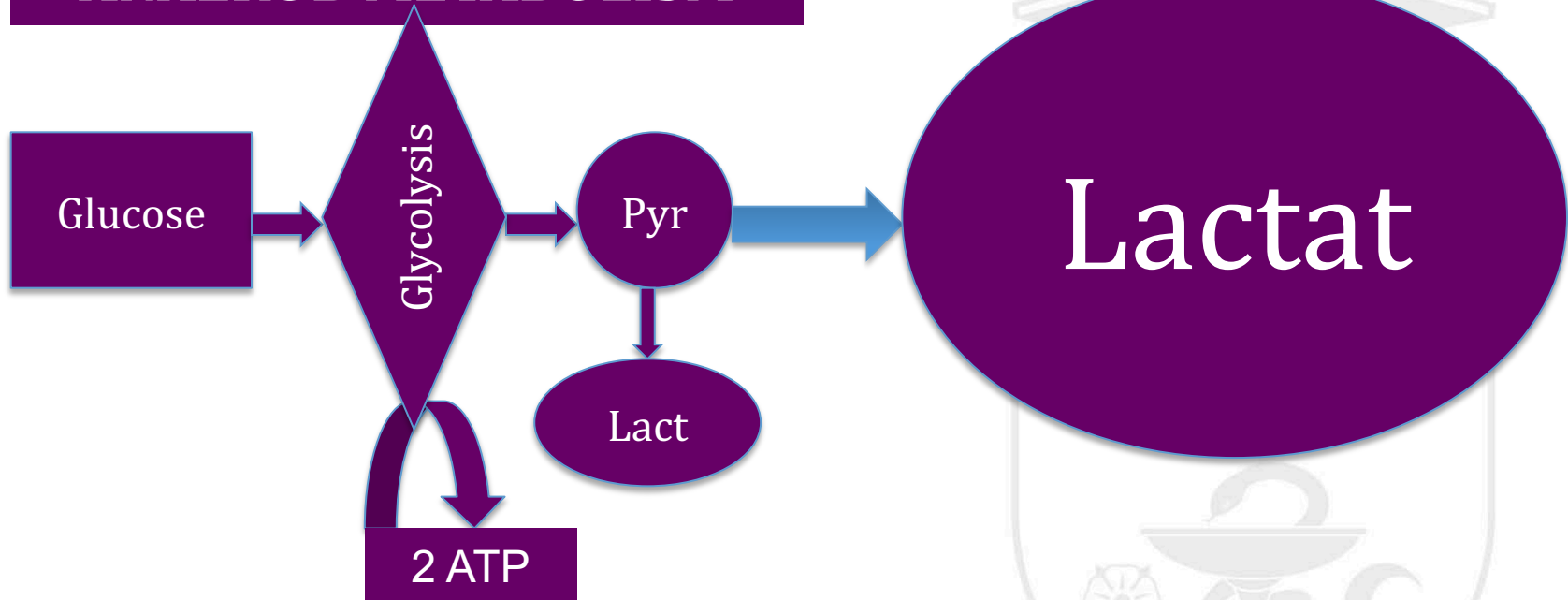




physiological – pathophysiological features:

ISCHEMIA !!!

ANAEROB METABOLISM

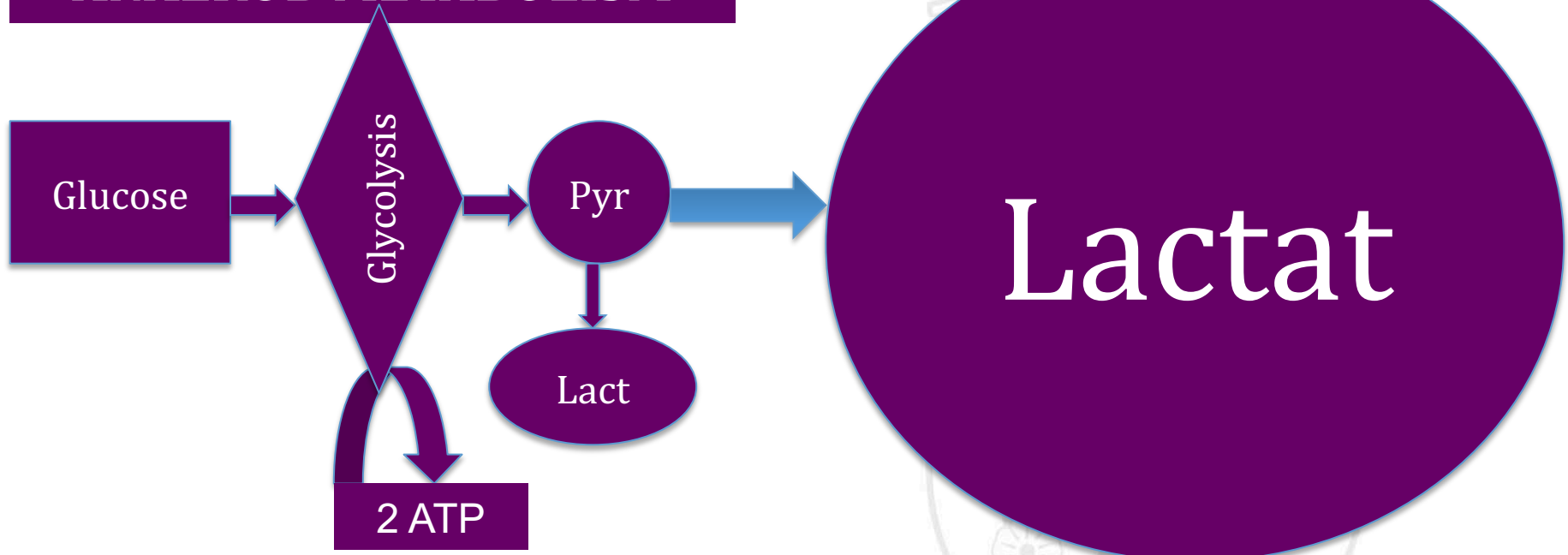




physiological – pathophysiological features:

ISCHEMIA !!!

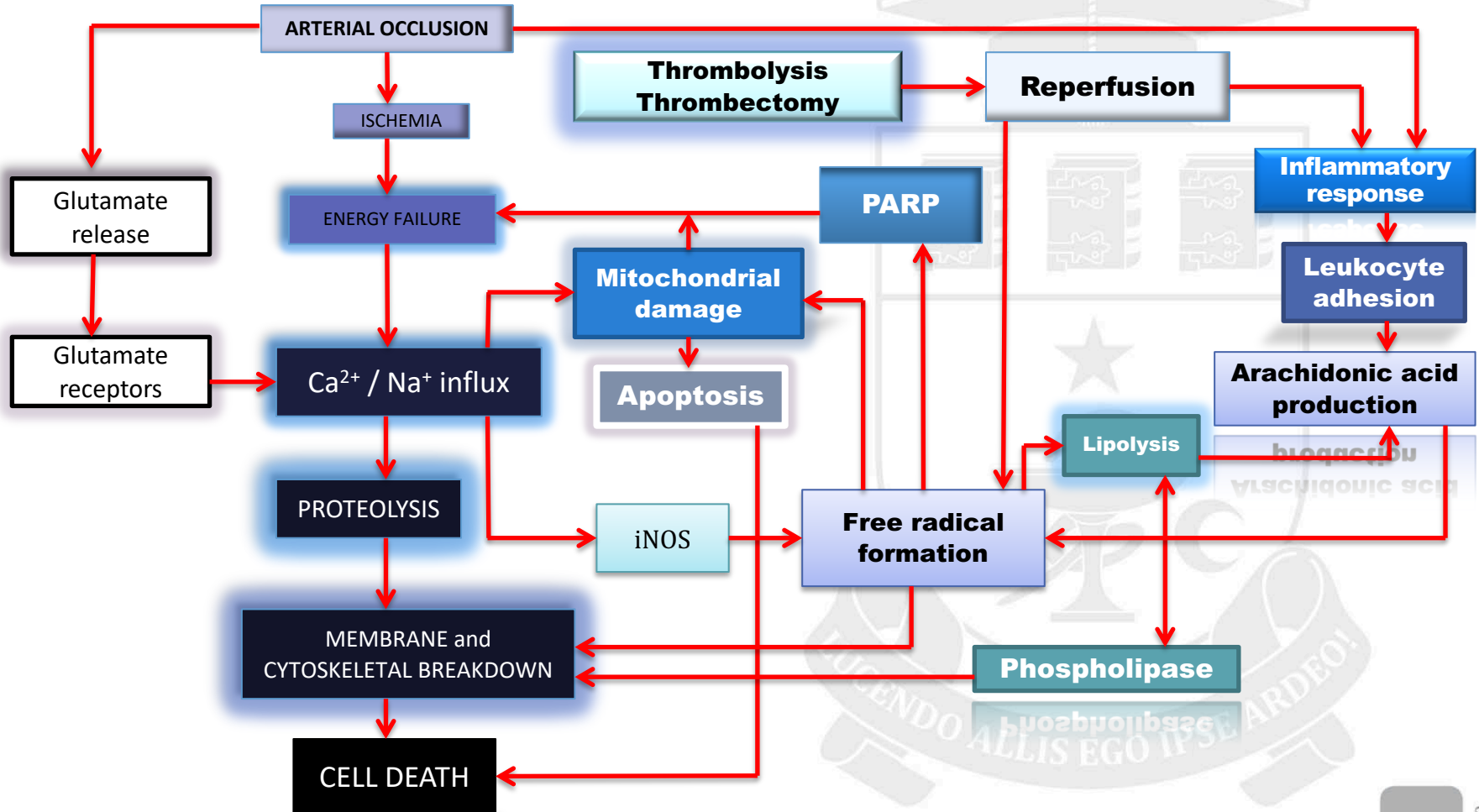
ANAEROB METABOLISM



CELL DEATH

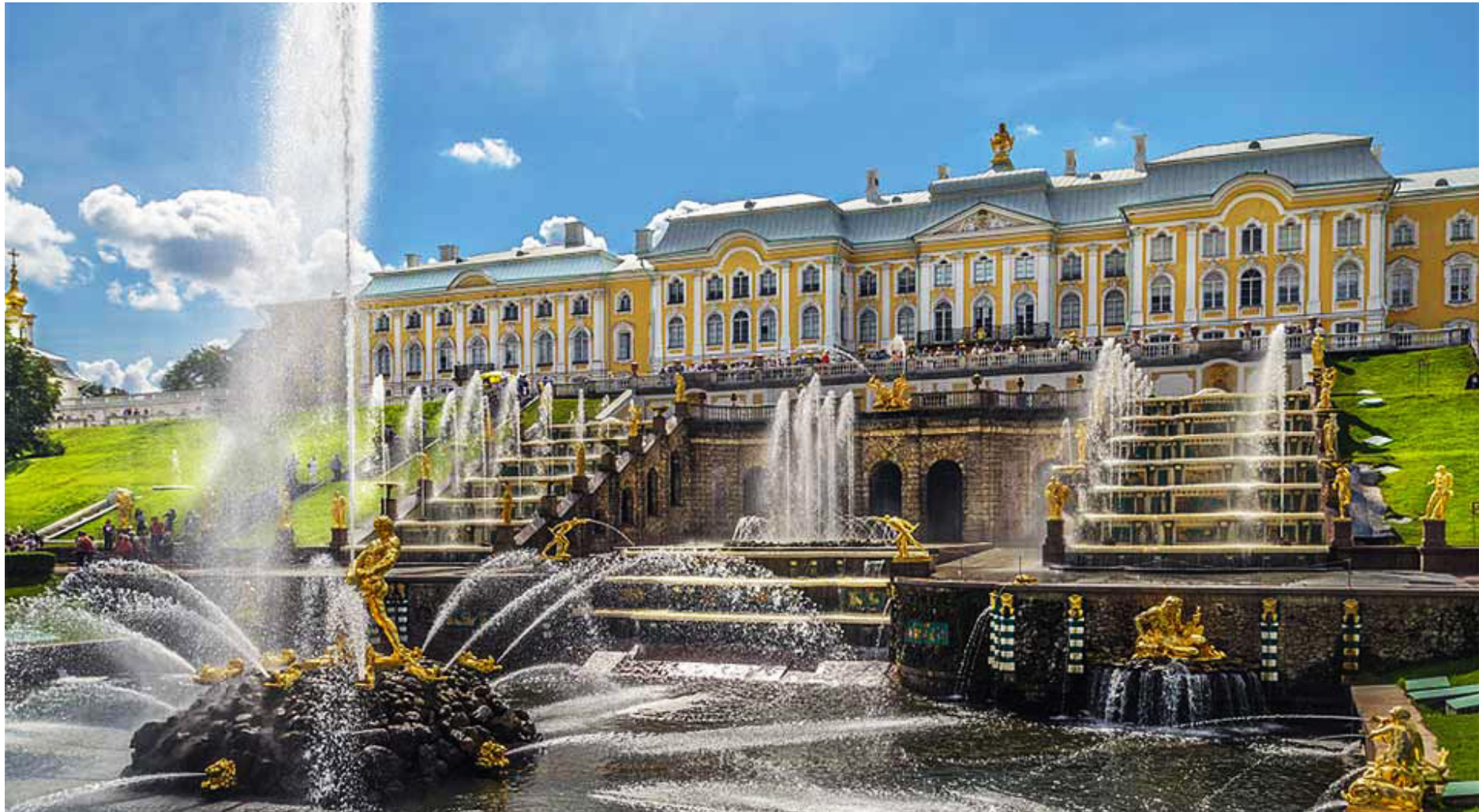


CASCADE OF CEREBRAL ISCHEMIA





Cascade of Peterhof Fountains





PATHOPHYSIOLOGY OF ISCHEMIC STROKE

Focal cerebral infarction occurs via two distinct pathways:

- (1) A necrotic pathway in which cellular cytoskeletal breakdown is rapid, due principally to energy failure of the cell; and
- (2) An apoptotic pathway in which cells become programmed to die.



ISCHEMIA:

**4,5
hours!**

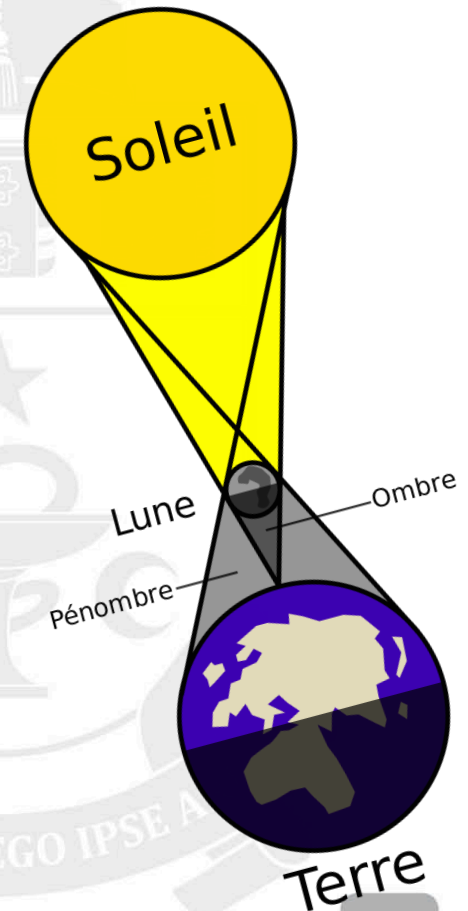
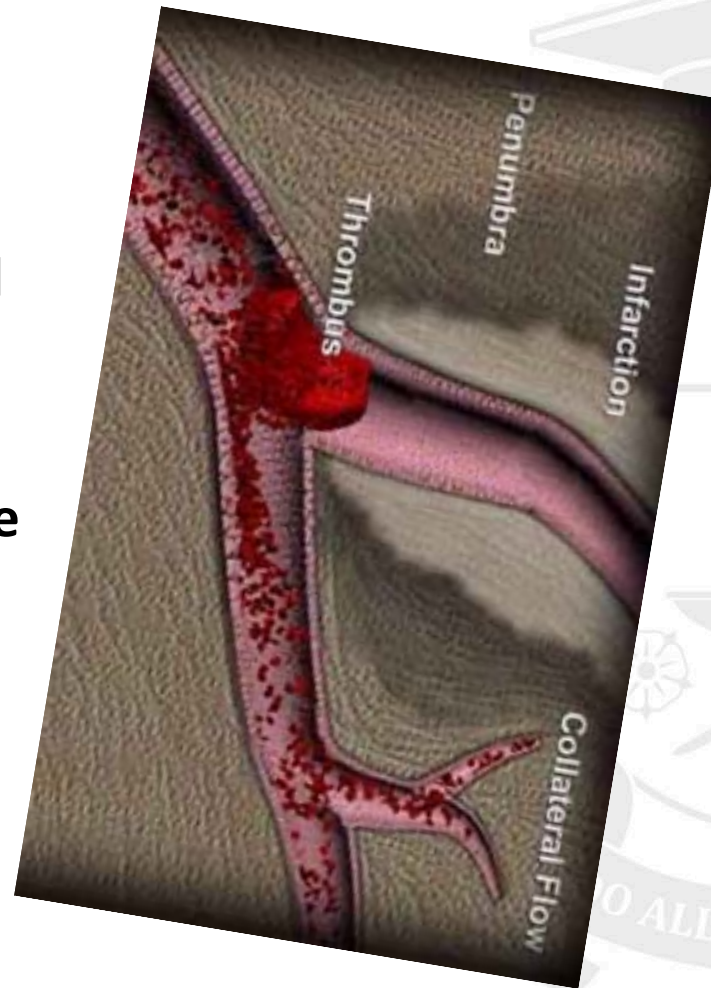




PENUMBRA

Size depends on:

- the proportions of diminishing regional cerebral blood flow
- the place of vascular occlusion and the function of the collateral circuit
- duration of hypoperfusion





STROKE

4.

ETHIOLOGY AND PATHOGENESIS



STROKE ETIOLOGY

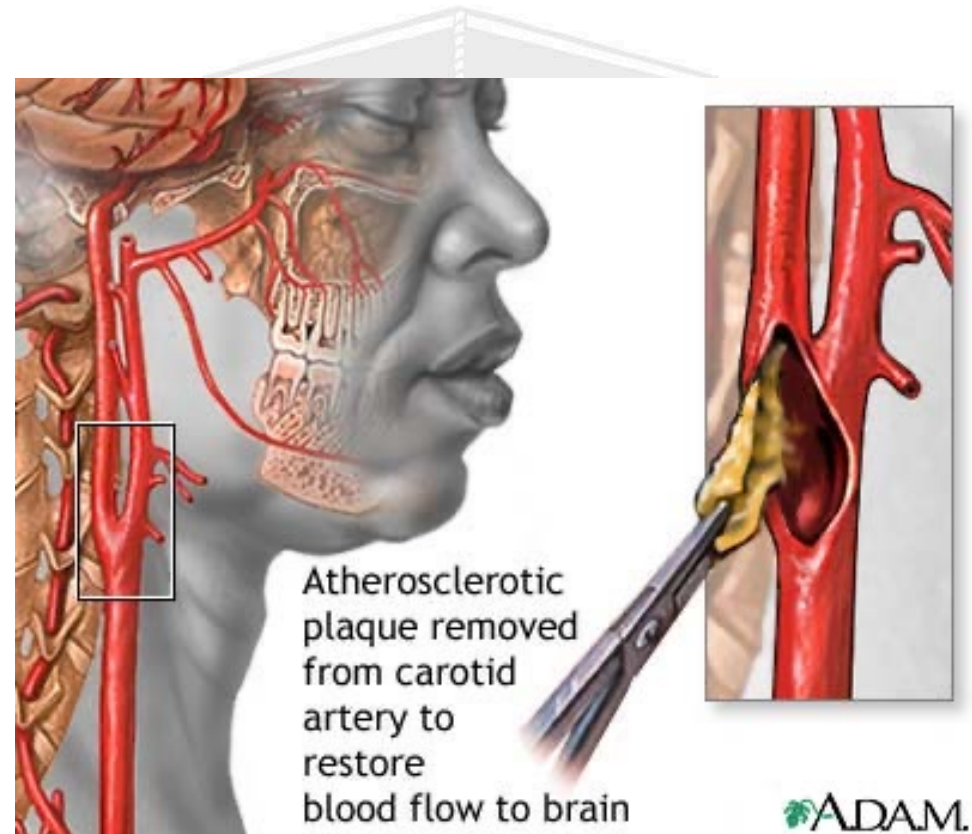
atherosclerosis
embolism
dissections

coagulopathy
vasculitis
other rare
causes



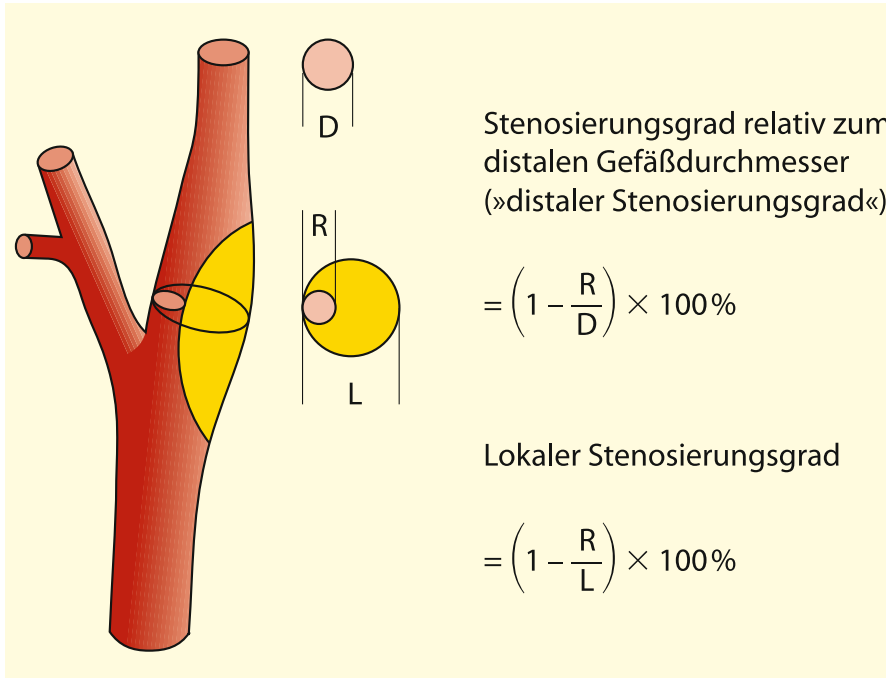
Atherosclerotic stenosis of the main arteries

- ***symptomatic*** (with neurological manifestations due to it)
- and
- ***asymptomatic*** (without neurological manifestations due to it).





Stenosis depending on the degree of narrowing



Calculation of the degree of narrowing
(Widder et al. 1986b (

<50% NASCET* *light*

50-70% NASCET *moderate*

70-90% NASCET
expressed

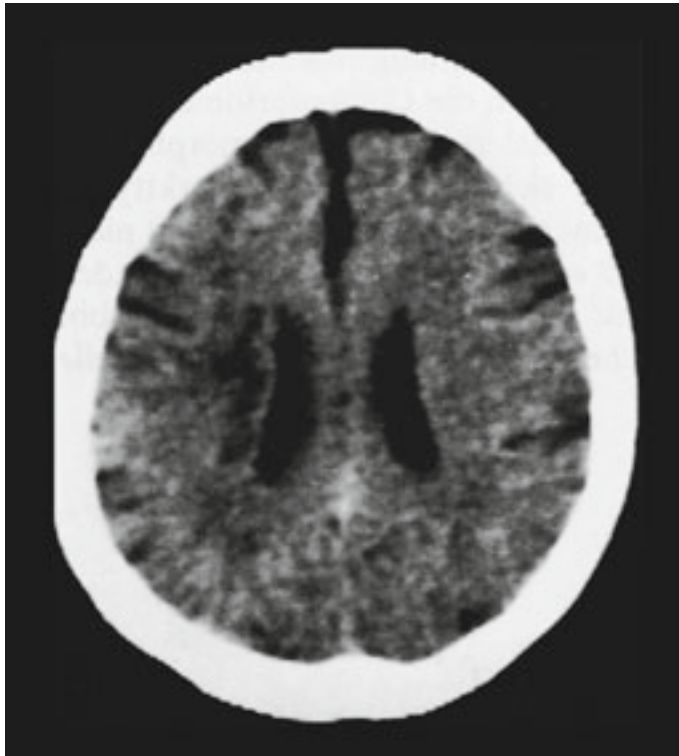
>90% NASCET
very pronounced

>95% NASCET
thread narrowing

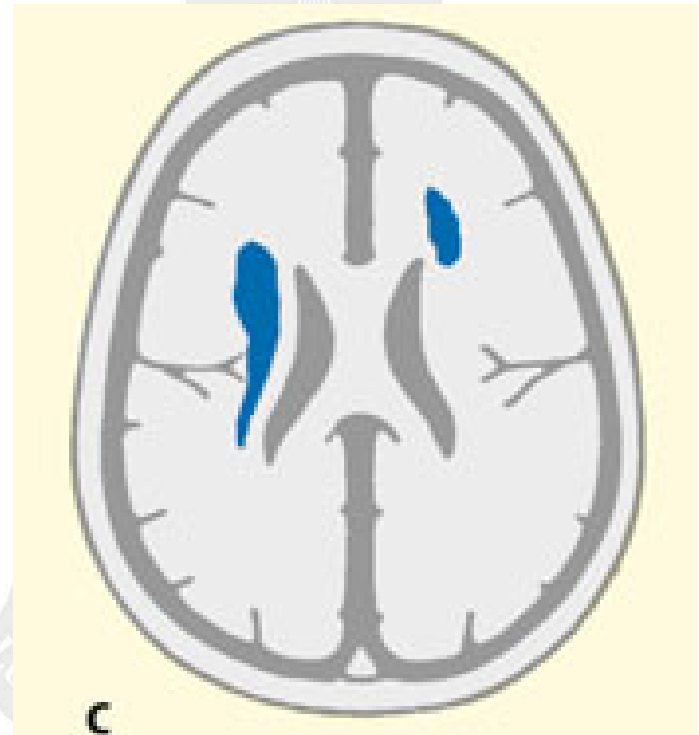


Stenosis ~80% causes hemodynamic ischemia

„last meadow“



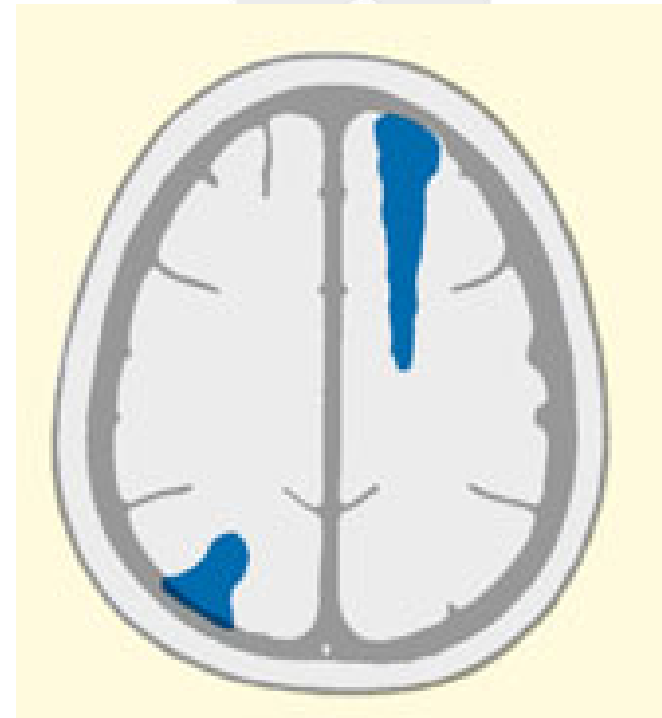
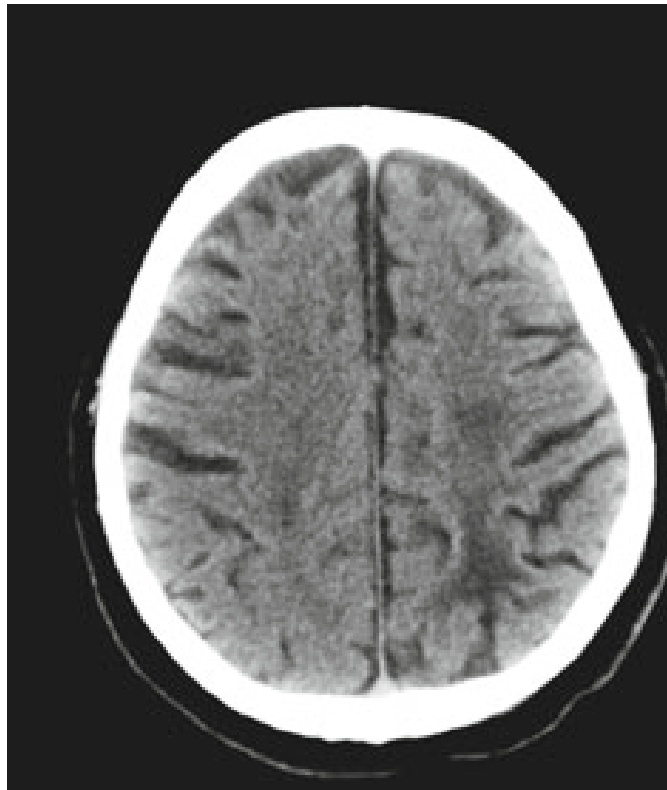
CT. Parasagittal infarction





hemodynamic ischemia

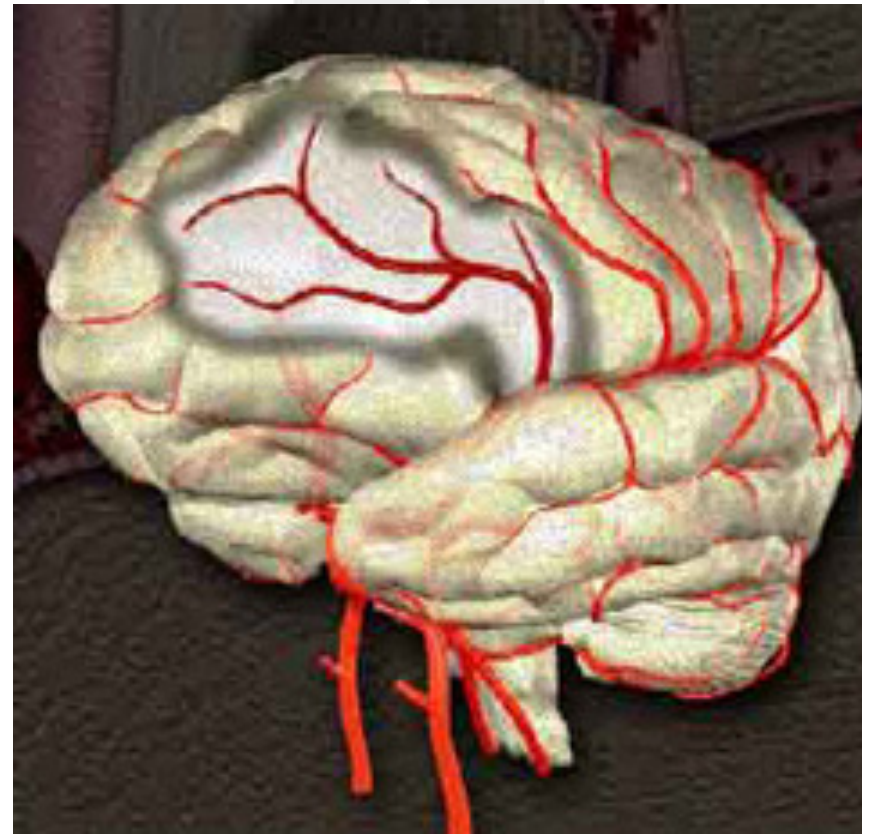
„watershed zones“





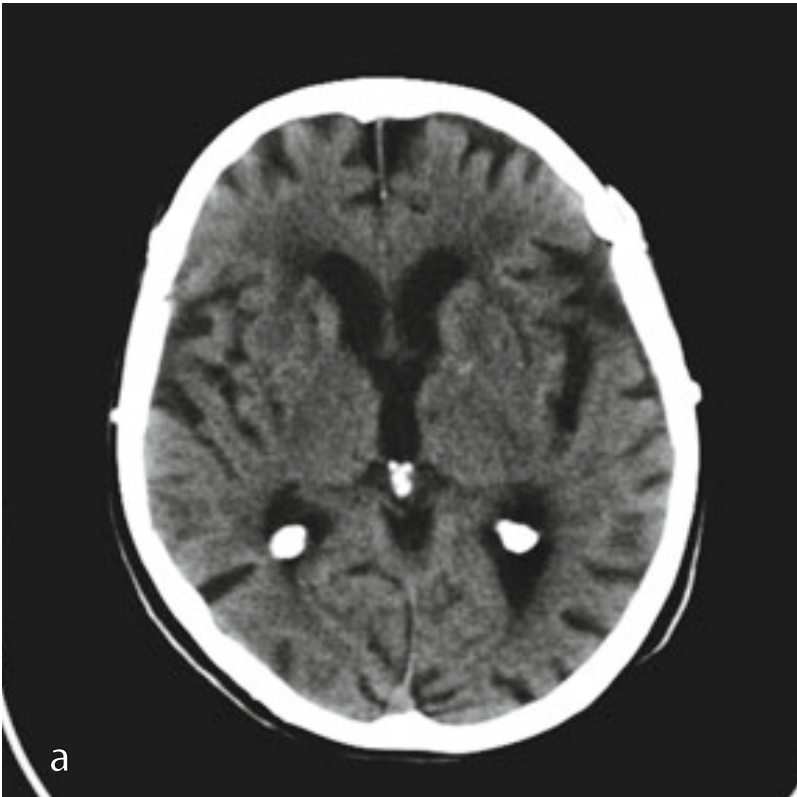
hemodynamic ischemia

„central infarction“

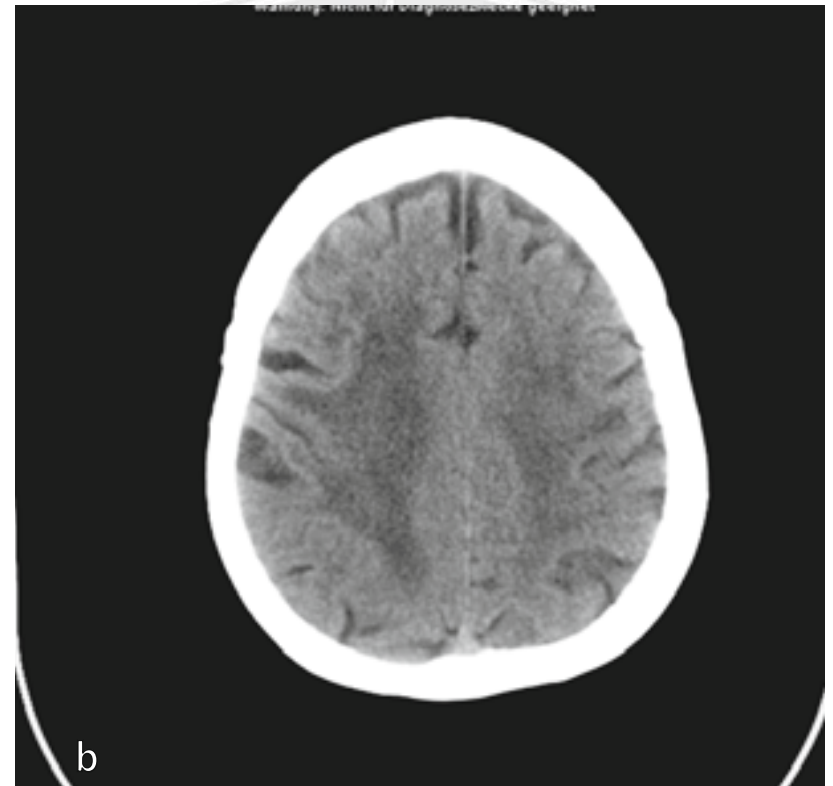




Lacunar infarction



a Status lacunaris



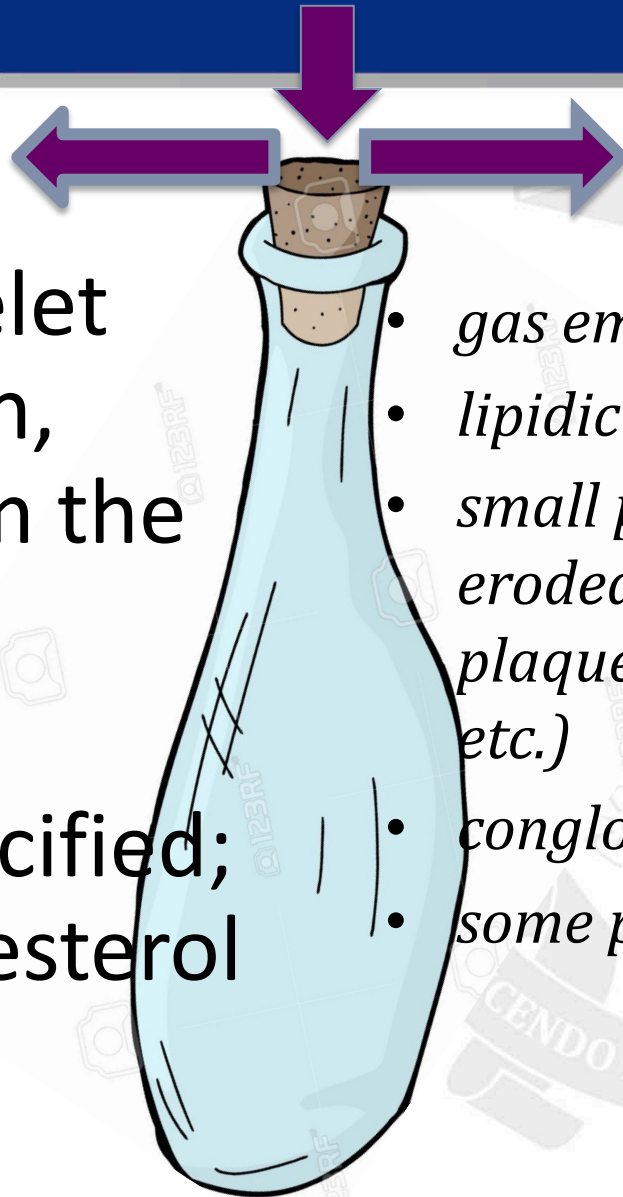
b Morbus Binswanger



EMBOL?

TROMBOTIC

- **Fresh:** Platelet composition, started from the ulcer plate
- **Organized:** partially calcified; rich in cholesterol

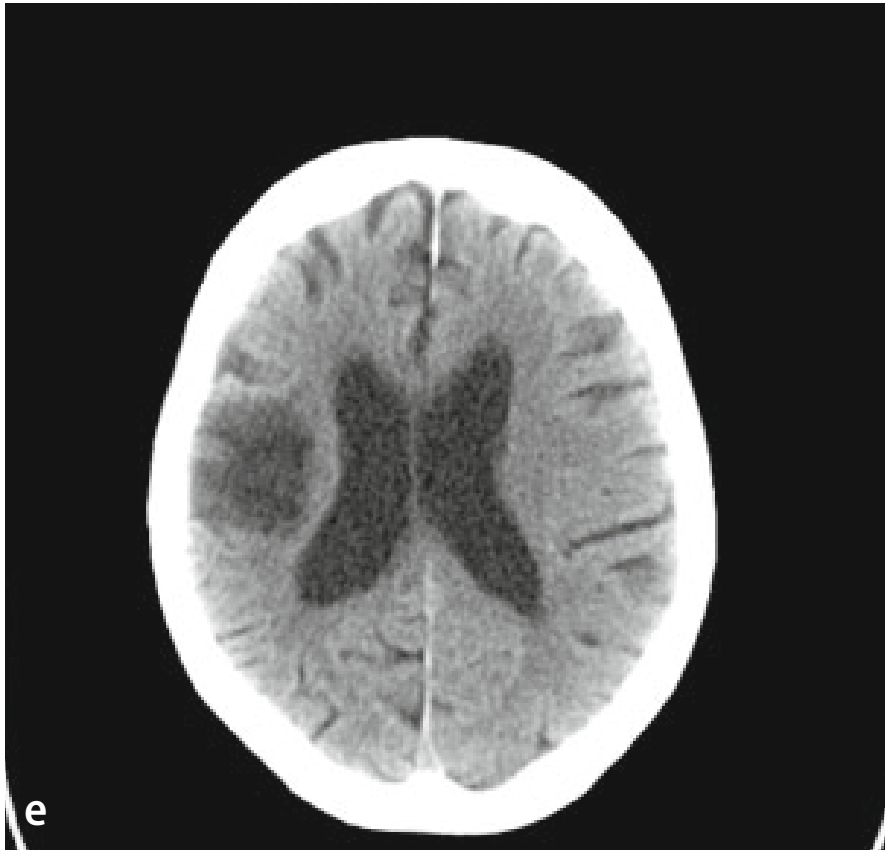


OTHER

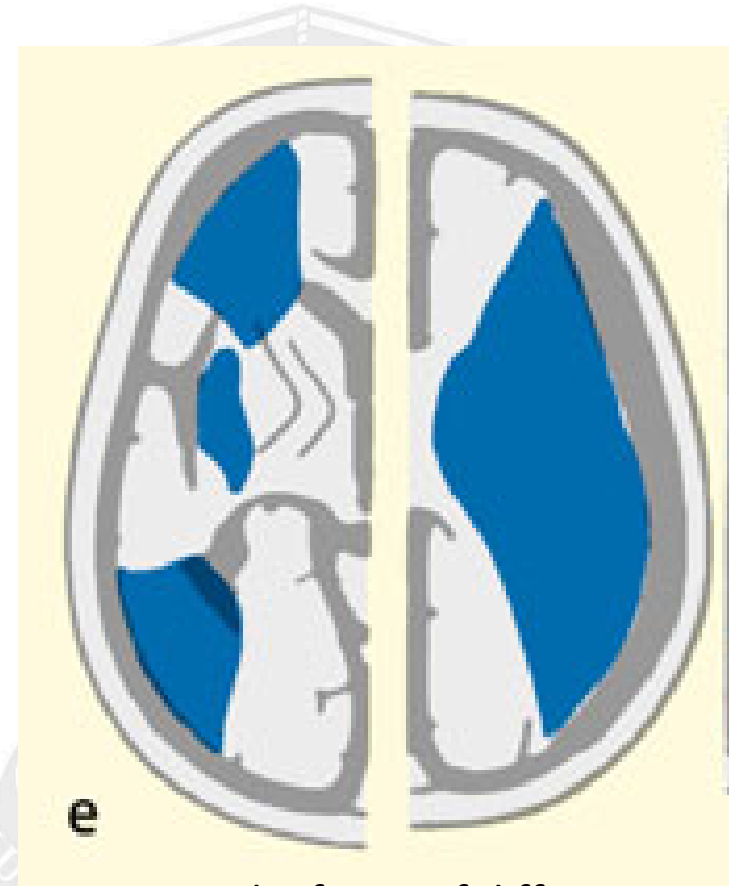
- *gas embolism*
- *lipidic embolism*
- *small pieces of tissue (from eroded atherosclerotic plaques, cancer metastases, etc.)*
- *conglomerates of bacteria*
- *some parasites*



EMBOLIC STROKE



e Territorial infarction in the middle branches of the middle cerebral artery



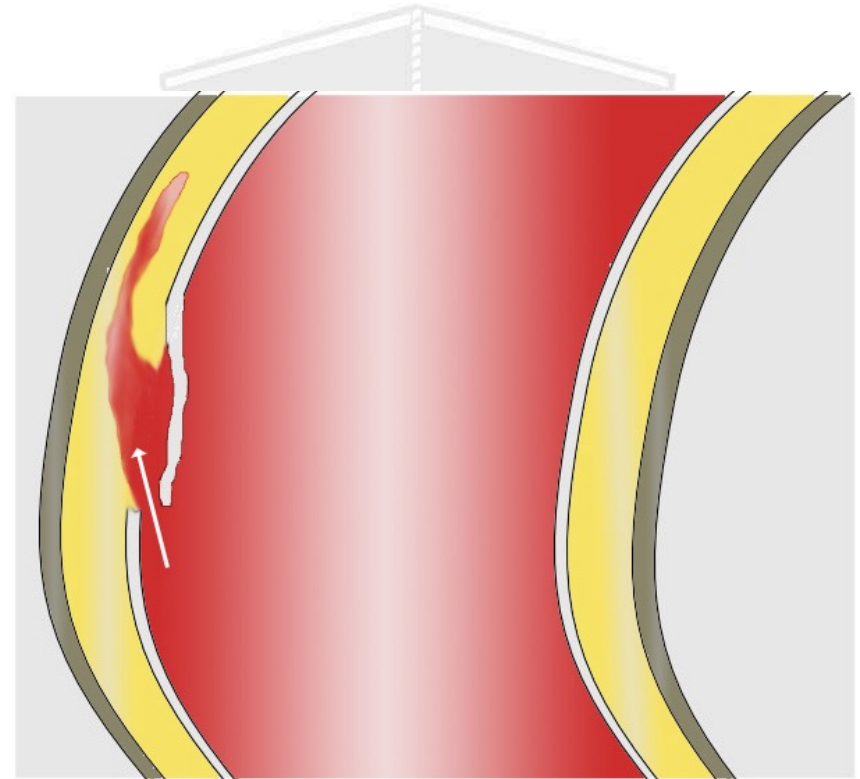
e Territorial infarcts of different thickness in the middle and posterior branches.

Ringelstein, 1985



DISSECTION

- *Dissections* are haemorrhages within the arterial wall (under the *intima*, in the medial tunica, under adventitia) that cuts / laments (detaches one another) of its layers and thus damages blood circulation.

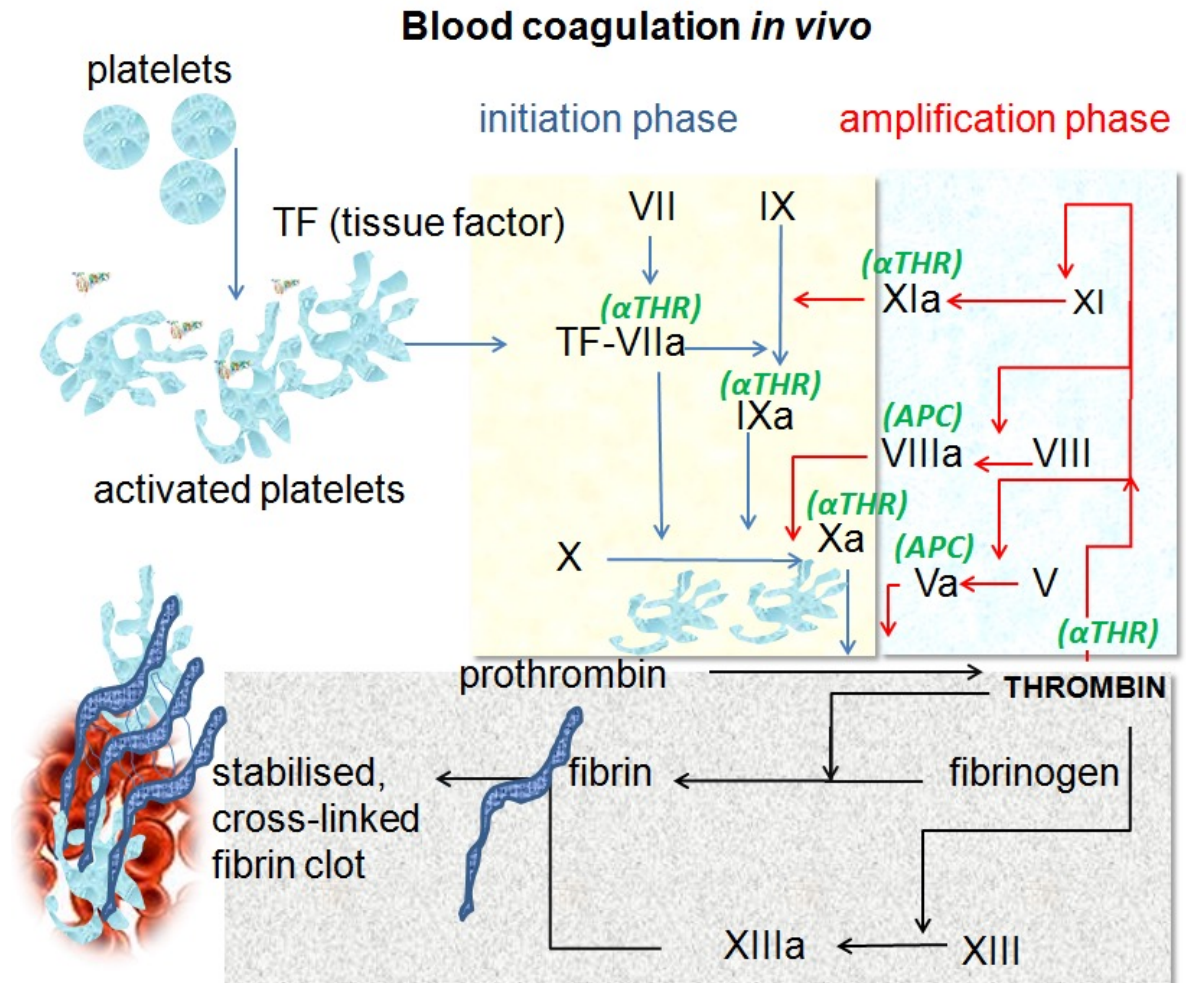


half of the cases are caused by trauma



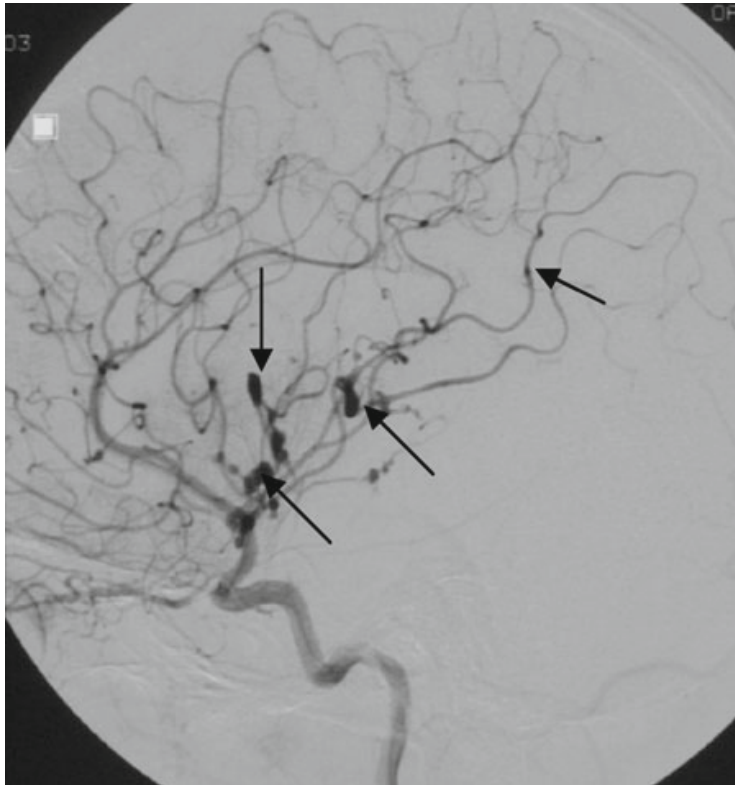
COAGULOPATHY

- Hipercoagulation





VASCULITIS



Angiography in vasculitis. Irregular (arrow) vessels follow successively with regular caliber vessels, sometimes vascular dilatations are chain-like (arrowhead).

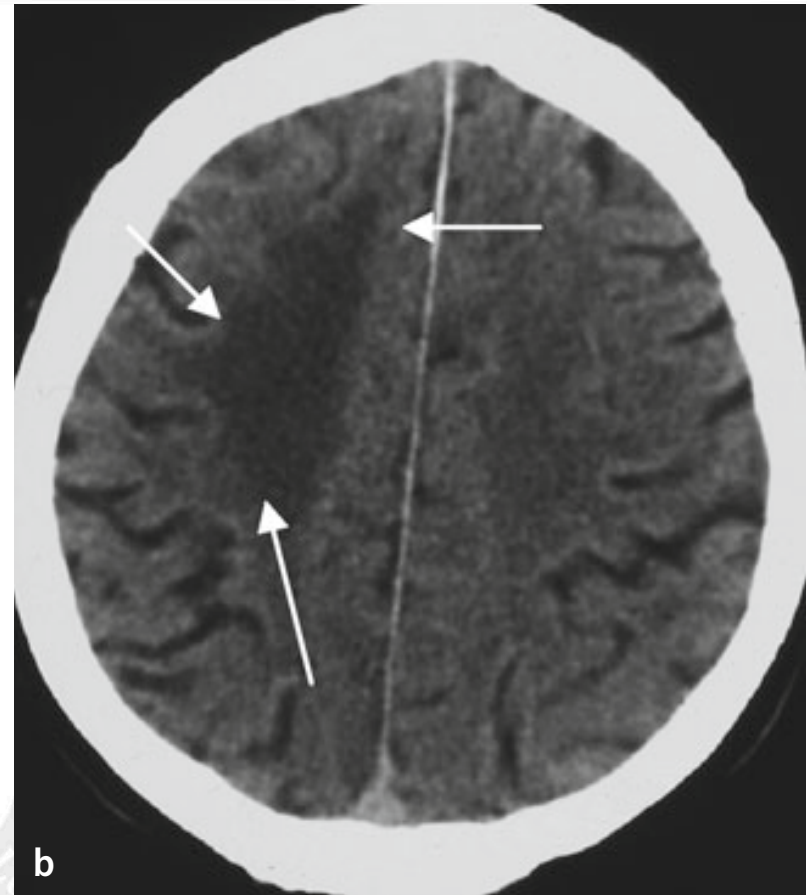
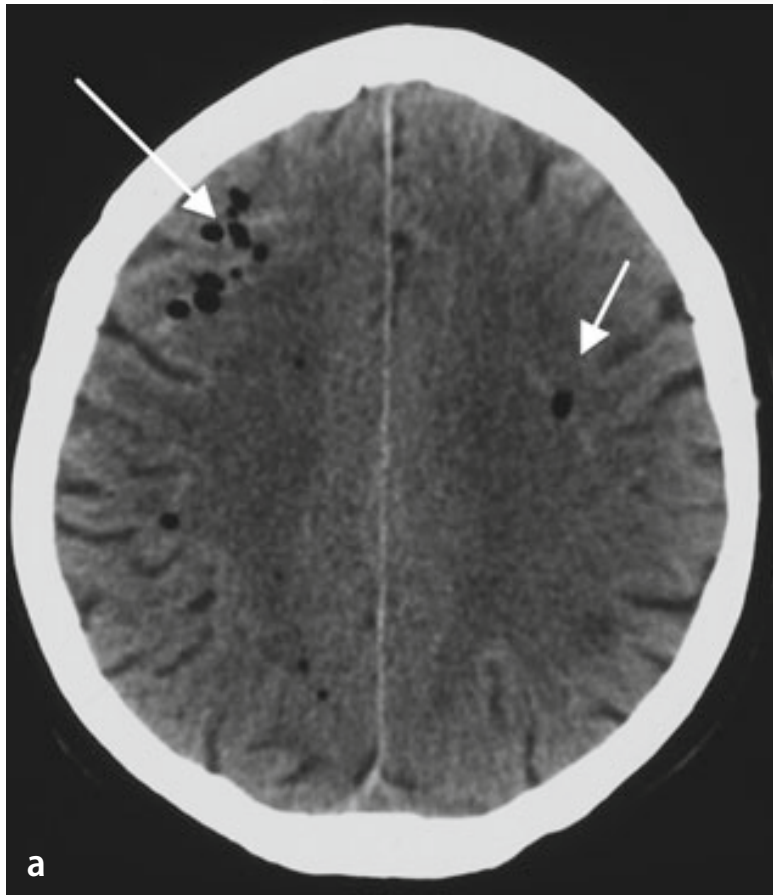
Specific antibodies, circulating immunocomplexes and endotoxins can directly damage the vascular wall, followed by vasospasm or thrombosis.

Vasculitis that may be accompanied by ischemic stroke:

- *Arthritis with giant cells*
- *Arteritis Takayasu*
- *Polyarteriitis nodosa*
- *Granulomatosis Wegener*
- *Churg-Strauss syndrome*
- *Behçet syndrome*
- *Isolated cerebral vasculitis*
- *Systemic lupus erythematosus*
- *Sjögren syndrome*



acute multiple air EMBOLISM (after surgical intervention on the heart)



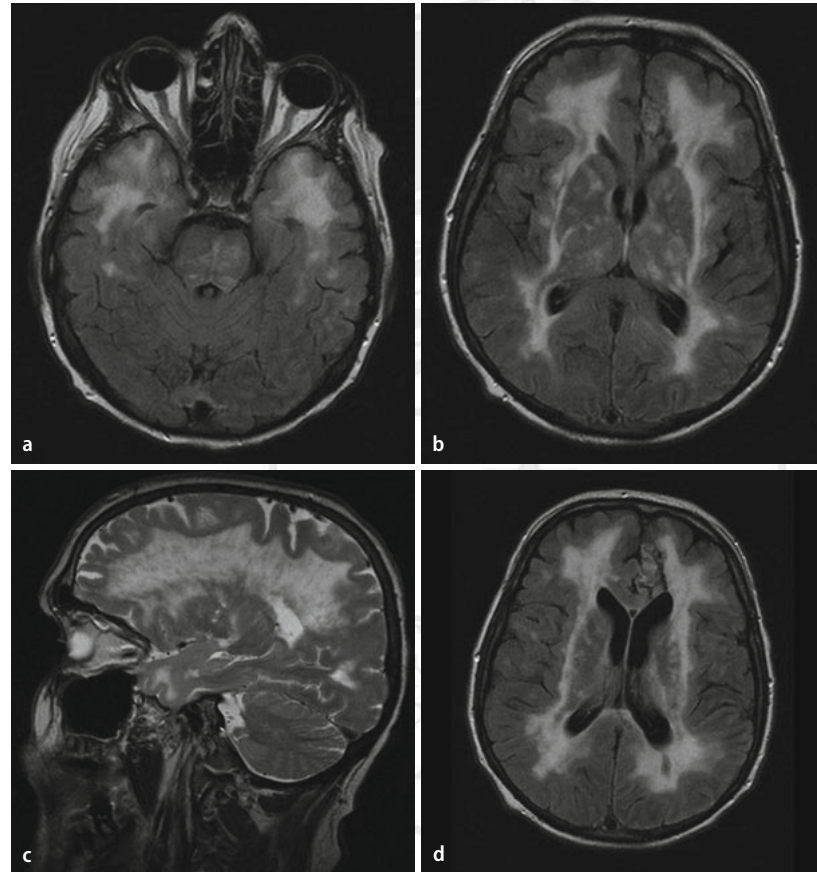
a Low density foci located on the border between the white and gray matter of the brain,
b A few days later: the air cleared, but in white matter, predominantly anterior, edema is noted (S. Hähnel, Heidelberg).



CADASIL

Cerebral Autosomal Dominant Arteriopathy With Subcortical Infarcts and Leukoencephalopathy

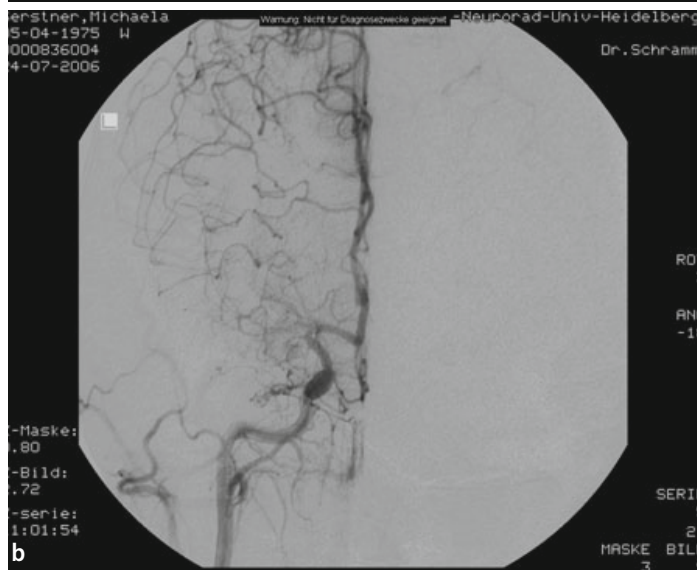
- is an autosomal-dominant inherited disease of small vessels that is manifested by recurrent stroke. It may progress to pseudobulbar paralysis or multi-infarct dementia.



MRI. a-d Subcortical extensive lesions with temporolateral and paraventricular prevalence



Moya-Moya Disease (japan: little cloud)



ANGIOGRAPHY

- a Left MCA Stenosis with collateral vascular net onset generation at the base of the skull.
- b Right-side unfolding with straight-line obturation, as well as a clearly pronounced network of collateral.



ISCHEMIC STROKE

5. CLASSIFICATION



ISCHEMIC STROKE CLASSIFICATION

CRITERIONS:

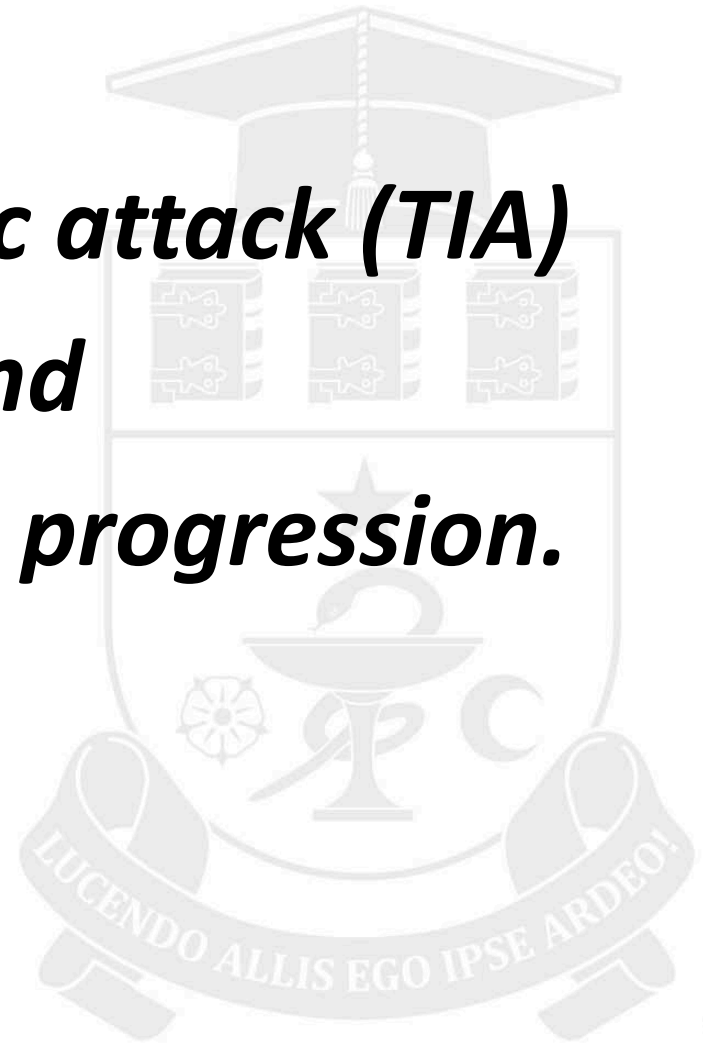
- 1. Evolution**
- 2. Size of artery**
- 3. Etiology**
- 4. Pathogenesis**
- 5. Vascular territory**
- 6. Expression of clinical manifestations**





EVOLUTION

- ***Transient ischemic attack (TIA)***
- ***ischemic stroke and***
- ***ischemic stroke in progression.***





Transient ischemic attack (TIA)

A sudden, focal neurologic deficit lasting fewer than 24 hours confined to an area of the brain or eye perfused by a specific artery, and presumed to be of vascular origin (Albers et al 2002).

- Studies using diffusion-weighted MRI show that about 1/3 of all events classified as TIA are associated with positive scans (Brazzelli et al 2014).



Ischemic STROKE

Ischemic stroke may have light, moderate and severe symptoms. Severe degrees, for example, can be manifested by global aphasia, hemiplegia, stable hemianopsia.

- To assess the severity of clinical manifestations in the acute phase of Stroke, the NIHSS scale is used.



The National Institutes of Health Stroke Scale (NIHSS)

is a tool used by healthcare providers to objectively quantify the impairment caused by a stroke.

Score	Stroke severity
0	No stroke symptoms
1 - 4	Minor stroke
5 - 15	Moderate stroke
16 - 20	Moderate to severe stroke
21 - 42	Severe stroke



Ischemic STROKE in progression

Clinically - imaging evolves negatively in progredient or saltatory (crescendo TIA) over hours. The preferential location is the internal capsula and the Varolio pons.

- It rarely meets and usually presents diagnostic difficulties



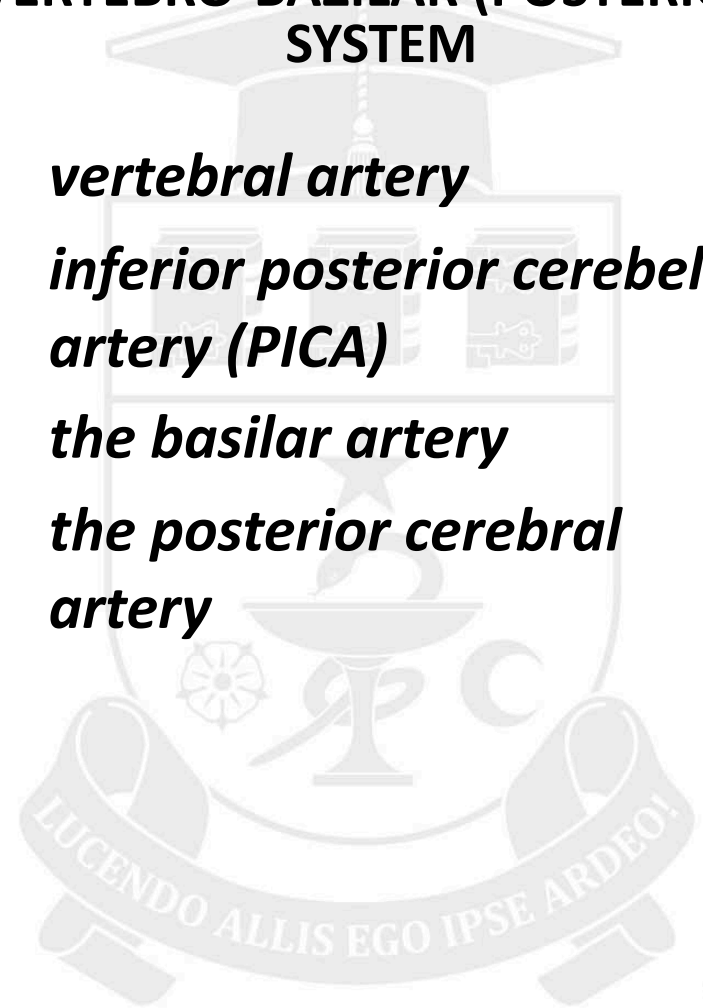
Ischemic stroke according vascular territory

CAROTIDIAN (ANTERIOR) SYSTEM

- *the internal carotid artery*
- *the ophthalmic artery*
- *the middle cerebral artery*
- *the anterior cerebral artery*

VERTEBRO-BAZILAR (POSTERIOR) SYSTEM

- *vertebral artery*
- *inferior posterior cerebellar artery (PICA)*
- *the basilar artery*
- *the posterior cerebral artery*





ESUS – embolic stroke of undetermined source

It refers to ischemic stroke that has an embolic morphological (imagistic) manifestation, but where examinations performed according to the standard protocol cannot certify the origin of the embolus.



ESUS – embolic stroke of undetermined source

It is assumed that in most cases ESUS comes from unidentified atrial fibrillation. The term ESUS replaced the previous term "cryptogenic stroke", that is, stroke of unidentified etiology.



6.

CLINICAL MANIFESTATIONS AND VASCULAR SYNDROMES



STROKE recognition

Симптомы инсульта

Если помощь пострадавшему не будет оказана в течении 3,5 часов, ему грозит глубокая инвалидность!

FAST тест на симптомы инсульта

F
1

Face
Парализовало правую или левую половину лица

A
2

Arm
Слабость в одной руке

S
3

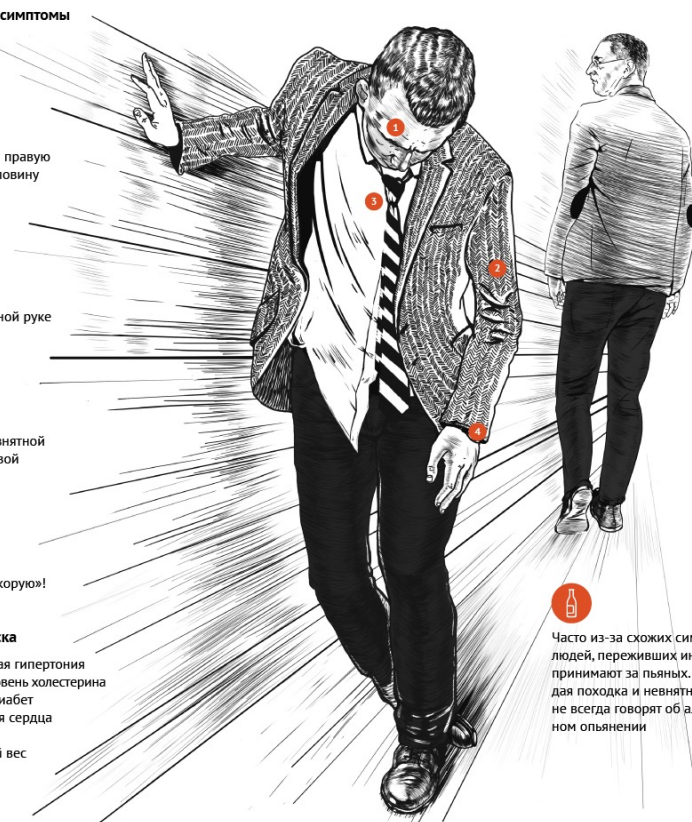
Speech
Речь стала невнятной и неразборчивой

T
4

Test
Вызывайте «скорую»!

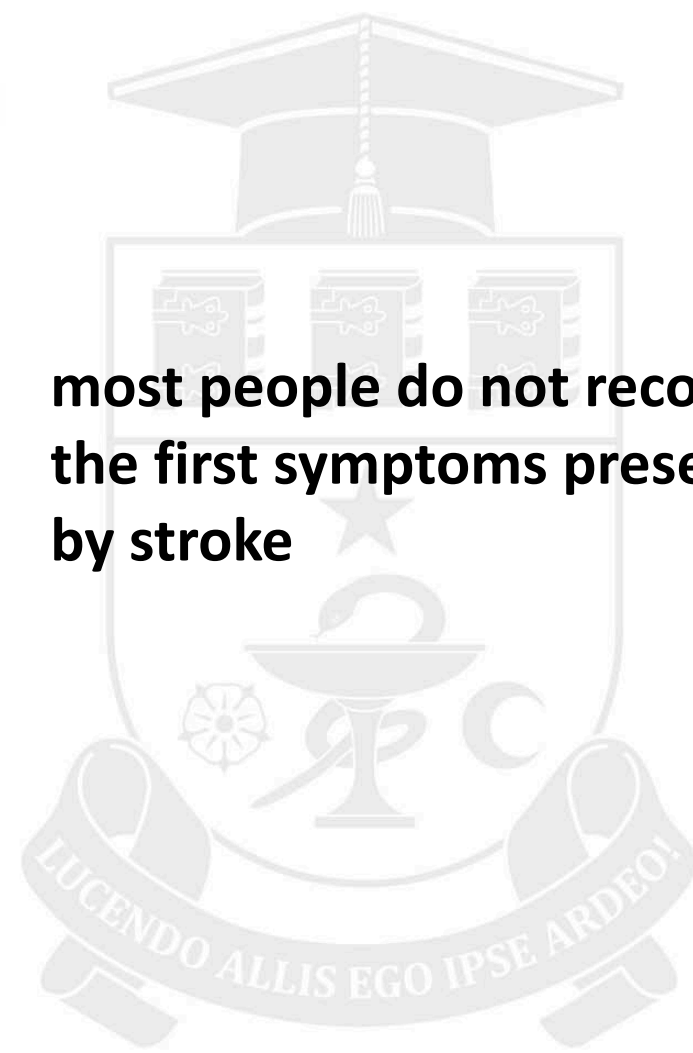
Факторы риска

- Артериальная гипертония
- Высокий уровень холестерина
- Сахарный диабет
- Заболевания сердца
- Курение
- Избыточный вес



Часто из-за схожих симптомов людей, переживших инсульт, принимают за пьяных. Нетвердая походка и невнятная речь не всегда говорят об алкогольном опьянении

- most people do not recognize the first symptoms presented by stroke





Early (alarming) clinical manifestations of a stroke

- the sudden onset of numbness and / or weakness / asymmetry of the face, trunk of the body, arm, wrist, thigh, leg - all usually on the same side (right / left);
- unexpected installation of vision disorders in one or both eyes, doubling of vision, blurring of vision, blemishes in the field of vision, blurred vision;



Early (alarming) clinical manifestations of a stroke

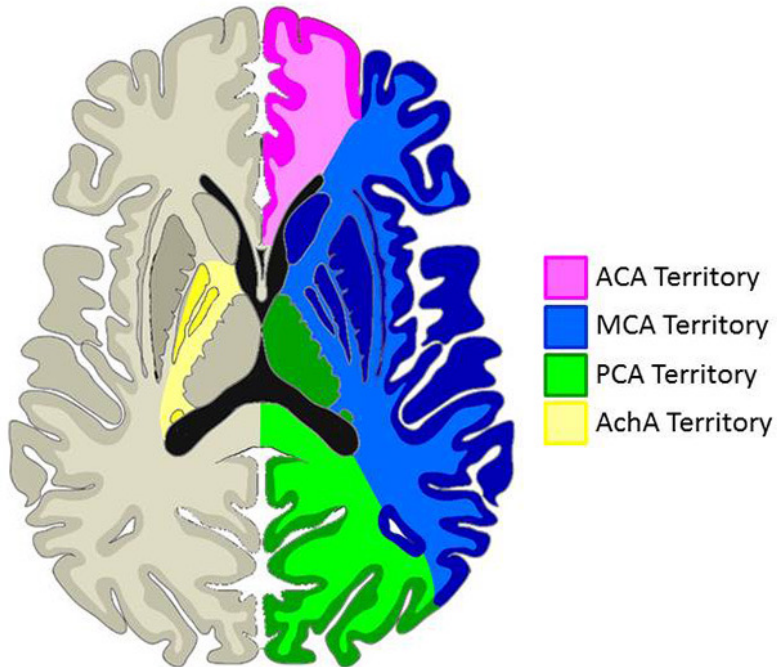
- occurrence of confusion, dizziness, nausea, vomiting, movement and balance coordination disorders;
- the onset of severe headache, neck tightness;
- seizure development; loss of consciousness and noisy breathing.



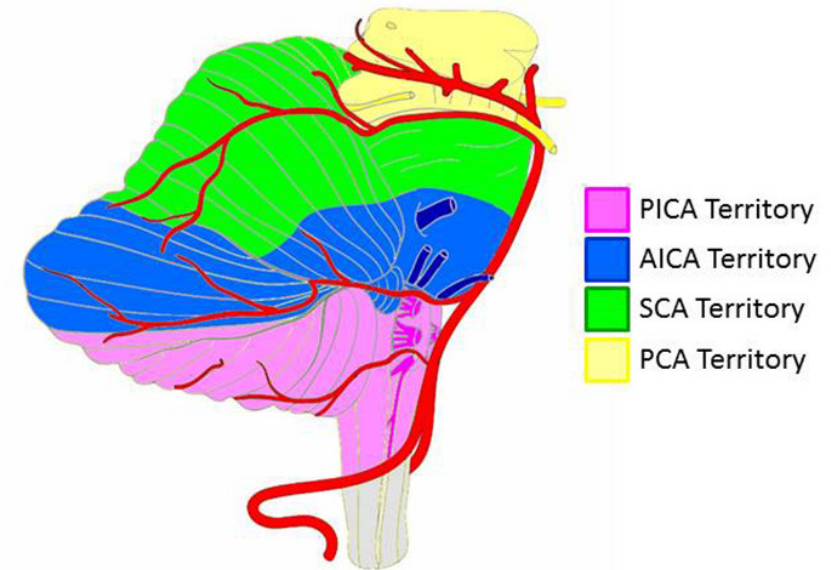
Clinical manifestations of ischemic stroke

VASCULAR SINDROMS

HEMISPHERIC



VERTEBRO-BAZILAR

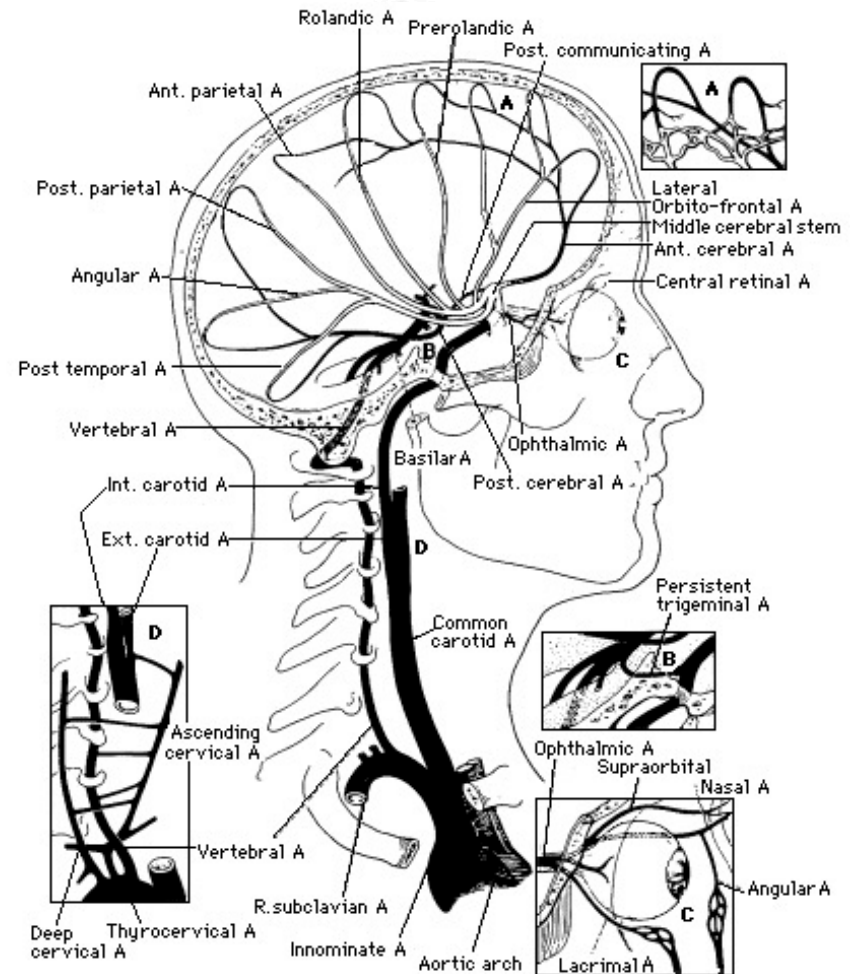




Clinical manifestations of ischemic stroke

CAROTIDIAN TERRITORY

- **INTERNAL CAROTIDE artery syndrome**
- **ophthalmic artery syndrome**
- **MIDDLE cerebral artery syndrome**
- **ANTERIOR cerebral artery syndrome**
- **BILATERAL Anterior Cerebral Artery syndrome (bilateral anterior infarct)**

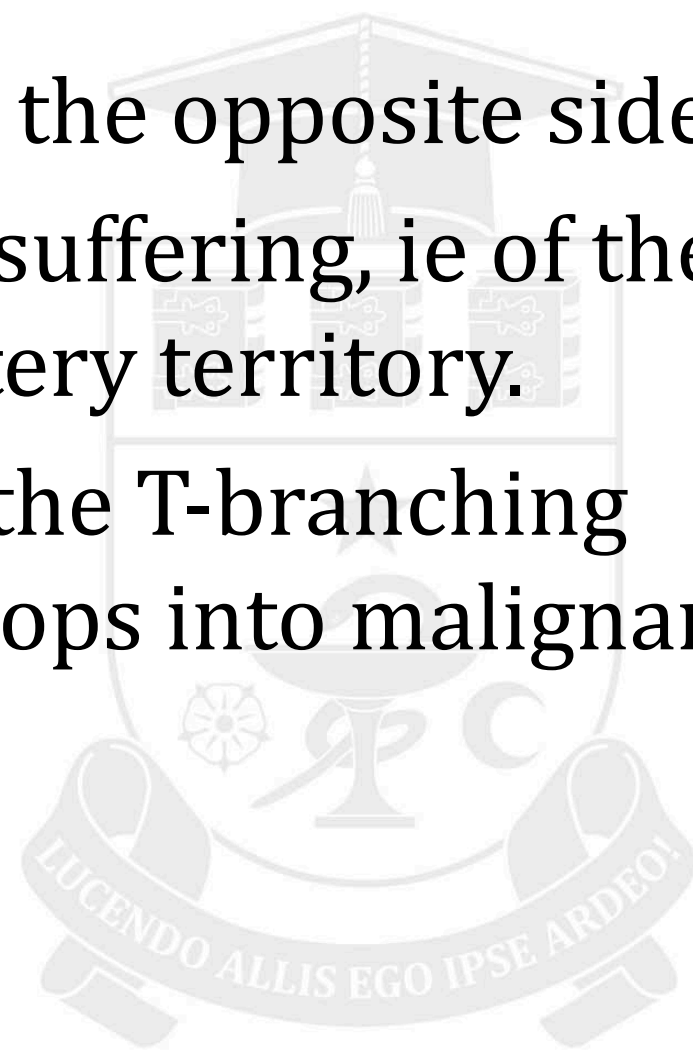




Clinical manifestations of ischemic stroke

INTERNAL CAROTIDE artery syndrome

- Hemisindromes on the opposite side.
- “Central” ischemic suffering, ie of the middle cerebral artery territory.
- Acute occlusion in the T-branching portion often develops into malignant infarction.





Clinical manifestations of ischemic stroke

MIDDLE CEREBRALE artery syndrome

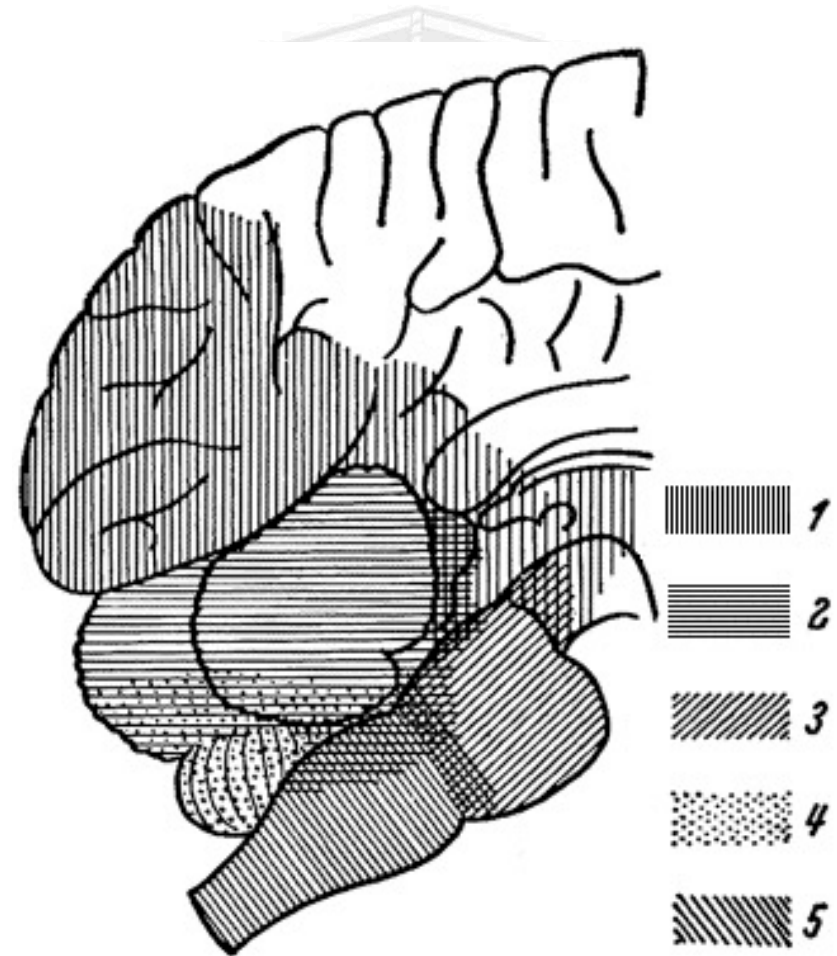
- It represents the "classic" variant, the most common of a stroke
- Hemiparesis with accentuation in the upper limb, hemi-hypoesthesia, speech disorders.
- A wide variety of motor disorders of speech, neuropsychological syndromes (global, non-fluent aphasia; apraxia; alexia; acalculia).
- Eventually, early muscle hypertonus, visual field disorders may develop.





Clinical manifestations of ischemic stroke in vertebral-basilar territory

- **VERTEBRAL** artery syndrome
- **PICA** – *posterior inferior cerebelar artery syndrome*
- **WALLENBERG** syndrome
- **BASILAR** artery syndrome (*caudal, middle, apex*)
- **POSTERIOR** cerebral artery syndrome
- **BILATERAL POSTERIOR** cerebral artery syndrome
- **Subclavian artery steal syndrome**

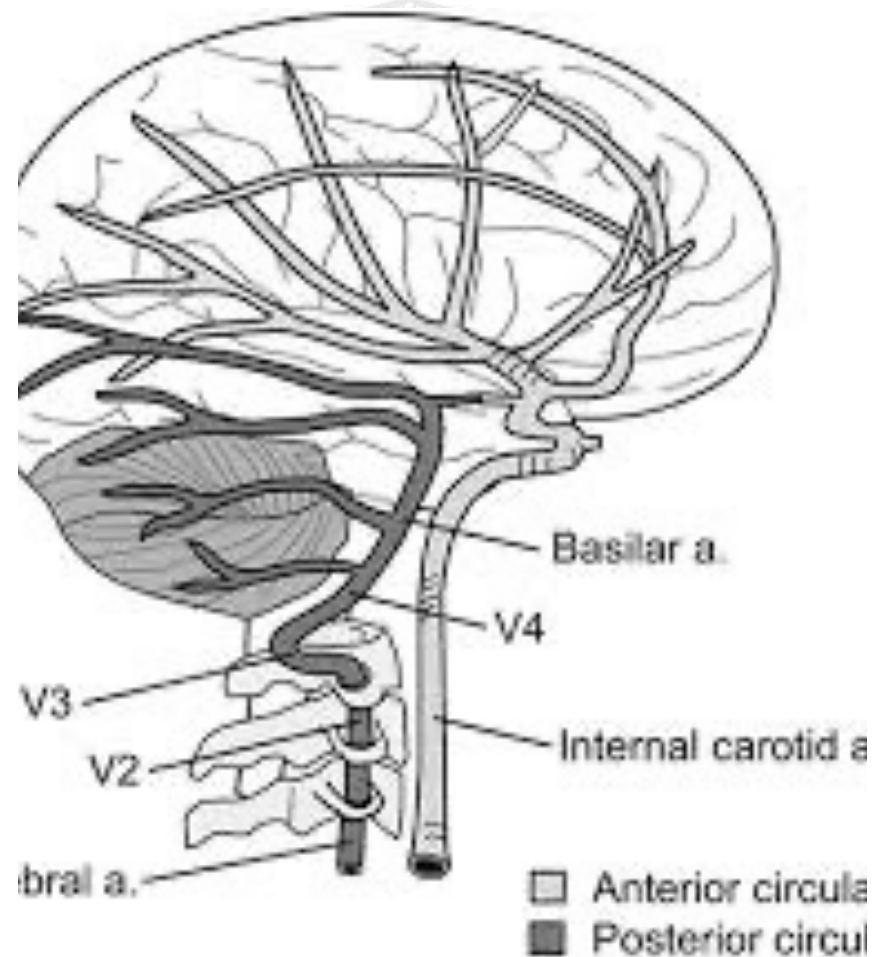




Clinical manifestations of ischemic stroke

Vertebral artery syndrome

- vertigo, nystagmus, diplopia, muscular atony
- basilar artery syndrome with the respective clinical symptomatology.
- when one (left or right) vertebral artery is healthy, blocking of the opposite side remains clinically asymptomatic.

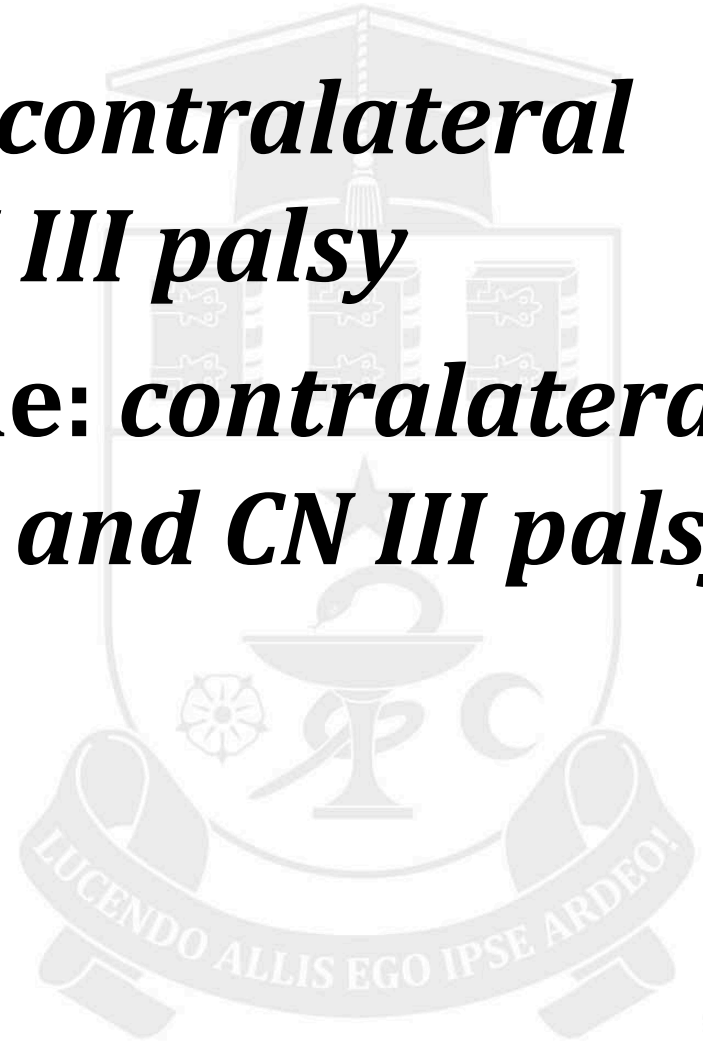




Penetrating branches of PCA

Weber Syndrome: *contralateral hemiplegia and CN III palsy*

Benedikt Syndrome: *contralateral ataxia or athetosis and CN III palsy*





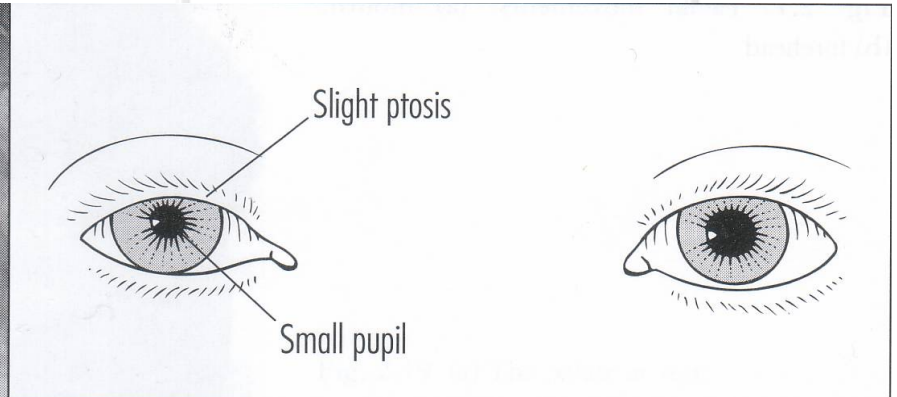
PICA

Wallenberg Syndrome: *Ipsilateral facial sensory loss, contralateral body sensory loss, vertigo, dysarthria, dysphagia, and Horner's syndrome*





Horner's syndrome

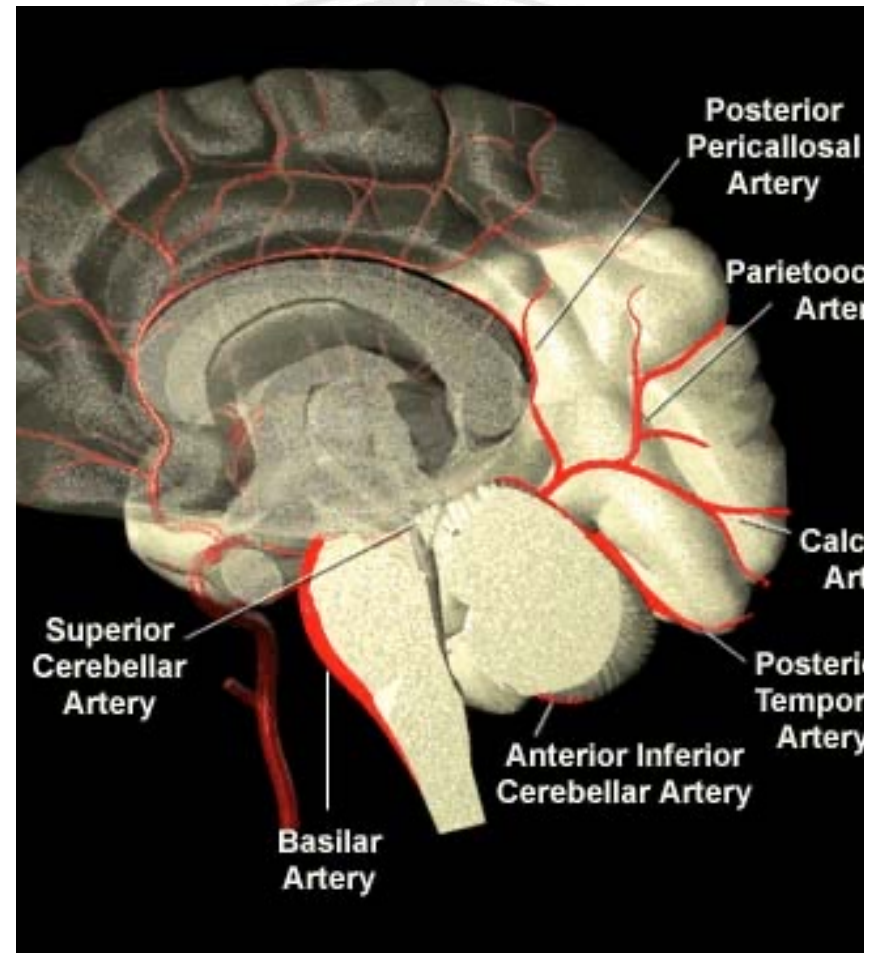


- ***Ptosis***
- ***Miosis***
- ***Anhidrosis***



POSTERIOR INFERIOR CEREBELLAR ARTERY syndrome (PICA)

- bears the responsibility for the blood supply to ~ 2/3 of the ipsilateral cerebellum.
- spontaneous rotary nystagmus, dysmetria and severe intentional tremor, rebound phenomenon.
- hemispheric cerebellar infarction occurs, life-threatening by dislocation and involvement of the brainstem





clinical features in DISECTIONS

- Local manifestations, by compression: pain in the anterior triangle of the neck (differential diagnosis with carotidodynia *), Horner syndrome, caudal cranial nerve lesions, nn. VIII and X.
- Remote embolic (frequent) and hemodynamic (rare) manifestations.
- They often heal without consequences, but permanent occlusions can occur.



Angiography. The ICA occlusion is straight for dissection.

Jae Hyuk Kwak, et al Neurointervention
2011;6:78-83

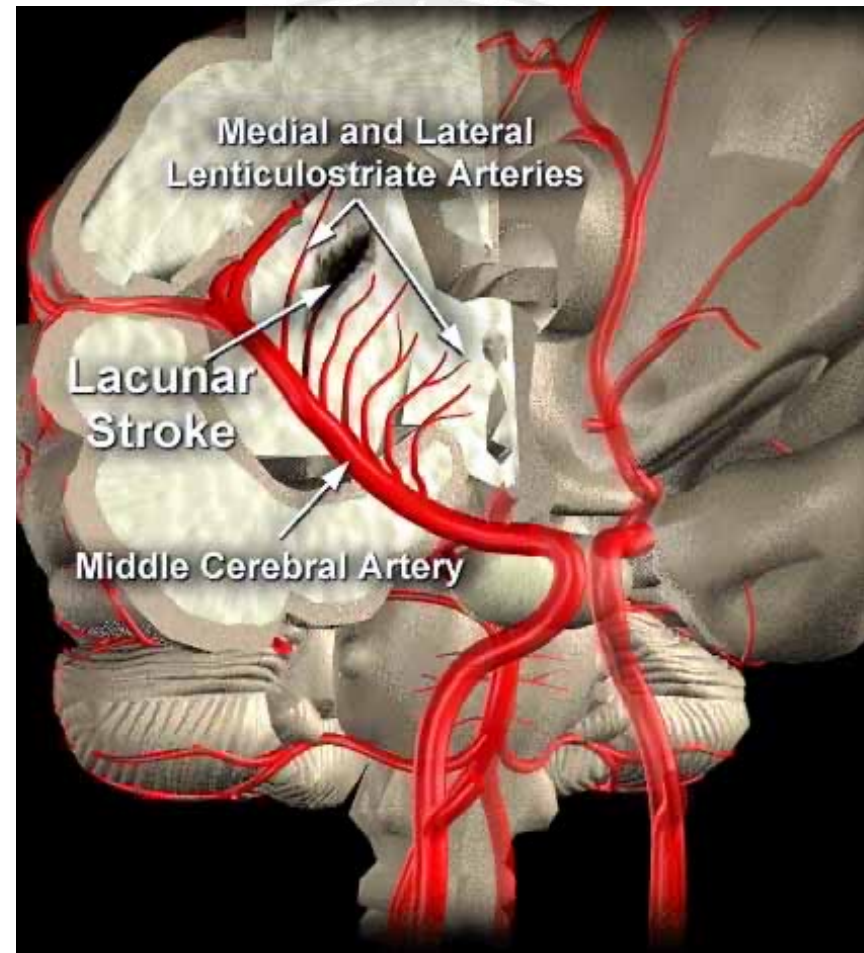


clinical manifestations in **LACUNAR INFARCTS**

Typical symptoms and syndromes:

- pure motor hemisymphomatics,
- pure sensory alternating symptoms
- atactic hemiparesis or dysarthria-clumsy-hand-syndrome (left-handed syndrome)

The diagnosis of clinically suspected lacunar infarction requires confirmation by CT or MRI investigation.





ISCHEMIC STROKE

7.

COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE



COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE

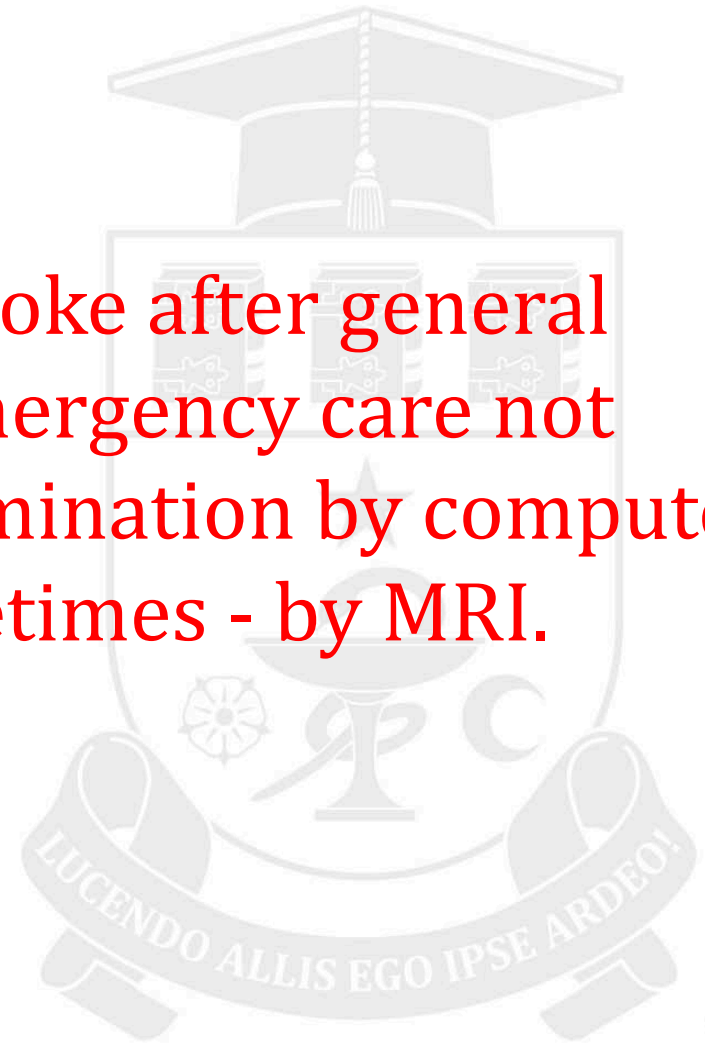
The most modest standards refer to:

- 24-hour and 7/7 CT access
- high-performance ultrasonographic diagnosis
- a standard clinical-biochemical laboratory (including for the diagnosis of CSF).



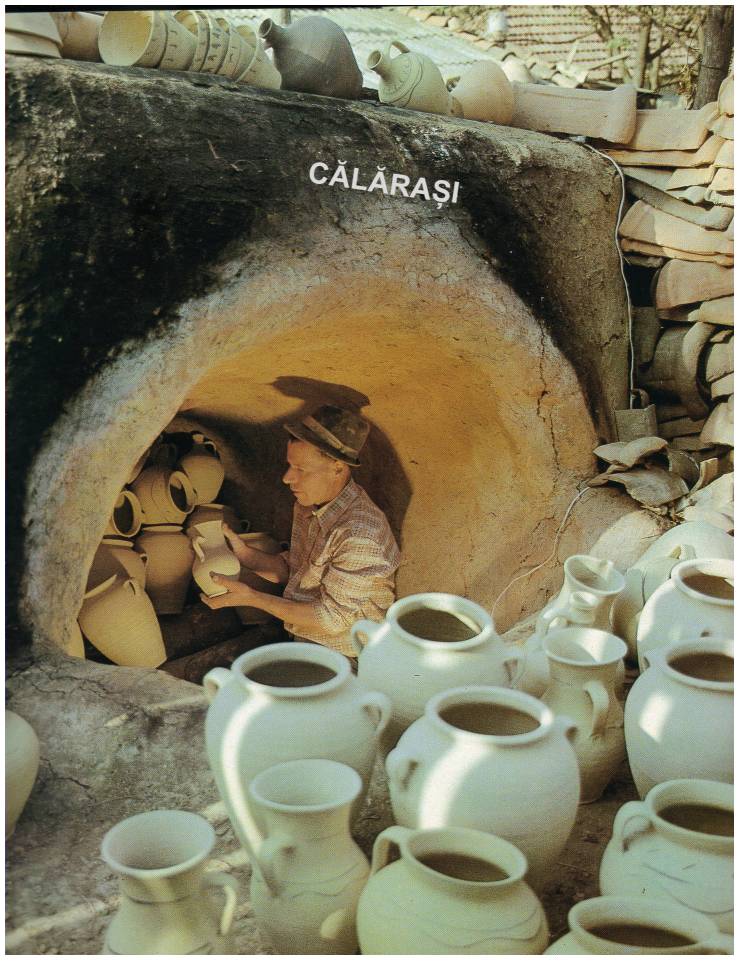
COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE

The patient with stroke after general stabilization and emergency care not postponed requires examination by computer tomography, sometimes - by MRI.





24-hour and 7/7 CT access





COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **COMPUTED TOMOGRAPHY**

- is the most important diagnostic examination in stroke (especially during the first 4.5 hours)
- allows diagnosis of intracranial hemorrhage
- frequently offers suggestions for early ischemic manifestations and a scar following an old infarct
- determines the location, mode, duration and extent of ischemia.



COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **COMPUTED TOMOGRAPHY**



hyperdense thrombus in the proximal portion of the MCA



COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **COMPUTED TOMOGRAPHY**



Manifestations of early cortical ischemia on the right island, as well as in the frontal operculum (arrow)



COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **COMPUTED TOMOGRAPHY**



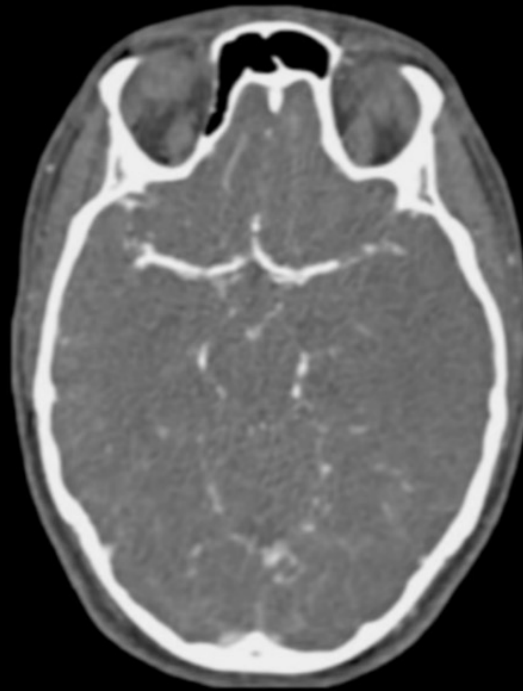
Very early signs of infarction with loss of differentiation between cortex and cerebral white matter in MCA territory on the right



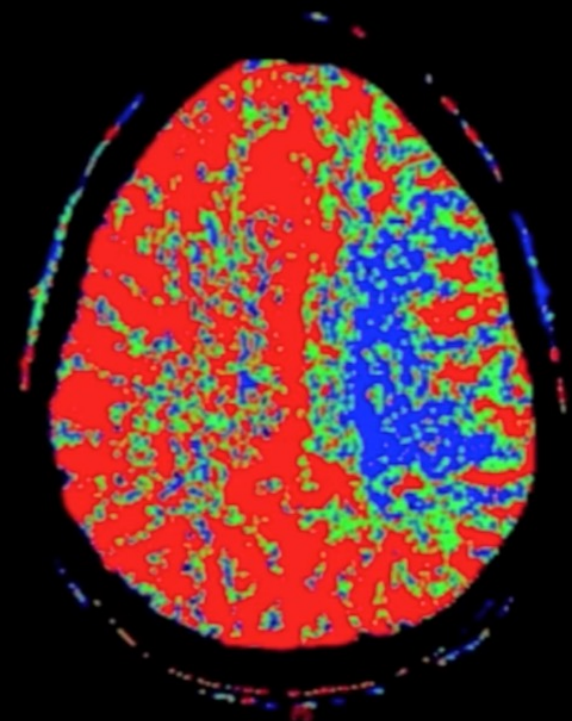
COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **COMPUTED TOMOGRAPHY**



Non-contrast



CT angiography

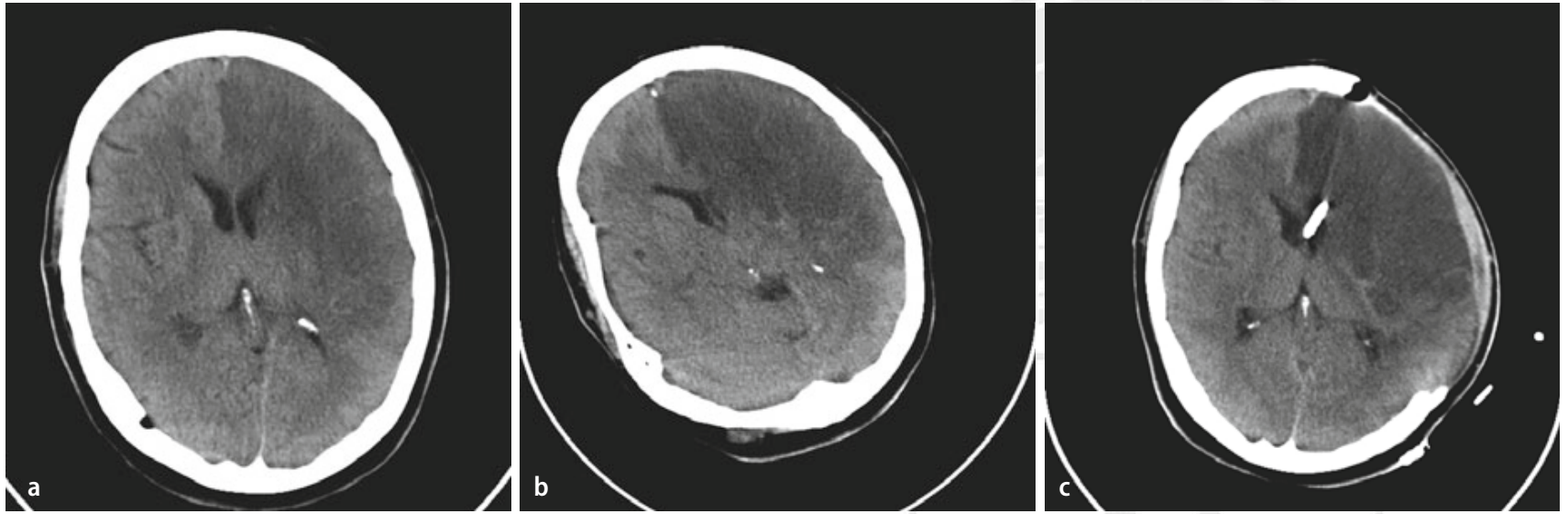


Perfusion

Initial manifestations of ischemic stroke in the left MCA territory

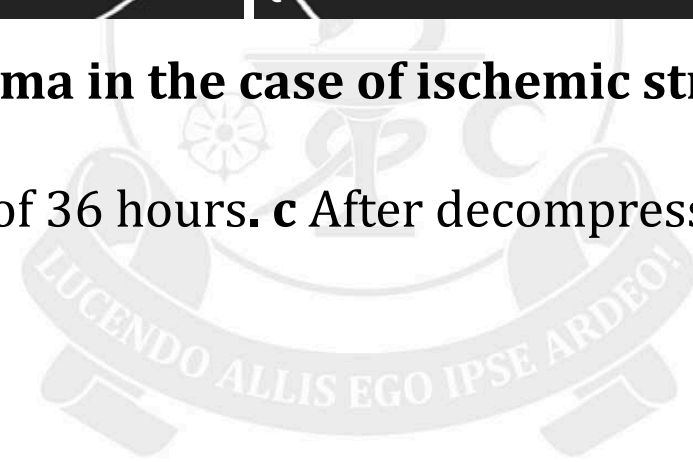


COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **COMPUTED TOMOGRAPHY**



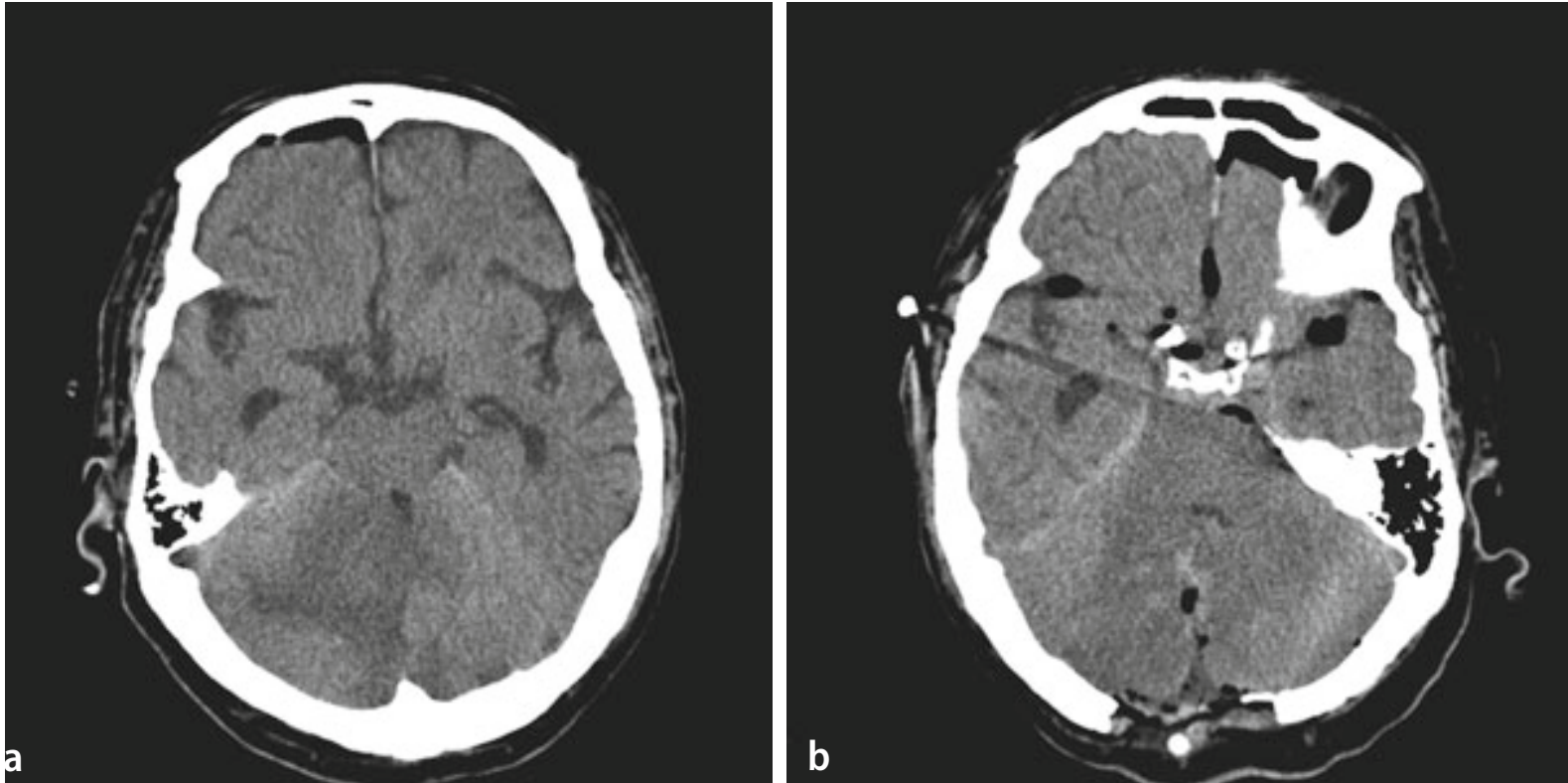
Progressive evolution of cerebral edema in the case of ischemic stroke.

Between both CT exams **(a, b)** is a term of 36 hours. **c** After decompression





COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **COMPUTED TOMOGRAPHY**



Space-substituting cerebellar infarction.

(a) Infarction in the territory of the posterior inferior cerebral artery on the right **(b)** After the decompression intervention



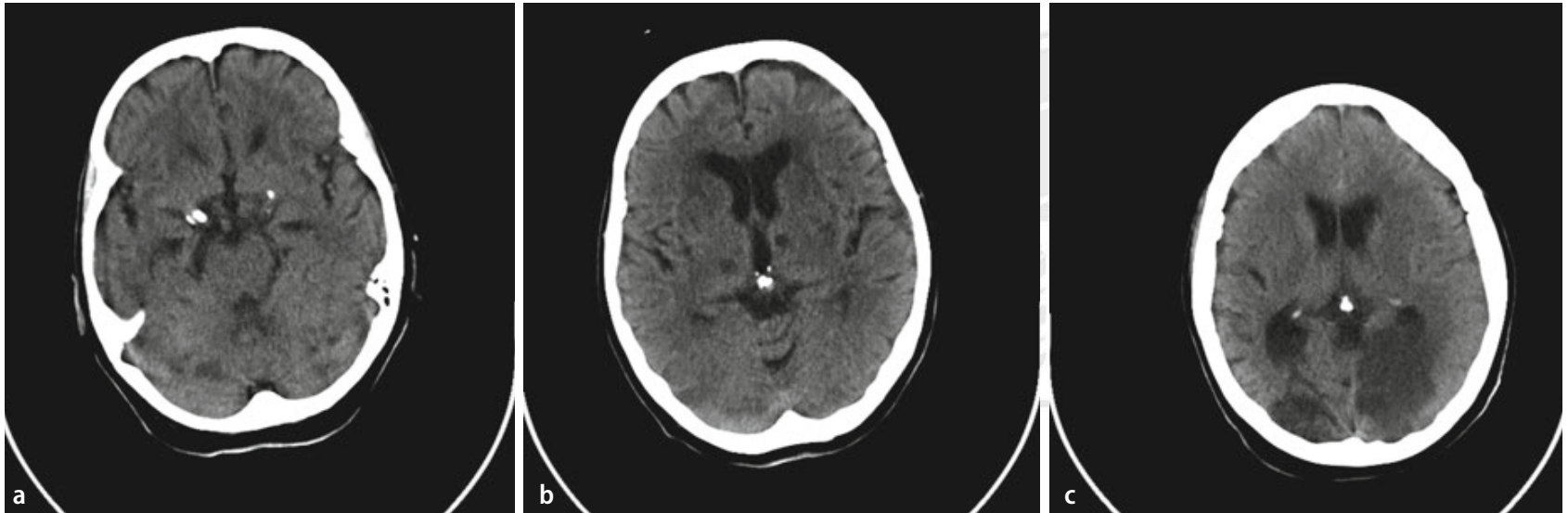
COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **COMPUTED TOMOGRAPHY**



Complete infarction in **MCA territory**, approximately three days with still pronounced replacement of space and midline deviation



COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **COMPUTED TOMOGRAPHY**



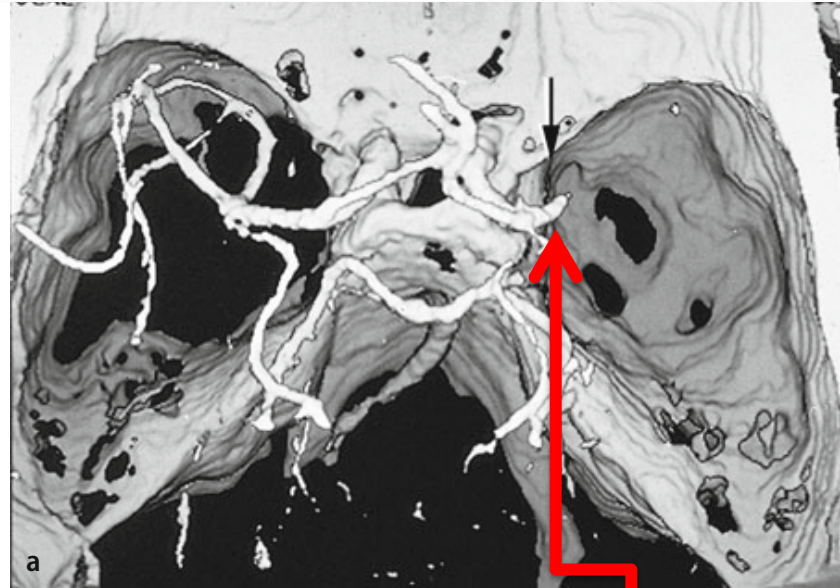
Example of lesion after basilar artery embolism.

a Bilateral cerebellar infarction, multiple infarctions caused by transient obstructions of different cerebellar arteries. **b** Bilateral thalamic infarction by obstruction of the perforating arteries to the thalamus at the level of the basilar apex and the proximal portion of the posterior cerebral arteries. **c** Extended bilateral infarction in the posterior circulation caused by obstruction of both posterior cerebral arteries.

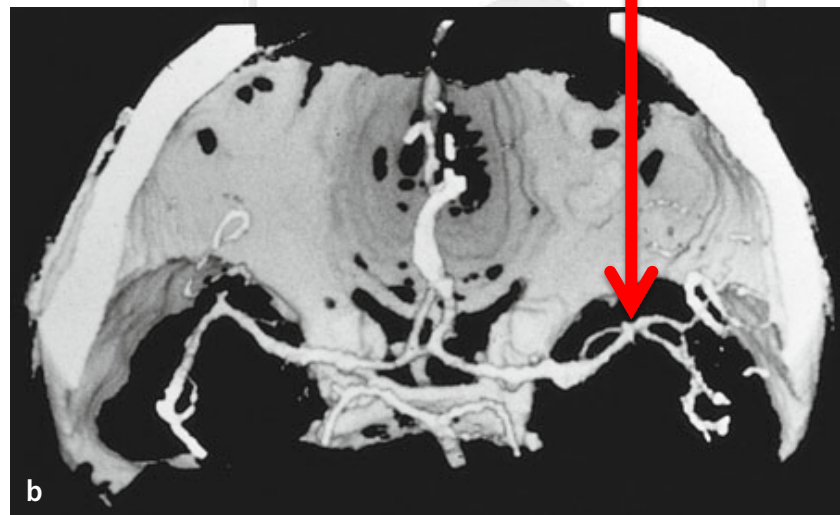


COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **CT-angiography**

a CT-angiography in 3D reconstruction with proximal closure of left MCA. It is observed the truncation of the vessel (arrow)

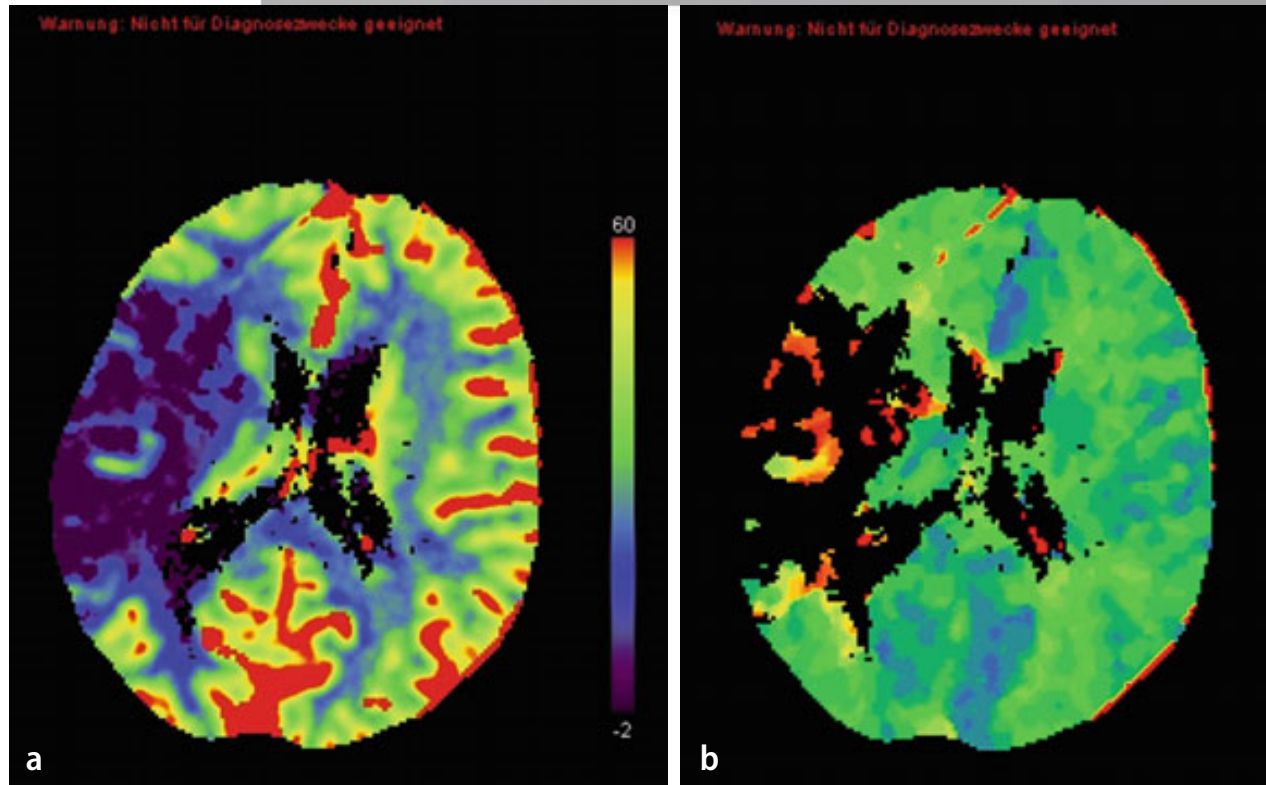


b After thrombolysis the reperfusion of the blocked vessel occurs (R. Von Kummer, Dresden)





COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **CT-perfusion**

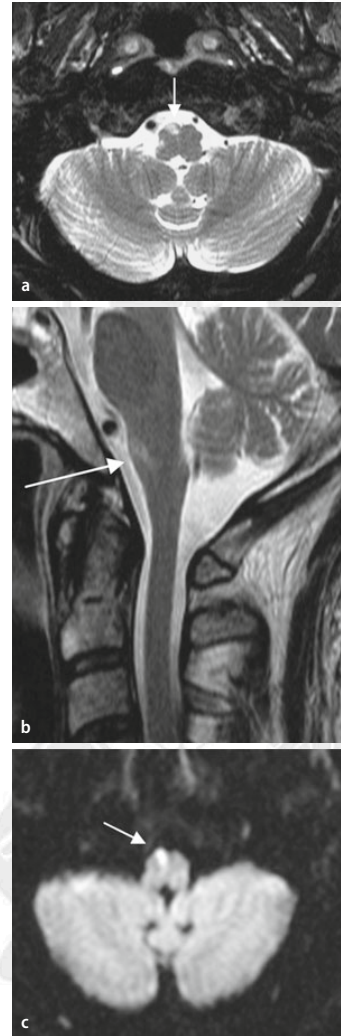


CT-perfusion. Presentation of blood volume (a) and Time to Peak (b) in case of carotid occlusion on the right.



COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **Magnetic Resonance Imaging**

- it is the first choice examination in the case of stroke of brainstem
- will be performed in cases where the therapeutic decision depends on it
- **Contraindications:**
 - the pacemaker
 - the phobias
 - metal implants of unknown material and terms
 - disturbances of vital functions that require supervision and emergency help



MRI

presentation of minor infarction in the *medulla oblongata* in axial (**a**), sagittal (**b**), T2-weighted and diffusion (**c**). The infarction areas are indicated by arrows.



COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **MRI-angiography**



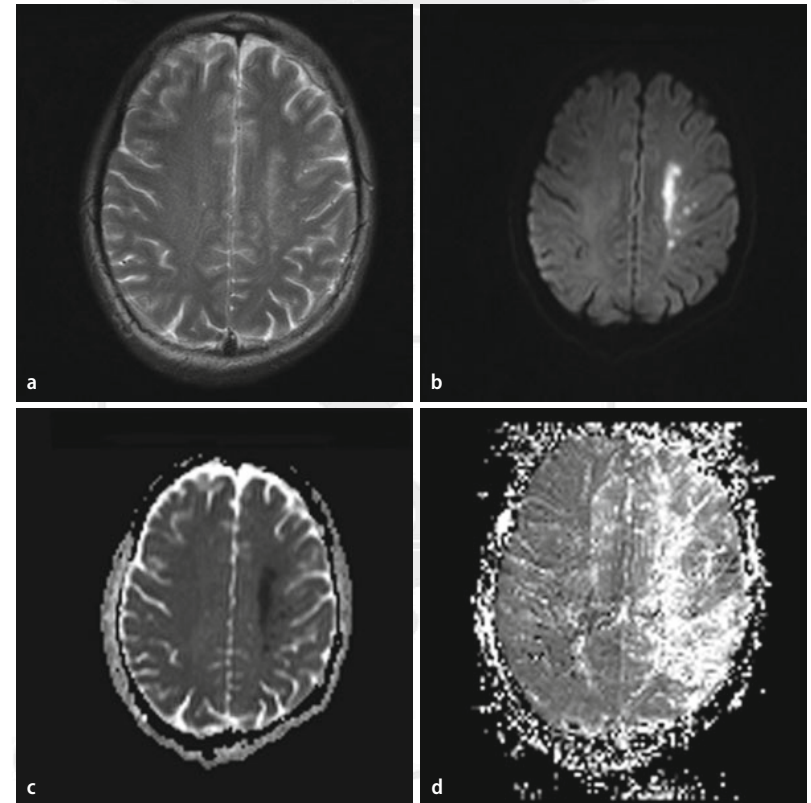
Time-of-flight-angiography from the level of the aortic arch and arteries providing blood to the brain till the level of Willisii artery polygon. Normal version.

- a non-aggressive, non-invasive method used in patients who do not wish to be examined by conventional angiography. It is also suitable for the search of large aneurysms or in case of suspected sinus and vein thrombosis (MRI-venogram).



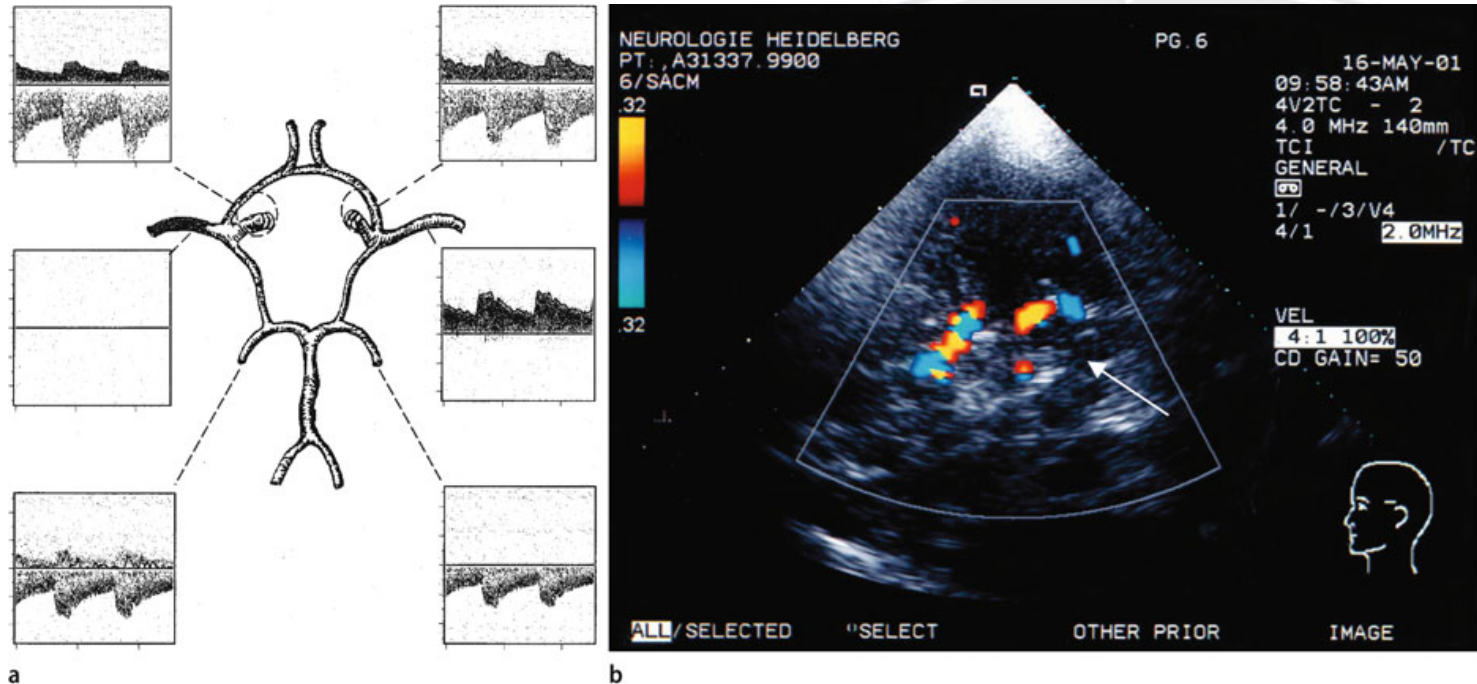
COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **MRI-diffusion and MRI-perfusion**

- It is possible to visualize the areas of early ischemia, as well as to help to differentiate the penumbra from the definitive infarction.
- At present, there are several clinical studies in which the value of thrombolysis based on MRI examination will be estimated ("tissue window" instead of "therapeutic window").





COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **Ultrasonography**



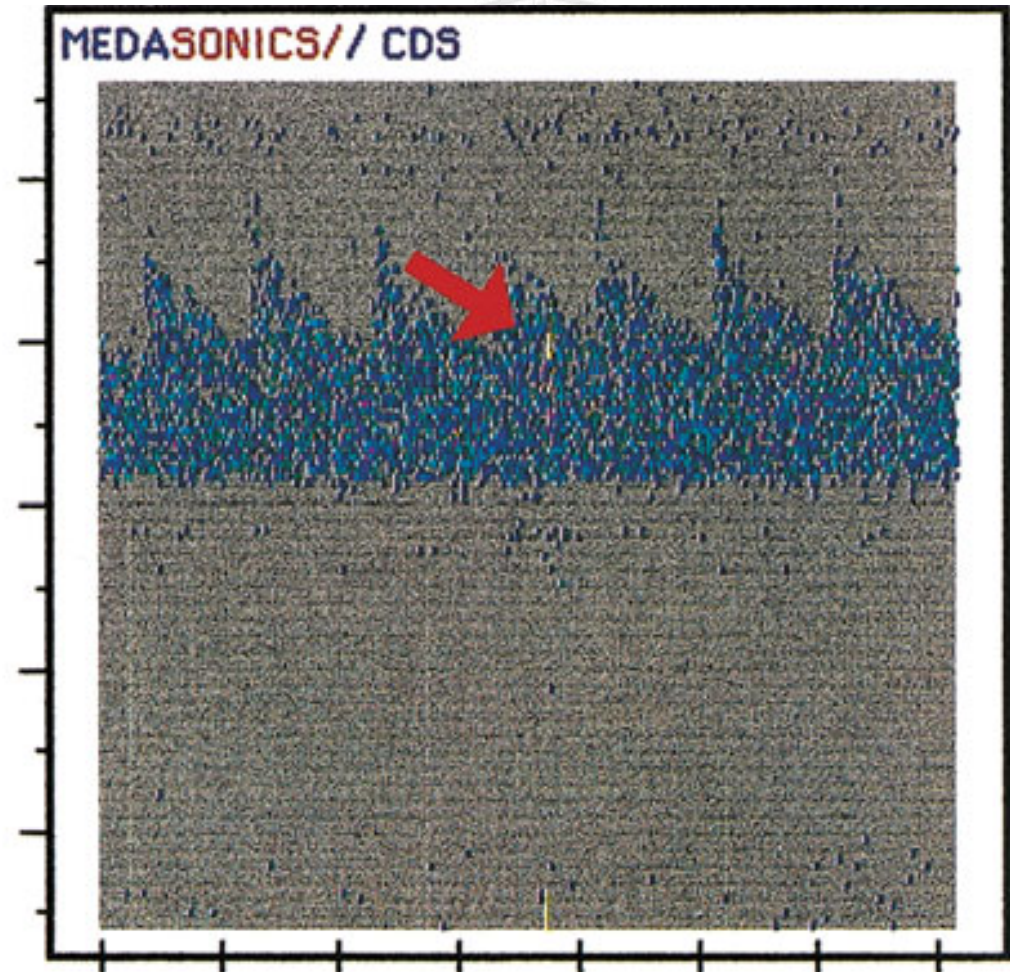
Ultrasound vascular diagnosis.

A. Transcranial Doppler ultrasonography in acute left middle cerebral artery occlusion. **B.** Doppler duplex ultrasonography presentation of a left middle cerebral artery obstruction (arrow).



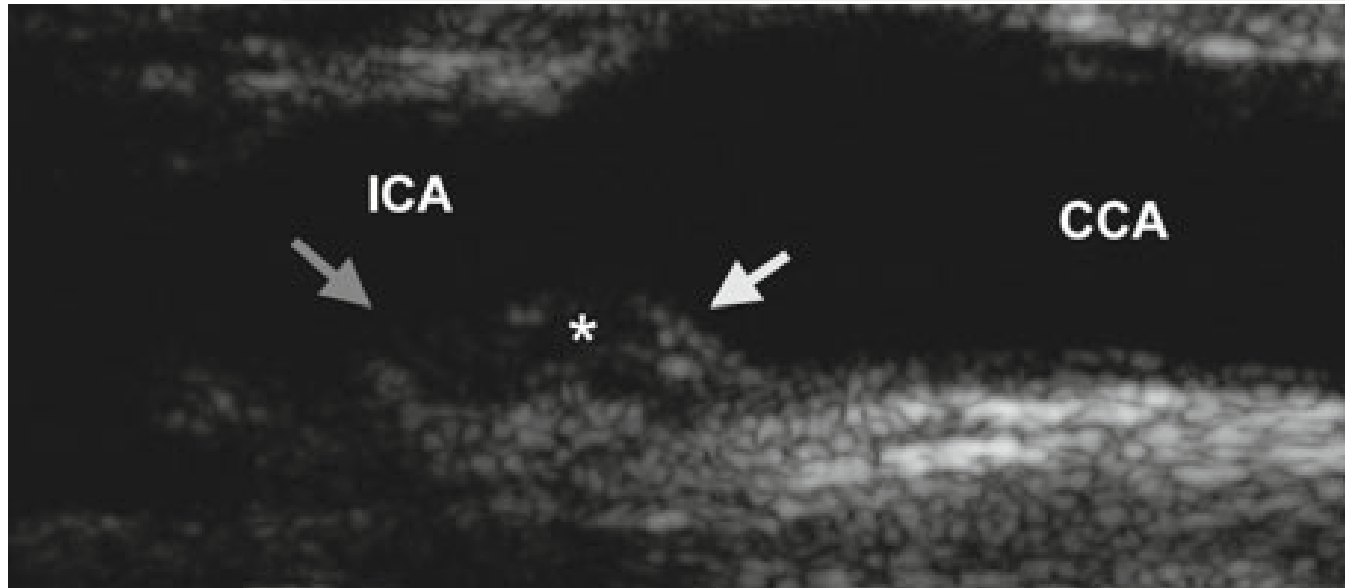
COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE : **Ultrasonography**

Detection of emboli by transcranial dopplerography "High intensity transient signal" (arrow "HITS") (With permission of R. Winter, Heidelberg)





COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE: **Ultrasonography**



The structure of the plaque in B-image. Heterogeneous plaque (arrows) at the bifurcation level. The fibrous delimiting layer produces a harder echo signal proximally (white arrow) than distal (arrow). The plaque shows an upper surface of soft consistency, which reaches the lumen of the vessel. Underneath the top layer of the plaque is made an area with poor echo signal (*), which corresponds to lipid storage. (With permission of S. Meairs, Mannheim).



COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE: **Digital subtraction angiography**

Indications for angiography:

- before or during the intervention procedures,
- suspicion of pseudo-aneurysm after dissection,
- intracranial vascular stenosis,
- suspicion of pseudoocclusion,
- suspicion of vasculitis.

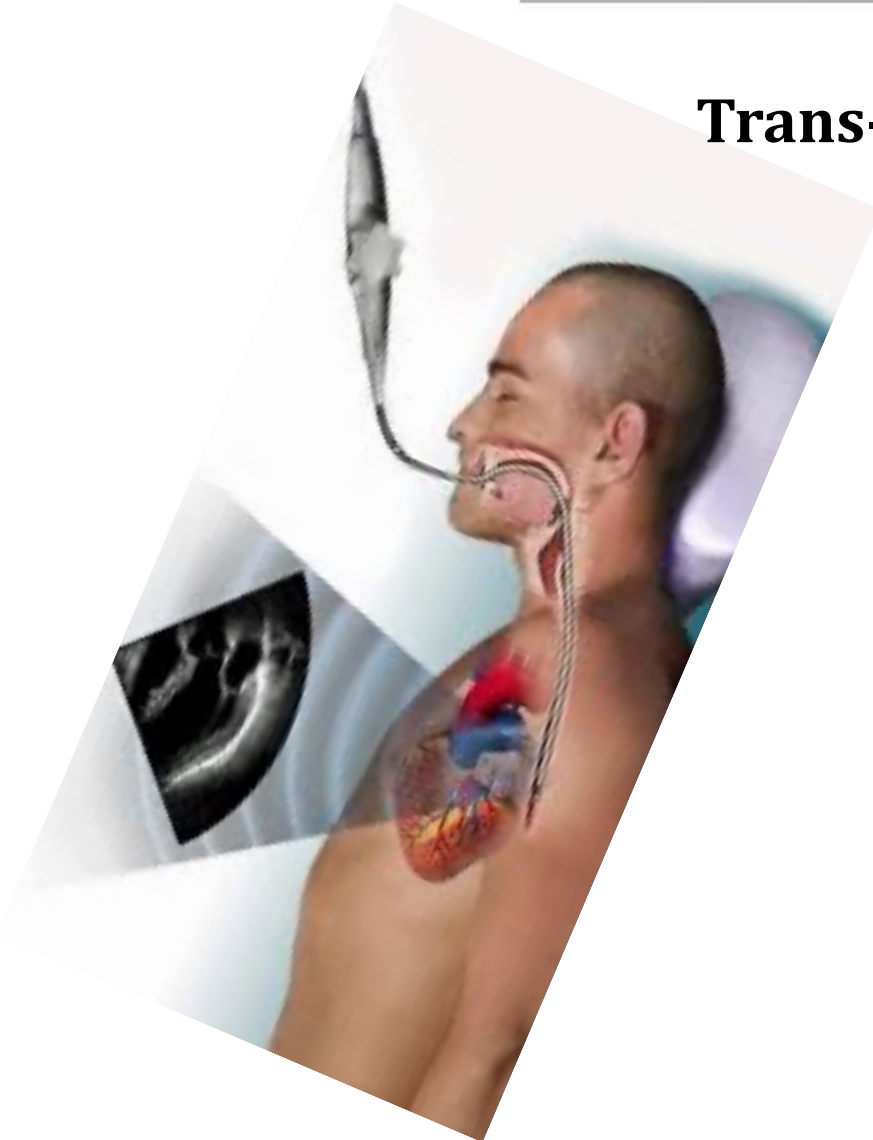




COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE: complementary examination of the heart

Trans-esophageal echocardiography

- will be performed when the suspicion of cardiogenic embolism remains after previous examinations.
- it has significant advantages in detecting intracardiac thrombi and atrial septal changes (*foramen ovale patent*, atrial septal aneurysm), as well as atherosclerotic changes in the aortic arch.





COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE: **laboratory exams**

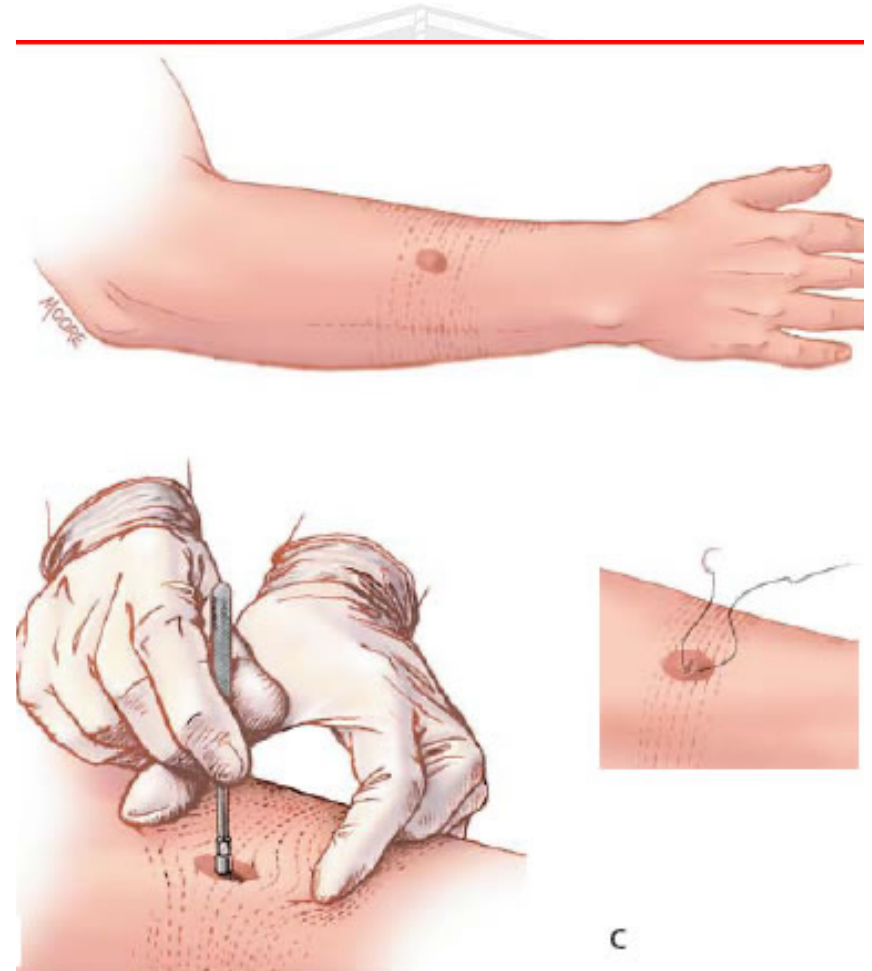
- identification of general risk factors for atherosclerotic disease
- examination of the function of other organs and certification of rare etiologies of stroke (vasculitis, coagulopathies, etc.)
- the special analysis of the CSF will be performed only in the case of suspicion of vasculitis.





COMPLEMENTARY DIAGNOSIS OF ISCHEMIC STROKE: **biopsy**

- **Vessels and muscle biopsies** will be performed in case of suspicion in vasculitis.
- **Skin biopsies are performed in case of:**
 - suspicion in CADASIL - a genetically determined microangiopathy
 - suspicion in mitochondriopathies (MELAS)
 - suspicion in genetic diseases (Notch-3 mutations).





ISCHEMIC STROKE

7.

TREATMENT of ISCHEMIC STROKE



STROKE

preclinical treatment

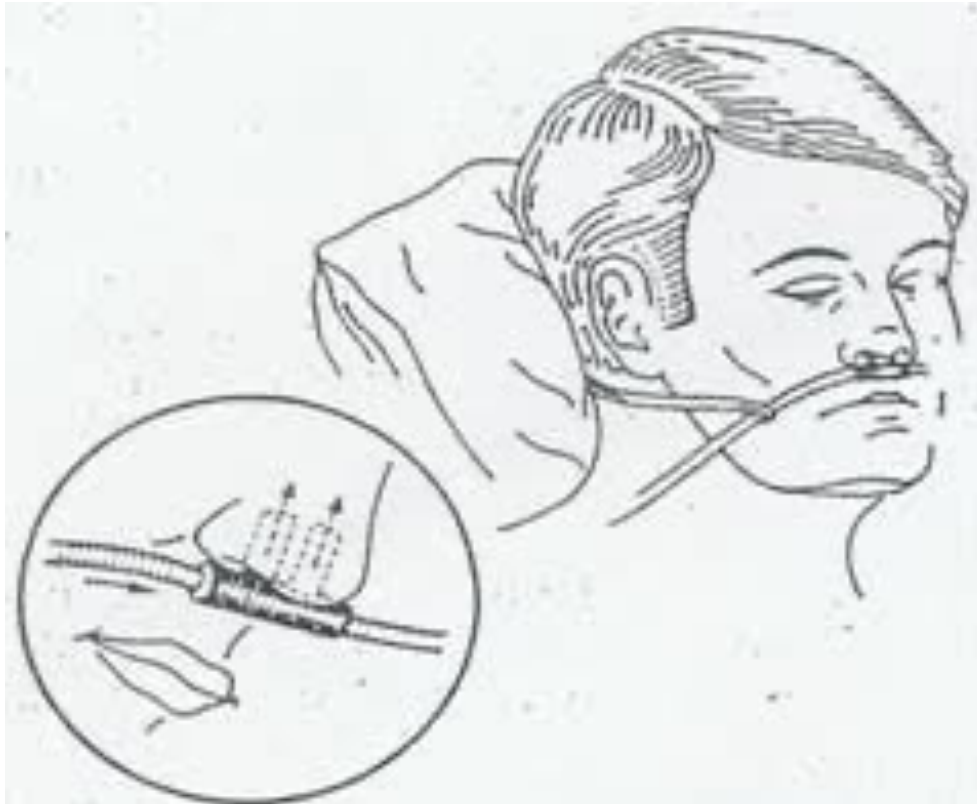
- Stabilization and normalization of general body functions (cardio-circulatory, pulmonary, hydroelectrolytic, metabolic)
- transport to a clinic specialized in diagnosis and treatment (stroke unite)





STROKE

general treatment: OXYGENATION



- Respiratory failure in the first hours is relatively rare (large hemispheric or vertebro-basilar infarctions, large intracerebral haemorrhages, severe subarachnoid haemorrhages or seizures)
- There is a risk of aspiration
- The patient with a medium / severe acute stroke will be provided with oxygen through nasal catheter (2-4 l / min).



STROKE

general treatment: BLOOD PRESSURE



- it will be permanently monitored and will be reduced only when its values exceed **220/110 mmHg** or if thrombolysis treatment is undertaken (**Decreasing T.A. in other cases in the stroke patient can be dangerous!**)
- in the case of hemodynamic infarcts, it is necessary to increase the blood pressure by hyperoncotic or drug infusions



STROKE

general treatment: BLOOD PRESSURE

CIRCUMSTANCUYI	TREATMENT
Systolic A.T. 180-220 mmHg and/or diastolic A.T.105-120 mmHg	no need to treat!
Systolic A.T. ≥ 220 mmHg and/or diastolic A.T. 105-120-140 mmHg	Urapidil 10-50 mg i.v. then 4-8 mg/h i.v.* Captopril 6,25-12,5 mg p.o/i.m. Clonidin 0,15-0,3 mg i.v./s.c. Dihydralazin 5 mg i.v. plus Metoprolol 10 mg
Diastolic A.T. ≥ 140 mmHg	Nitroglycerină 5 mg i.v., then de 1-4 mg/h i.v. Natriumnitroprussid 1-2 mg



ISCHEMIC STROKE

general treatment: BLOOD GLUCOSE

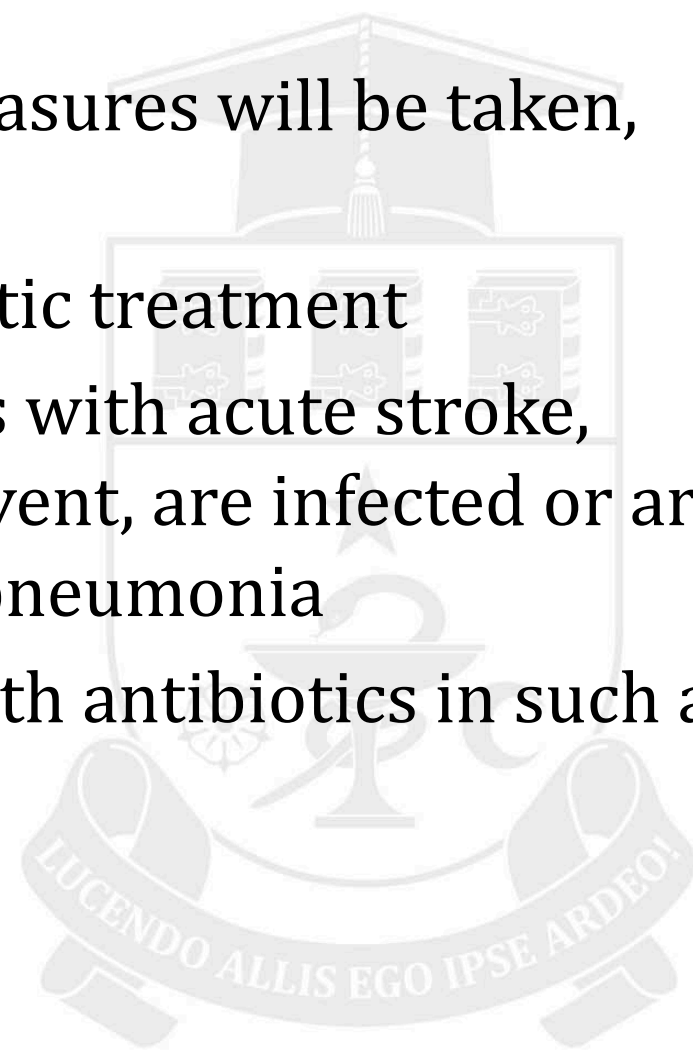
- Is not recommended sugar level more than **150 mg/dl (8.3 mmol/l)**.
- Values more than **200 mg/dl (11.1 mmol/l)** are treated by subcutaneous, less often intravenous, short-acting insulin.
- In case of hypoglycemia (at the first test) immediately i/v infusion of *Sol. Glucose 10%* will be done.
- Except this circumstance in the first days after a stroke, glucose-containing infusions will be avoided.



ISCHEMIC STROKE

general treatment: **INFECTIONS** and **HYPERTERMIA**

- at $t > 37.5^{\circ}\text{C}$ corrective measures will be taken, including physical cooling
- only well-targeted antibiotic treatment
- a large number of patients with acute stroke, already before to stroke event, are infected or are at high risk of aspiration pneumonia
- prophylactic treatment with antibiotics in such a situation is not effective.





ISCHEMIC STROKE

general treatment: PROFILAXY OF TROMBOSES

- high risk of thrombosis and pulmonary embolism
- subcutaneous administration of low molecular weight heparin (certoparin or enoxaparin) 2000-5000 IU s.c. anti-Xa-action
- concomitant for the early prophylaxis of thrombosis intense curative gymnastics will be instituted

Elastic socks and other mechanical, pneumatic compression systems have no action or at least one very light expressed in the prevention of vein thrombosis of the lower limbs.



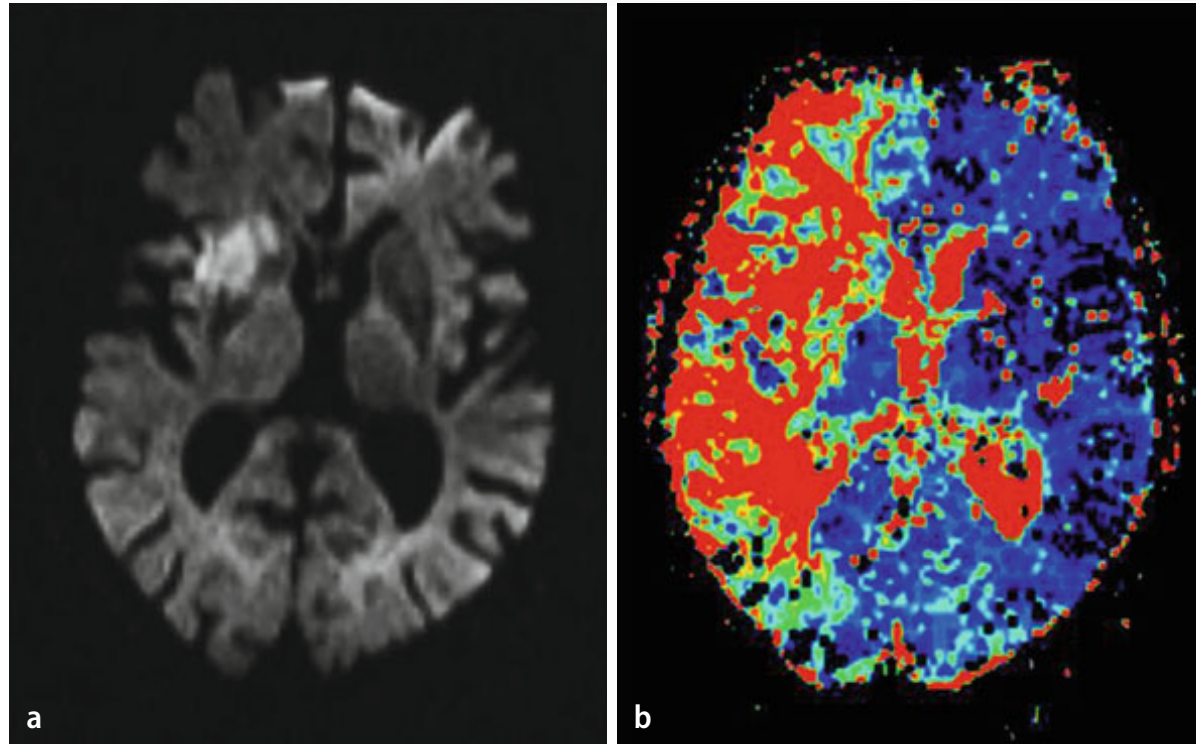
treatment to improve perfusion **(RECANALIZATION – THROMBOLYSIS)**

- Systemic (intravenous) thrombolysis with rtPA (0.9 mg / kg weight; maximum dose being 90 mg, 10% of the total dose is given in bolus, the remaining 90% as an infusion for 60 minutes) in the therapeutic window up to 4, 5 hours after the onset of clinical manifestations; **highest efficiency - the first 90 minutes!**
- Correct interpretation of the early CT exam!
- CT-angiography: endovascular therapy?
- Contraindications for thrombolysis: haemorrhage, severe clinical signs with consciousness obstruction; early signs of infarction of large proportions (relative contraindication)



ISCHEMIC STROKE

treatment to improve perfusion **(RECANALIZATION – THROMBECTOMY)**

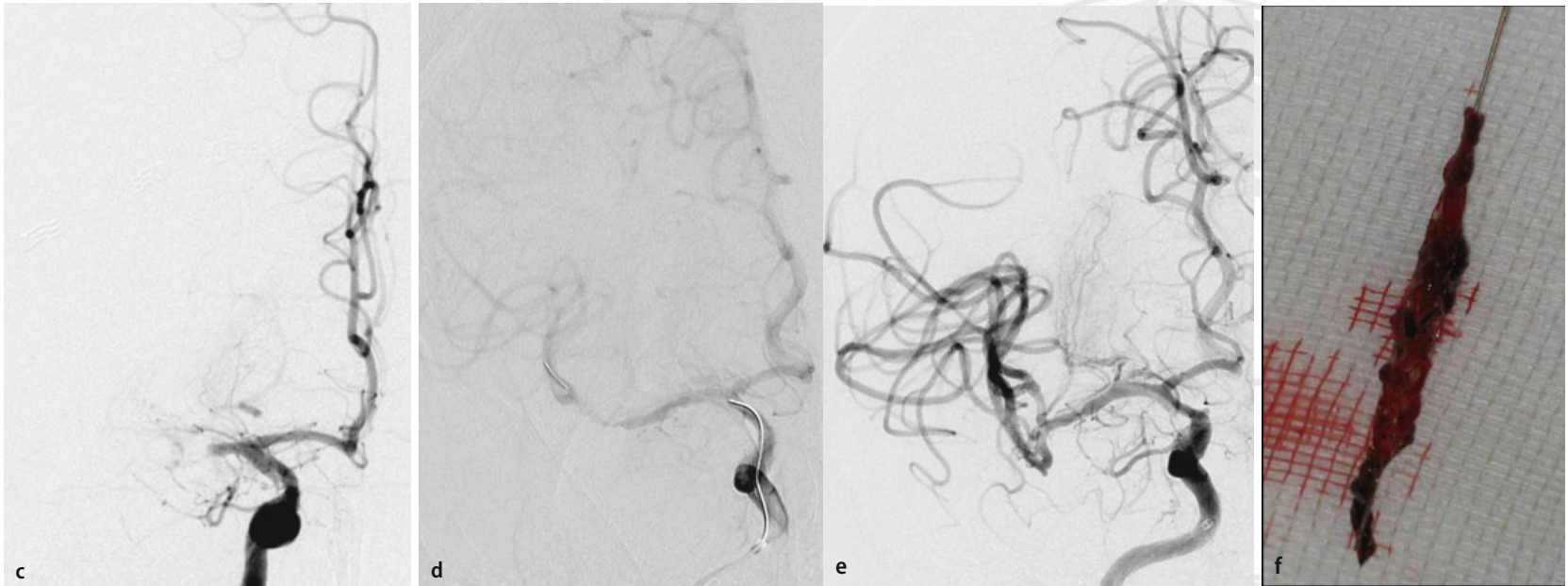


Diffusion restriction in the left basal nuclei (a) with an extensive perfusion disorder in the left hemisphere (b) in a patient 3 hours after the onset of stroke in the left hemisphere.

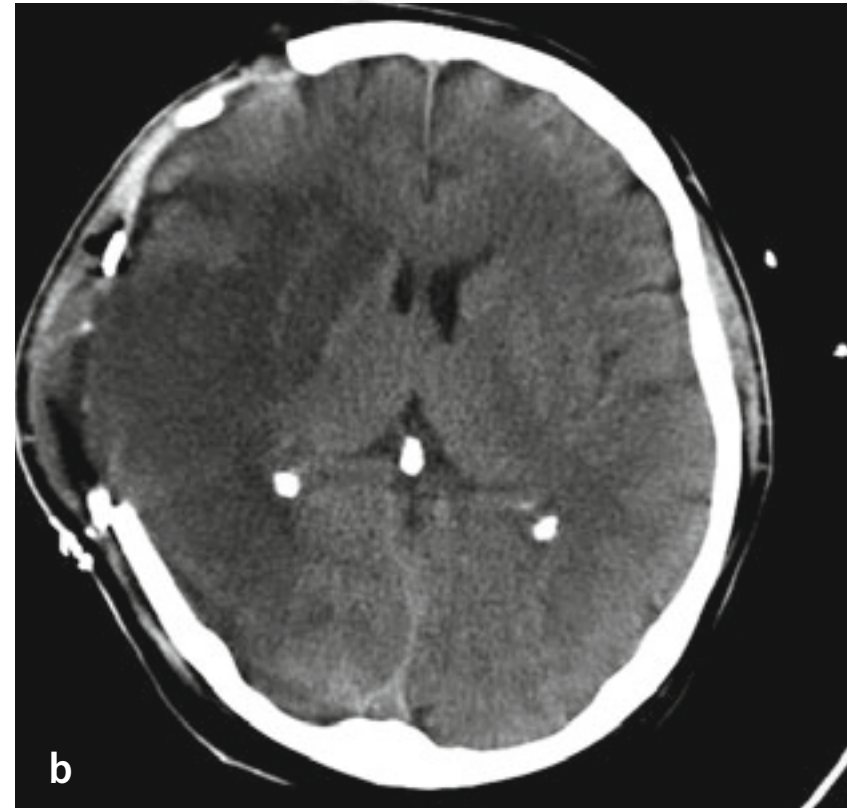
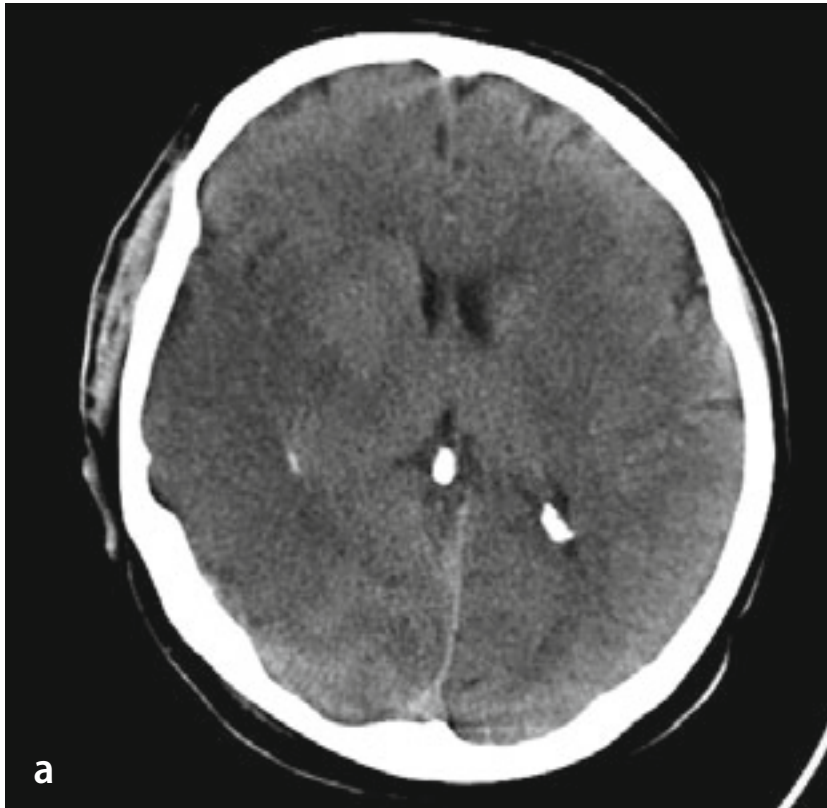


ISCHEMIC STROKE

treatment to improve perfusion **(RECANALIZATION – THROMBECTOMY)**



c angiography demonstrates the obstruction of the main trunk of the left middle cerebral artery. **d** The blocked sector is penetrated through a microcatheter, inside the thrombus a Stent-Retriever is opened, thus creating a channel of blood flow penetration. **e** The system is withdrawn by suction, thus obtaining a full recanalization of the vessel. **f** A Stent-Retriever system with the thrombus removed.

**treatment: DECOMPRESSION****(malignant infarction + cerebellum)**

MCA malignant infarction until (a) and after decompression (b)
should be performed even earlier than 48 hours after the onset of symptoms, usually within the first 24 hours.



REHABILITATION TREATMENT

- 1. Physiotherapy (gymnastics)***
- 2. Speech therapy***
- 3. Kinesitherapy (ergotherapy)***

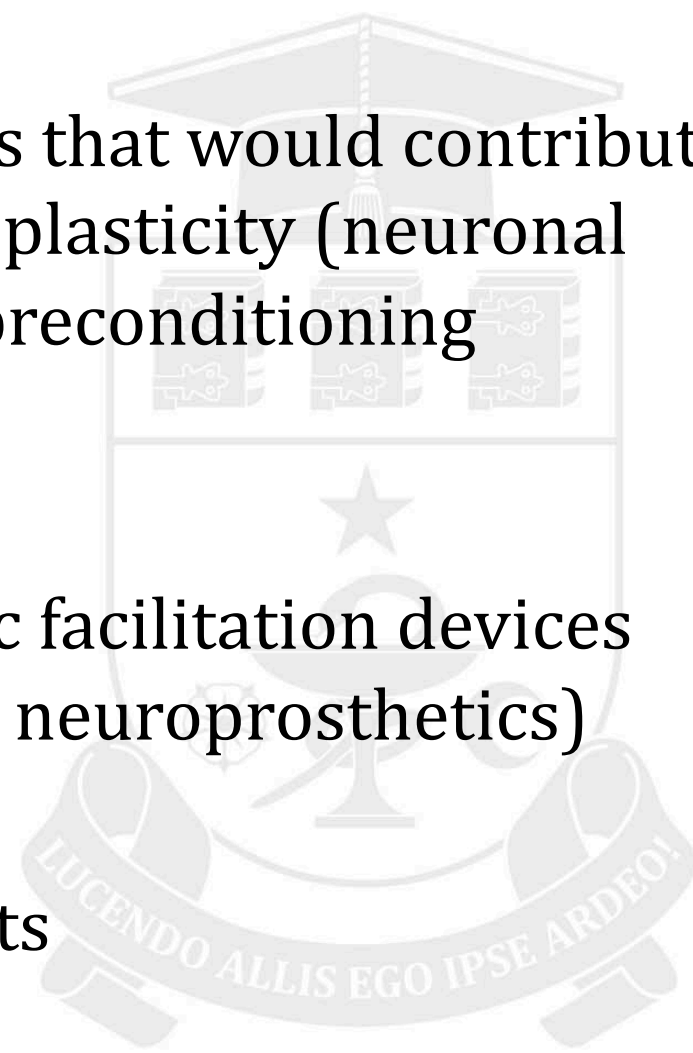




- is started on the day of disease onset
- complex treatment: medicine, kinetotherapeutic and speech therapy
- initiation / continuation of secondary stroke prophylaxis
- education of the healthy style of life



- techniques and substances that would contribute to brain regeneration and plasticity (neuronal growth factors, ischemic preconditioning phenomenon)
- mechanical and cybernetic facilitation devices (communication facilities, neuroprosthetics)
- functionally adapted robots





ALGORITHM of diagnosis and treatment of the patient with ischemic stroke

Recognition + Call emergency service



Prehospital Support + Transportation + Phone Advert



STROKE Unite



Imagistical Examination(CT, MRI, Angio-CT/MRI)



TREATMENT:
general + recanalization + rehabilitation + secondary profilaxy



ISCHEMIC STROKE

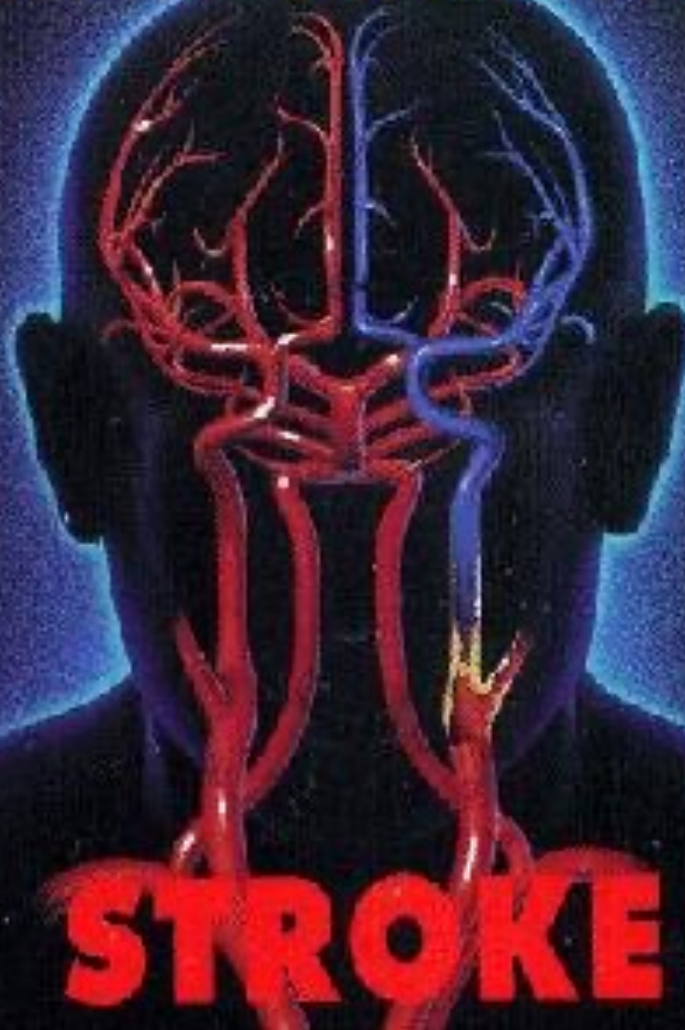
7.

Ischemic stroke prophylaxis



BRAIN ATTACK

YOU CAN PREVENT IT!



STROKE





ZIUA MONDIALĂ
A CREIERULUI
22 iulie 2017



Accidentul Vascular Cerebral: previne-l și tratează-l

90% din atacurile cerebrale
pot fi prevenite, dacă controlați:



Hipertensiunea
arterială



Nivelul de zahăr
și colesterol



Activitatea fizică



Obezitatea



Aritmiile cardiace



Alcoolul



Fumatul



Regimul alimentar

RECUNOAȘTE ATACUL CEREBRAL ȘI SOLICITĂ IMEDIAT AMBULANȚA!



Durere
de cap



Vedere
neclară



Vorbire
neclară



Pierderea
echilibrului



Slăbiciune,
amorțeală
în mână
sau picior



Pierderea
conștienței



Ischemic stroke prophylaxis

Primary prophylaxis

- **determining and treating the risk factors that can contribute to the onset of stroke in one individual patient**

Secondary prophylaxis

measures for the prevention of stroke, than ischemic stroke of any severity (TIA →→→→ light, middle, severe) has already happened

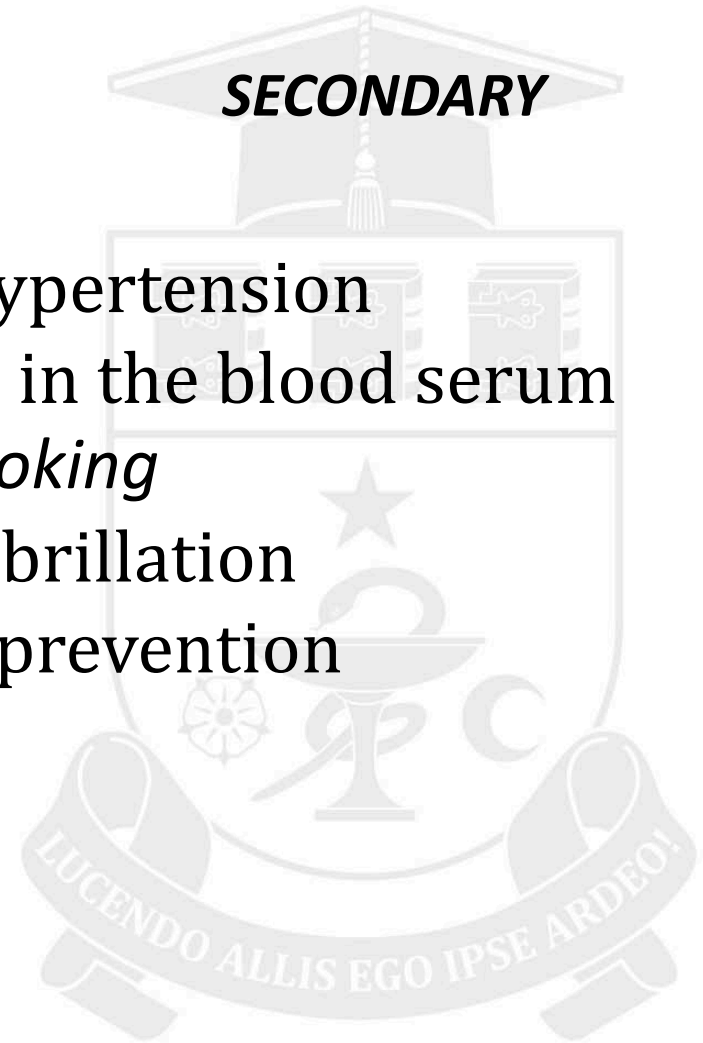


Ischemic stroke prophylaxis

PRIMARY

- Arterial hypertension
- The level of lipids in the blood serum
 - *Smoking*
 - Atrial fibrillation
 - Invasive prevention

SECONDARY





Ischemic stroke prophylaxis

Arterial hypertension

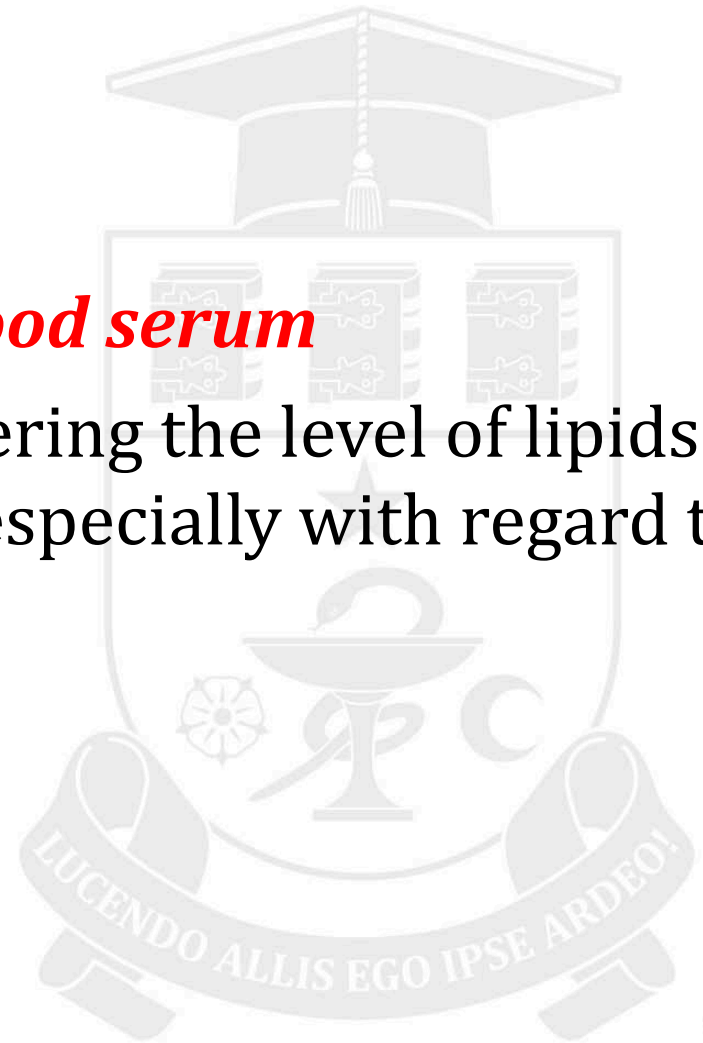
- Treatment of high blood pressure reduces stroke frequency by $\sim 40\%$
- $\downarrow \text{NaCl} + \downarrow \text{body weight} + \uparrow \text{sport} - \text{alcohol} = = =$
Normal systolic A.T. (efficient and durable!)
- Antihypertensive drugs may adversely affect the metabolic profile (eg beta-blockers and thiazide diuretics)



Ischemic stroke prophylaxis

The level of lipids in the blood serum

The preventive effect of lowering the level of lipids in the blood has been proven, especially with regard to cardiovascular morbidity.

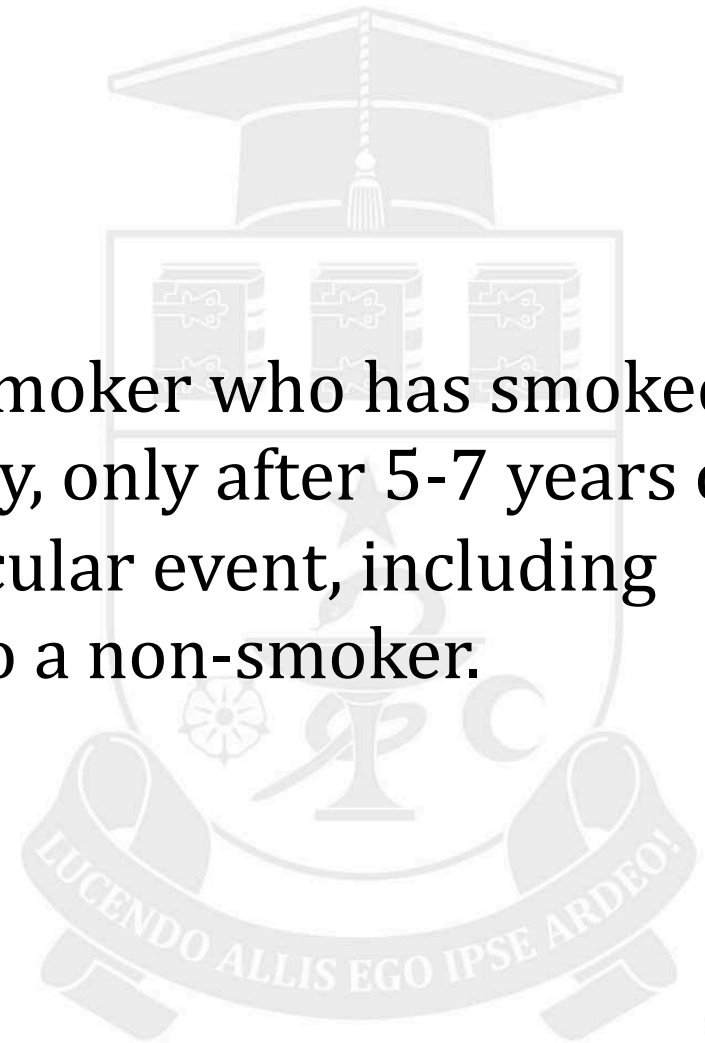




Ischemic stroke prophylaxis

Smoking

- In the case of a cigarette smoker who has smoked 20 or more cigarettes a day, only after 5-7 years of abstinence the risk of vascular event, including ischemic stroke, returns to a non-smoker.





Ischemic stroke prophylaxis

Atrial fibrillation

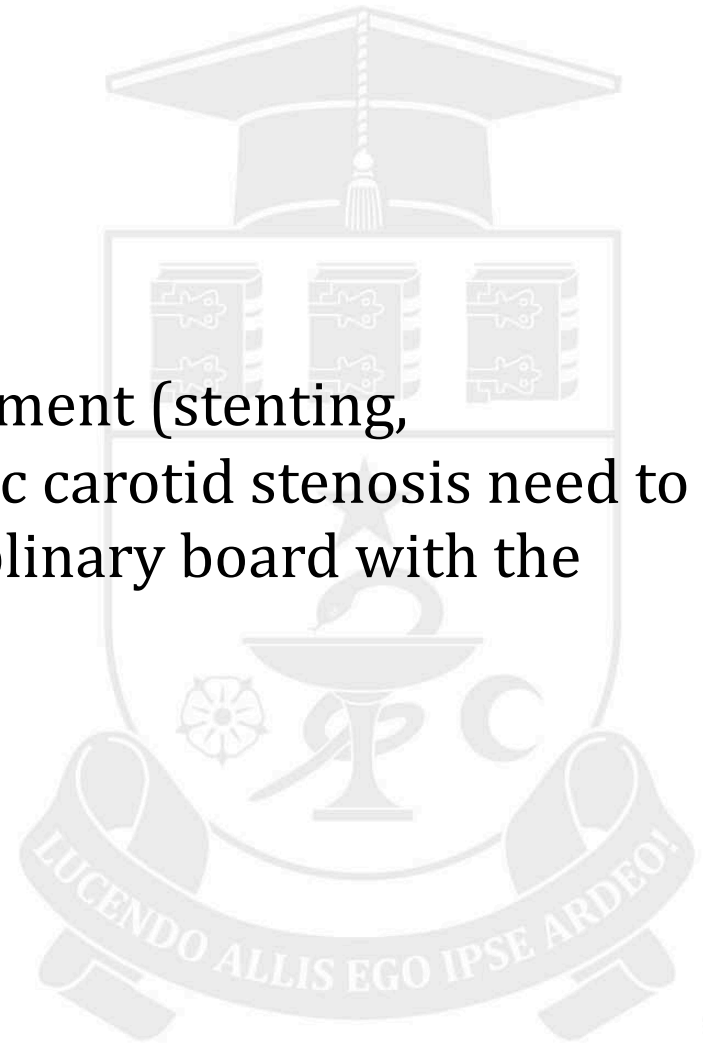
- The untreated AF carries an annual risk for a stroke of 3-15% depending on the risk factors.
- At long-term anticoagulation with vitamin K antagonists (VKA) the risk can be reduced by approximately 60-80% (target INR 2-3).
- New direct oral anticoagulants (dabigatran rivaroxaban, apixaban, edoxaban) have a significantly lower risk of intracranial hemorrhage.
- Anti-platelets are essentially less effective



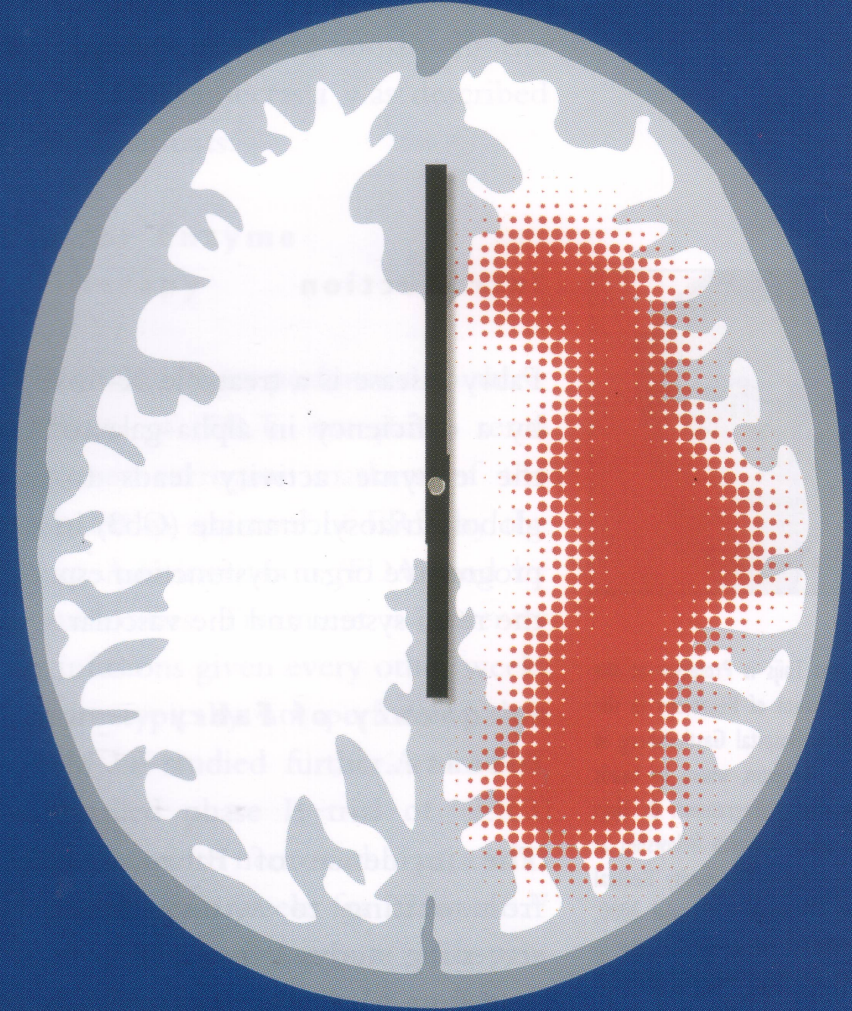
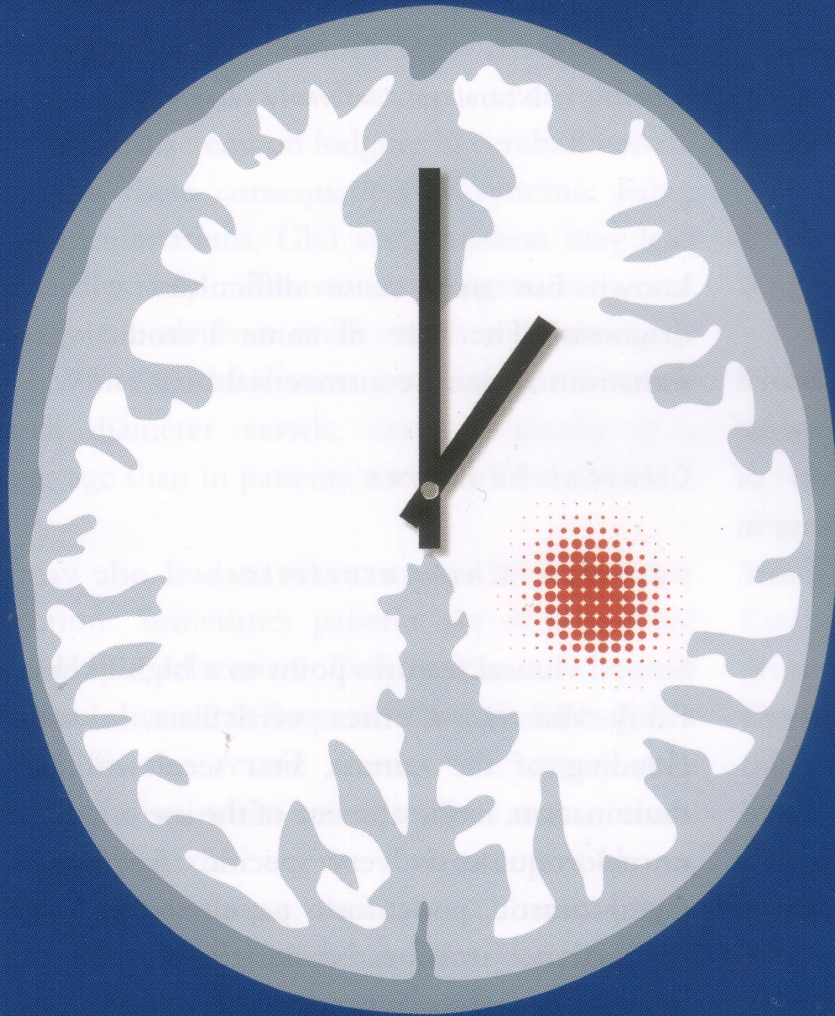
Ischemic stroke prophylaxis

Primary invasive prevention

The indications for invasive treatment (stenting, endarterectomy) of asymptomatic carotid stenosis need to be determined only in an interdisciplinary board with the participation of neurologists.



TIME MEANS BRAIN



A close-up, caricature-style illustration of a man's face. He has a wide-eyed, surprised expression, with his eyes looking upwards and to the sides. He is wearing round glasses. His mouth is replaced by a zipper, which is partially unzipped, showing a dark interior. The background is a gradient of blue and orange. The text "THE END" is written in a white, bold, sans-serif font, arched over the top of his head.

THE END

QUESTIONS ???