Headaches

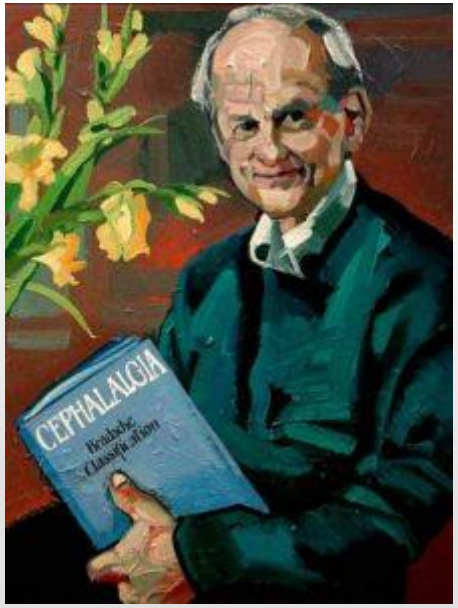
Diagnosis and management

Klinika Neurologii Dziecięcej WUM

IHS CLASSIFICATION ICHD-3

https://beta.ichd-3.org/

Above 280 types of headaches are described !



Jes Olesen
Chairman
Headache Classification Committee of the International Headache Society

- **Headache Classification Committee of the International Headache Society (IHS) : The International Classification of Headache Disorders, 3rd edition.**
  Cephalalgia 2018; 38 (1): 1–211.

- I. The primary headaches
- II. The secondary headaches
- III. Painful Cranial Neuropathies, Other Facial Pain and Other Headaches

Headache diagnosis:

1/ History talking !!! : localisation of the pain, character,
how long ?, how often ?, precipitating factors;
if aura present ? , character of aura, nusea, vomiting, if depends on body position, physical activity, weather, menstruation, sleep, history of headache in family, concomitant disease, concomitant treatment

In practice it is recommended to provide ***patient's diary***

2/ Physical examination (blood pressure), Neurological examination

3/ Laboratory: (morphology, CRP, OB., thyroid function,
Fe, Borrelia burgdorferi)

4/ Neuroimaging: MR of the head, angio MR, angio CT

5/ ECG

6/ Ophthalmologist consultation

7/ Laryngologist consultation- sinusitis can be a cause of chronic headache

8/ Stomatologist consultation

9/ Psychologist

HISTORY TALKING is very important to classify headaches

BÓLE GŁOWY Imię Nazwisko .....

WYWIAD:

1. ZWIĄZEK CZASOWY:
   PORA DNIA .....
   PO JAKIM CZASIE OSIĄGA MAKSYMALNE NATĘŻENIE .....
   .....
   CZĘSTOTLIWOŚĆ .....
   CZAS TRWANIA .....
2. PROFIL BÓLU:
   OBECNOŚĆ OBJAWÓW PRODROMALNYCH (POPRZEDZAJĄCYCH BÓL GŁOWY)
   .....
   .....
   .....
   ZWIĄZEK Z PRZYJMOWANYMI LEKAMI .....
   MIESIĄCZKA .....
3. LOKALIZACJA BÓLU: JEDNOSTRONNY, OBUSTRONNY, OKOLICA CZOŁOWA, POTYLICZNA, TWARZ, GŁOWA CAŁA, SZYJA .....
   .....
4. CHARAKTERYSTYKA BÓLU: PULSUJĄCY, STAŁY, „RAŻENIE PRĄDEM", ZACISKAJĄCY
   .....
   INTENSYWNOŚĆ .....
5. ZWIĄZEK Z INNYMI OBJAWAMI: NUDNOŚCI, WYMIOTY, UTRATA PRZYTOMNOŚCI, ŁZAWIENIE, NAGŁE UPADKI, SZTYWNOŚĆ KARKU, ZAWROTY GŁOWY, ŚWIATŁOWSTRĘT
   .....
   .....
6. CZYNNIKI SPUSTOWE LUB NASILAJĄCE DOLEGLIWOŚCI: WYSIŁEK, POZYCJA, POTRAWY, LEKI, POGODA, LĘK .....
   .....
7. CZYNNIKI ŁAGODZĄCE DOLEGLIWOŚCI: CIEMNE POMIESZCZENIE, POZYCJA, UCISK SKÓRY GŁOWY, LEKI
   .....
   .....
8. WYWIAD: .....
   WCZEŚNIEJSZE BÓLE GŁOWY .....
   .....
   .....
   OCENA POPRZEDNICH BÓLÓW GŁOWY .....
   .....
   .....
   LEKI .....
   NADCIŚNIENIE TĘTNICZE .....
   NOWOTWORY, PODRÓŻE, URAZY, DODATNI WYWIAD LUB RYZYKO INFEKCJI HIV
9. WYWIAD RODZINNY .....
   .....
   .....
10. NARAŻENIE: ROZPUSZCZALNIKI, CHEMIKALIA ETC

Oznaczenie* (zgodnie z Rozporządzeniem Ministra Zdrowia z dn. 21.12.2010) –
(nazwisko, imię, tytuł zawodowy, uzyskane specjalizacje, numer prawa wykonywania zawodu, podpis)

Headache database - useful questions

Do you have one or two types of headache?
How did the headaches begin?
How long has the headache been present?
Are the headaches static, intermittent or progressive?
How often does the headache occur?
How long does the headache last?
Do the headaches occur at any special time or under special circumstances?

Are the headaches related to specific foods, medication or activities?
Are there warning symptoms?
Where is the pain located?
What is the quality of the pain?
Are there associated symptoms during the headache?
What do you do during the headache?
What makes the headache better?
What makes the headache worse?
Do symptoms continue in between the headaches?
Are you being treated for any other medical problems?
Do you take any medication for any reason regularly or intermittently?
Does anyone in your family have headaches?
What do you think is causing your headaches, and why do you have them?

TENSION – TYPE HEADACHE

Tension headaches affect about 1.4 billion people (20.8% of the population) and are more common in women than men (23% to 18% respectively)

Episodic tension-type headache diagnostic criteria:

Description: Episodes of headache, typically bilateral, pressing or tightening in quality and of mild to moderate intensity, lasting minutes to days. The pain does not worsen with routine physical activity and is not associated with nausea, but photophobia or phonophobia may be present. Increased pericranial tenderness may be present on manual palpation.

A. At least 10 episodes of headache fulfilling criteria B through D. Infrequent and frequent episodic subforms of TTH are distinguished as follows:

Infrequent episodic TTH: Headache occurring on <1 day per month on average (<12 days per year).

Frequent episodic TTH: Headache occurring on 1 to 14 days per month on average for >3 months (≥12 and <180 days per year)

B. Headache lasting from 30 minutes to seven days.

C. At least two of the following four characteristics:

Bilateral location.

Pressing or tightening (nonpulsating) quality.

Mild or moderate intensity.

Not aggravated by routine physical activity such as walking or climbing stairs.

D. Both of the following:

No nausea or vomiting.

No more than one of photophobia or phonophobia.

E. Not better accounted for by another ICHD-3 diagnosis.

Various precipitating factors may cause tension-type headache:

- Stress: headache usually occurs in the afternoon after long stressful work hours or after an exam
- Sleep deprivation
- Uncomfortable stressful position and/or bad posture
- Irregular meal time (hunger)
- Eyestrain
- Tension-type headaches may be caused by muscle tension around the head and neck. One of the theories says that the main cause for tension-type headaches is **teeth clenching which causes a chronic contraction of the temporalis muscle.**

TTH Treatment:

- Prevention
- ibuprofen, paracetamol, acetaminophen, aspirin
- relaxation techniques

Doses of analgesic

Ibuprofen

5-10 mg/kg/dose, every 6-8 h, max. dose
40 mg/kg/day
Patients > 12 yo and adults: 200 – 400 mg, max. 1,2 g

Paracetamol

15 mg/kg/dose, every 4-6 h, max. 60 mg/kg/day
First dose can be higher 20 – 25 mg/kg
Patients >12 yo and adults: 0,5 – 1 g, max. 4 g/day

Pyralgina (metamizol)

- 15 yo (> 53 kg): 0,5 – 1 g every 6-8 h,
- Max. 3-5 g/day

MIGRAINE

- Migraine is believed to be due to a mixture of environmental and genetic factors.
- About two-thirds of cases run in families.
- Changing hormone levels may also play a role, as migraine affects slightly more boys than girls before puberty and two to three times more women than men.
- Mechanisms are not fully known – probably involve the nerves and blood vessels of the brain
- **Migraine** is believed to be a **neurovascular disorder** with evidence supporting its mechanisms starting within the brain and then spreading to the blood vessels. One theory is related to increased excitability of the cerebral cortex and abnormal control of pain neurons in the trigeminal nucleus of the brainstem. High levels of the neurotransmitter serotonin (5-hydroxytryptamine, are believed to be involved.

Migraine

1.1 Migraine without aura

1.2 Migraine with aura
1.2.1 Migraine with typical aura
1.2.1.1 Typical aura with headache
1.2.1.2 Typical aura without headache
1.2.2 Migraine with brainstem aura

1.2.3 Hemiplegic migraine
1.2.3.1 Familial hemiplegic migraine (FHM)
1.2.3.1.1 Familial hemiplegic migraine type 1 (FHM1)
1.2.3.1.2 Familial hemiplegic migraine type 2 (FHM2)
1.2.3.1.3 Familial hemiplegic migraine type 3 (FHM3)
1.2.3.1.4 Familial hemiplegic migraine, other loci
1.2.3.2 Sporadic hemiplegic migraine (SHM)
1.2.4 Retinal migraine (migraine headaches accompanied by visual disturbances or even temporary blindness in one eye).

1.3 Chronic migraine (headache that fulfills diagnostic criteria for *migraine headache* and occurs for a greater time interval. Specifically, greater or equal to 15 days/month for longer than 3 months).

MIGRAINE

1.4 Complications of migraine (describe migraine headaches and/or auras that are unusually long or unusually frequent, or associated with a seizure or brain lesion)

1.4.1 Status migrainosus (when migraine lasting longer than 72 hours)

1.4.2 Persistent aura without infarction

1.4.3 Migrainous infarction

1.4.4 Migraine aura-triggered seizure

1.5 Probable migraine

1.5.1 Probable migraine without aura

1.5.2 Probable migraine with aura

1.6 Episodic syndromes that may be associated with migraine:

1.6.1 Recurrent gastrointestinal disturbance

1.6.1.1 Cyclical vomiting syndrome

1.6.1.2 Abdominal migraine

1.6.2 Benign paroxysmal vertigo

1.6.3 Benign paroxysmal torticollis

MIGRAINE without aura

Description: Recurrent headache disorder manifesting in attacks lasting 4–72 hours .
Typical characteristics of the headache are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity and association with nausea and/or photophobia and phonophobia.

Diagnostic criteria:

A. At least five attacks fulfilling criteria B–D (for migraine with aura 2 attacks)
B. Headache attacks lasting 4–72 hours (when untreated or unsuccessfully treated) - in children and adolescents patients < 18 yo attack can last 2-72 hours
C. Headache has at least two of the following four characteristics:
1. unilateral location (in children and adolescents can be bilateral)
2. pulsating quality
3. moderate or severe pain intensity
4. aggravation by or causing avoidance of routine physical activity (e.g. walking or climbing stairs)
D. During headache at least one of the following:
1. nausea and/or vomiting
2. photophobia and phonophobia
E. Not better accounted for by another ICHD-3 diagnosis.

Migraine triggers:

Stress

Sleep disturbances (too short sleep, too long sleep)

Menstruation

Foods that increase the release of serotonin i.e. red wine, avocado, cheese

MIGRAINE with aura

Description: Recurrent attacks, lasting minutes, of unilateral fully reversible visual, sensory or other central nervous system symptoms that usually develop gradually and are usually followed by headache and associated migraine symptoms.

Diagnostic criteria:

A. At least two attacks fulfilling criteria B and C

B. One or more of the following fully reversible aura symptoms:

1. visual
2. Sensory (often a feeling of pins-and-needles begins on one side in the hand and arm and spreads to the nose–mouth area on the same side)
3. speech and/or language
4. motor
5. brainstem
6. retinal

C. At least three of the following six characteristics:

1. at least one aura symptom spreads gradually over 5 minutes
2. two or more aura symptoms occur in succession
3. each individual aura symptom lasts 5–60 minutes
4. at least one aura symptom is unilateral
5. at least one aura symptom is positive
6. the aura is accompanied, or followed within 60 minutes, by headache

D. Not better accounted for by another ICHD-3

MIGRAINE AURA:

- **AURA** - mechanism of "cortical spreading depression"
- Aura appears gradually over a number of minutes and generally last less than 60 minutes.
- Symptoms can be visual, sensory or motor in nature and many people experience more than one.

- Visual effects occur most frequently: scintillating scotoma (an area of partial alteration in the field of vision which flickers and may interfere with a person's ability to read or drive)

These typically start near the centre of vision and then spread out to the sides with zigzagging lines which have been described as looking like fortifications or walls of a castle. Usually the lines are in black and white but some people also see coloured lines. Some people loose part of their field of vision known as hemianopsia while others experience blurring.

- Up to one-third of people have an AURA: typically a short period of visual disturbance which signals that the headache will soon occur

„Alice in Wonderland Syndrom”

MIGRAINE - MANAGEMENT

1. Management of the migraine attack :

- Analgesic: Paracetamol, Ibuprofen, Aspirin, Naproxen, tolfenamic acid (Migea), solpadeina (paracetamol+ kofeina+kodeina)

- Triptans !!! i.e. sumatryptan (Imigran orally or nasally)

- Metoclopramid
- Ergotamine
- Status migrainosus (when migraine lasting longer than 72 hours) - Metoclopramid, Diazepam, Mannitol, steroids

2. Preventive treatment :

- Propranolol, Flunaryzyna, amitryptylina, antiepileptic drugs

(VPA, topiramat, gabapentyna)

- Non pharmacological methods - use of stress reduction techniques such as cognitive behavioural therapy and relaxation techniques
- elimination of triggers, dietary

SECONDARY HEADACHES

- Trauma capitis, vascular diseases, hypertension, infection,epilepsy, increased intracranial pressure (tumour cerebri, idiopathic intracranial hypertension=pseudotumour cerebri),
  laryngological disease (sinusitis, otitis), ophatmologic disease (vision loss, glaucoma)
- Headache in stomatology: odontogenic pain, non-odontogenic pain, temporomandibular joint dysfunction syndrome
- Headache in tumour cerebri: Due to increase of intracranial pressure, bilateral, often in the morning with vomiting - neuroimaging cito !!!
- *Bruns' syndrome*: characterized by sudden and severe headache, accompanied by vomiting and vertigo, triggered by abrupt movement of the head. Principal causes are cysts and cysticerosis of the fourth ventricle, and tumours of the midline of the cerebellum and third ventricle.

Clinical features that may indicate intracranial pathology in children and adolescents with headache

Headache characteristics

Headache awakens the child or occurs consistently upon awakening from sleep
Short or paroxysmal headache; thunderclap headache (uncommon in children)
Associated neurologic signs and symptoms (eg, persistent nausea/vomiting, altered mental status, ataxia, etc)
Headache worsened in recumbent position or by cough, micturition, defecation, or physical activity
Absence of aura
Chronic progressive headache pattern
Change in quality, severity, frequency, or pattern of headache
Occipital headache
Recurrent localized headache
Lack of response to medical therapy
Headache duration of less than six months

Clinical features that may indicate intracranial pathology in children and adolescents with headache

Patient history

Inadequate history (description of headache and relative features)

Risk factor for intracranial pathology (eg, sickle cell disease, immune deficiency, malignancy or history of malignancy,coagulopathy, cardiac disease with right-to-left intracardiac shunt, head trauma, neurofibromatosis type 1, tuberous sclerosiscomplex, pre-existing hydrocephalus or shunt)

Age <6 years

Personality change , Deterioration of school work

Associated symptoms in the neck or back

Family history: Absence of family history of migraine

Examination findings

Child uncooperative (unable to complete neurologic examination)

Abnormal neurologic examination (eg, ataxia, weakness, diplopia, abnormal eye movements, other focal signs)

Papilledema or retinal hemorrhages

Growth abnormalities (increased head circumference, short stature or deceleration of linear growth, abnormal pubertalprogression, obesity)

Nuchal rigidity

Signs of trauma

Cranial bruits

Skin lesions that suggest a neurocutaneous syndrome (neurofibromatosis, tuberosis sclerosis complex)

Trigeminal neuralgia = tic douloureux

- Episodes of intense facial pain along the trigeminal nerve divisions.

- The trigeminal nerve is a paired cranial nerve that has three major branches: the ophthalmic nerve (V_1), the maxillary nerve (V_2), and the mandibular nerve (V_3).

  Trigeminal neuralgia most commonly involves V2 or V3.

- Each individual attack usually lasts from a few seconds to several minutes or hours, but these can repeat for hours with very short intervals between each attack. In other instances only 4-10 attacks are experienced daily. The episodes of intense pain may occur paroxysmally.

Trigeminal neuralgia

- To describe the pain sensation, patients often describe a trigger zones on the face so sensitive that touching or even air currents can trigger an episode; however, in many patients the pain is generated spontaneously without any apparent stimulation.
- It affects lifestyle as it can be triggered by common activities such as eating, talking, shaving and brushing teeth. Wind, chewing and talking can aggravate the condition in many patients.

- The attacks are said by those affected to feel like stabbing electric shock, burning, sharp, pressing, crushing, exploding or shooting pain that becomes intractable.

Trigeminal neuralgia

Several theories exist to explain the possible causes of neuralgia.

Leading research indicates that it is an enlarged or lengthened blood vessel – most commonly the superior cerebellar artery – compressing or throbbing against the microvasculature of the trigeminal nerve near its connection with the pons. Such a compression can injure the nerve's protective myelin sheath and cause erratic and hyperactive functioning of the nerve. This can lead to pain attacks at the slightest stimulation of any area served by the nerve as well as hinder the nerve's ability to shut off the pain signals after the stimulation ends.

- This type of injury may rarely be caused by an aneurysm ; by an AVM (arteriovenous malformation); by a tumour in the cerebellopontine angle.
- Other causes: multiple sclerosis, Herpes simplex (V1)

Trigeminal neuralgia

DIAGNOSIS: MR, angio MR

TREATMENT:

- carbamazepin, gabapentin, okskarbazepine, pregabalin, valproic acid, lamotrigine, phenytoin, clonazepam
- Surgical – non-destructive method- microvascular decompression or destructive methods

Glossopharyngeal neuralgia (GN)

- Affects the glossopharyngeal nerve and causes sharp, stabbing pulses of pain in the back of the throat and tongue, the tonsils, and the middle ear.
- Pain can last for a few seconds to a few minutes, and may return multiple times in a day or once every few weeks.
- Many individuals with GN relate the attacks of pain to specific trigger factors such as swallowing, drinking cold liquids, sneezing, coughing, talking, clearing the throat, and touching the gums or inside the mouth.
- GN can be caused by compression of the glossopharyngeal nerve, but in some cases, no cause is evident.
- It can be associated with multiple sclerosis.
- GN primarily affects the elderly.

Cerebrovascular diseases

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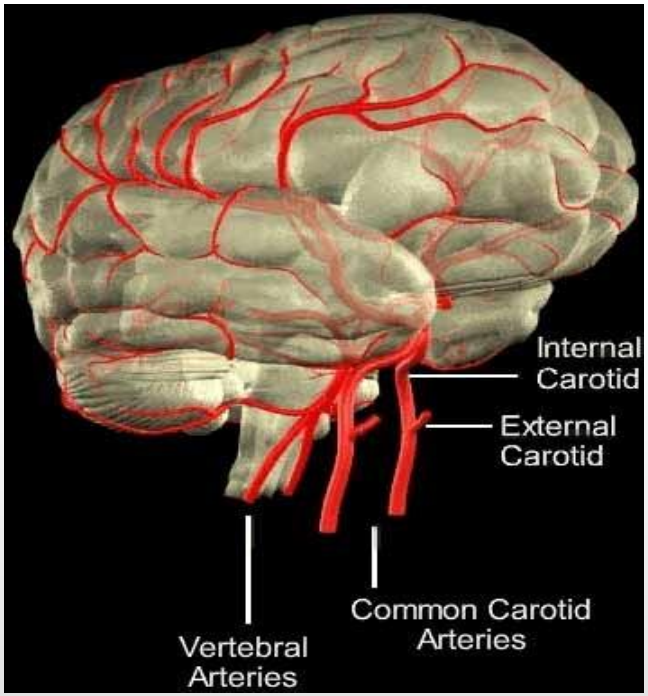
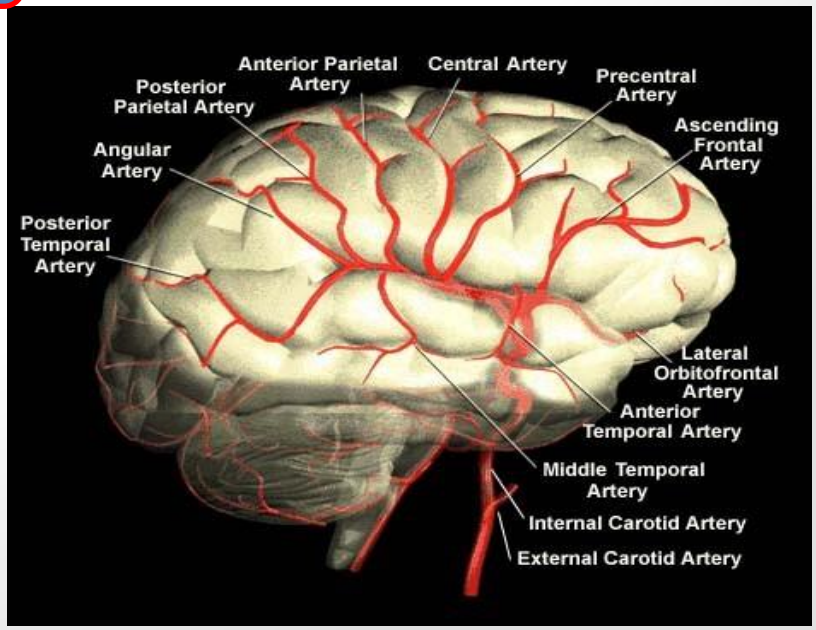
DEFINITION OF STROKE

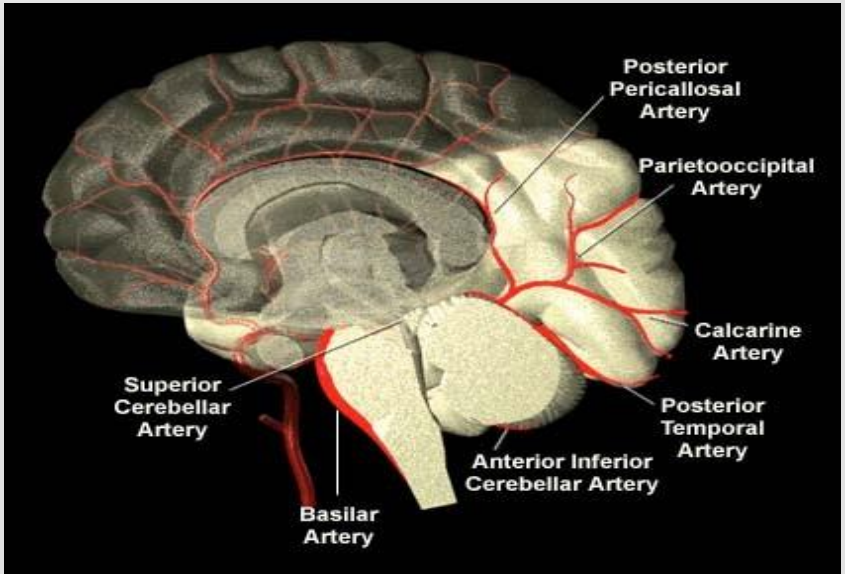
- Stroke, also known as cerebrovascular accident (CVA) or "brain attack", is a syndrome caused by a disruption in the flow of blood to part of the brain due to either occlusion of a blood vessel (ischemic stroke) or rupture of a blood vessel (hemorrhagic stroke). The interruption in blood flow deprives the brain of nutrients and oxygen, resulting in injury to cells in the affected vascular territory of the brain. Ischemic strokes are more common than hemorrhagic strokes.

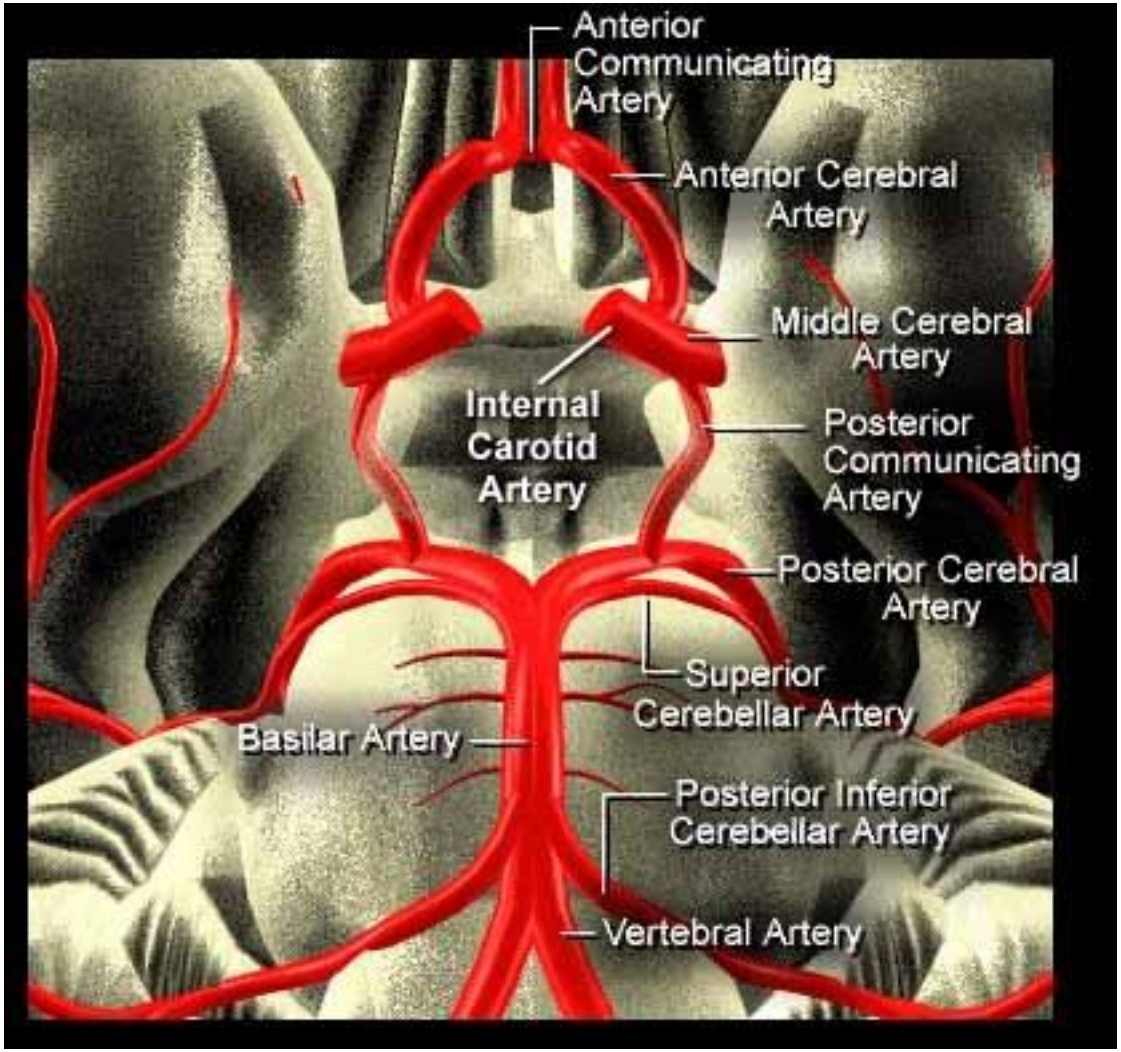
- When brain cells die, function of the body parts they control is impaired or lost, causing paralysis, speech and sensory problems, memory and reasoning deficits, coma, and possibly death.

Cerebral vascularization








Anterior Communicating Artery
Anterior Cerebral Artery
Middle Cerebral Artery
Internal Carotid Artery
Posterior Communicating Artery
Posterior Cerebral Artery
Superior Cerebellar Artery
Basilar Artery
Posterior Inferior Cerebellar Artery
Vertebral Artery

Types of stroke

Ischemic stroke (IS)
75-85 %

Intracerebral hemorrhage (ICH)
10-20%

Subarachnoidal hemorrhage (SAH)

5%

Venous stroke

0,5- 1%

BRAIN ISCHEMIA

- **Transient ischemic attack, TIA (<24 hours)**

- Reversible ischemic neurological deficit, RIND (<21 days)

- Minor stroke (non-disabling)
- Major stroke (disabling)

NON MODIFABLE FACTORS ASSOCIATED WITH AN INCREASED RISK OF STROKE

- Age (increased with age > 65)
- Gender (males > females)
- Geographic region (Eastern Europe > Western Europe; Asia > Europe or North America)
- Family history (stroke or heart disease < age 60)
- Genetic factors : CADASIL, Marfan's disease, homocystynuria, lack of protein C i S, cavernous malformation

POTENTIALLY MODIFABLE RISK FACTORS FOR STROKE

- Hypertension
- Diabetes mellitus
- Hyperlipidemia
- Smoking
- Atrial fibrillation
- Hyperhomocysteinemia
- Physical activity

OTHER POTENTIAL RISK FACTORS

- Migraine
- Oral contraceptives
- Obesity
- Pregnancy
- Alcohol abuse
- Drug abuse
- Sleep disorders (sleep apnea)

TYPES OF IS

- **ATHEROSCLEROTIC -** Plaques lead to stenosis, occlusion, distal embolisation (artery-to-artery embolism) and steal phenomena
- **EMBOLIC** (cardioembolic)
- **LACUNAR** (in arterial hypertension)

CARDIAC CAUSES OF IS

- **Atrial fibrillation**
- Recent MI
- Ventricular aneurysm (post MI)
- Akinetic segment (post MI)
- Dilated cardiomyopathy
- Mural or intraventicular thrombus
- Valvular abnormalities (mitral insufficiency, mitral and aortal stenosis). Also congenital
- **Infective endocarditis**
- Atrial septal aneurysm / defect
- **Patent foramen ovale (PFO)- paradoxical embolism**
- Myxoma
- Mechanical or bioprosthetic valve
- CABG, PTCA, other cardiac surgery

OTHER CAUSES OF IS

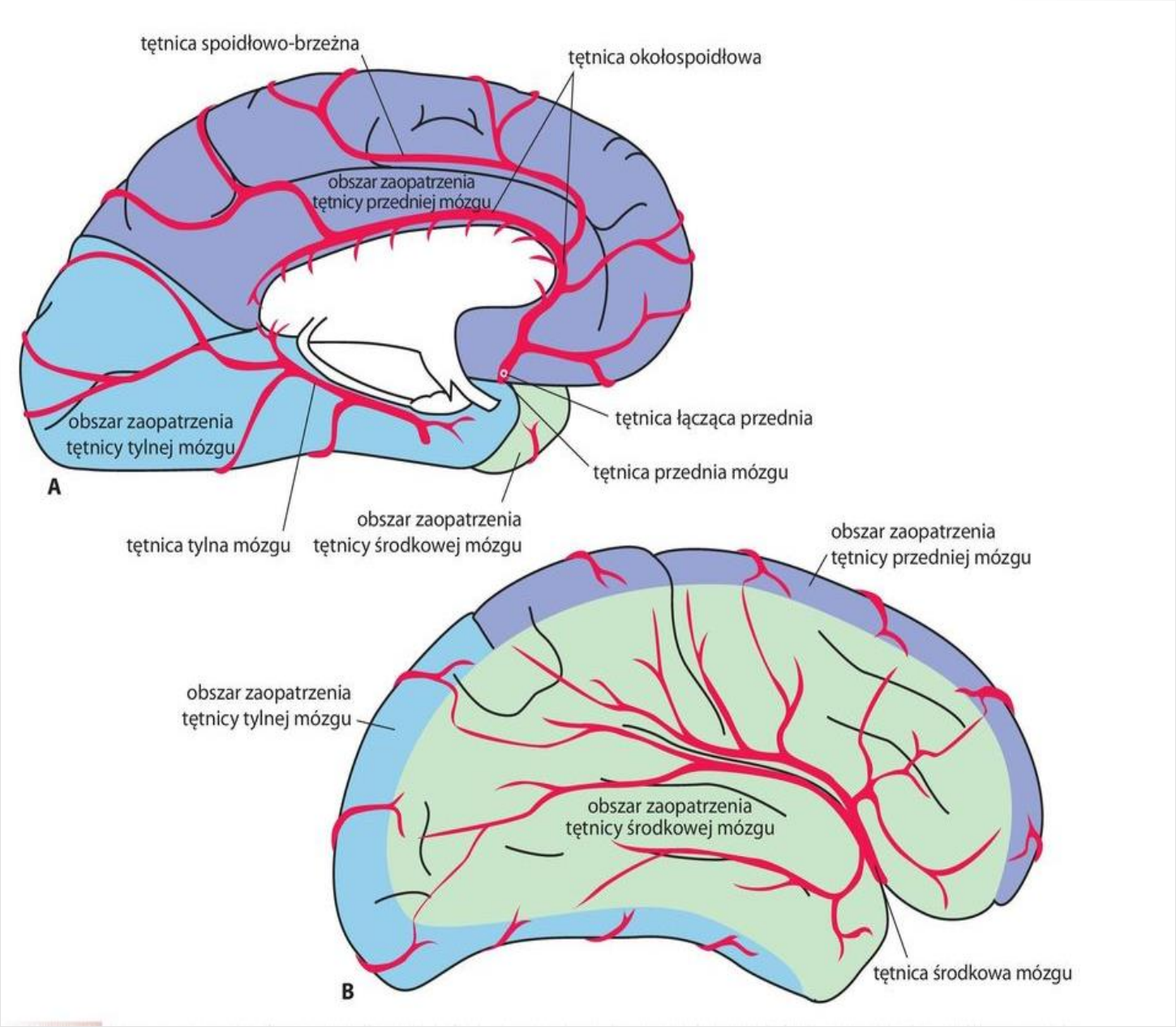
- Vasculopathies
  - Noninflamatory (dissection, vasospasm, others)
  - Inflamatory (PAN, SLE, vasculitis, others)
  - Infectious (syphilis, Herpes Zoster, COVID- 19, AIDS, others)

OTHER CAUSES OF IS

- **Hematologic and coagulation disorders**
  - Polycythemia, thrombocytosis, trombocytopenia
  - Antithrombin III deficiency
  - Protein C or S deficiency
  - Deficiency of factors V, VII, XII, XIII
  - Antiphospholipid/anticardiolipin antibodies
  - Malignancy
  - Pregnancy
  - Oral contraceptives

SYMPTOMS OF STROKE

- Symptoms depend on localization and size of lesion !!!!
- Less on etiology / cause of stroke
- Focal symptoms
- Global symptoms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tętnica okołospoidłowa
obszar zaopatrzenia
tętnicy przedniej mózgu
obszar zaopatrzenia
tętnicy tylnej mózgu
tętnica łącząca przednia
tętnica przednia mózgu
A
tętnica tylna mózgu
obszar zaopatrzenia
tętnicy środkowej mózgu
obszar zaopatrzenia
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tętnicy tylnej mózgu
obszar zaopatrzenia
tętnicy środkowej mózgu
tętnica środkowa mózgu
B

SYMPTOMS OF STROKE

Time course and evolution:

- Sudden or rapid onset of symptoms
  - in the morning, in daytime
  - on sleep, exercise

- Reach maximal intensity within 24 hours

- Gradual or stepwise worsening can occur

Focal neurological symptoms of stroke

- Cognitive impairments (aphasia, neglect, apraxia)
- Weakness or incoordination of limbs
- Facial weakness
- Numbness of limbs and/or face
- Cranial nerve palsies

Global symptoms and signs of stroke

- Headache
- Nausea and vomiting

- Altered mental status
  - syncope
  - seizure
  - Coma

- Hypertension and abnormal vital signs

- Nuchal rigidity

SYMPTOMS OF TIA / STROKE

- Carotid circulation
  - Ipsilateral monocular blindness
  - Contralateral weakness, numbness (hand, arm, face, leg)
  - Aphasia

Stroke in left hemisphere (ie, dominant)

- Right hemiparesis, variable involvement of face and upper and lower extremity
- Right-sided sensory loss, in a similar patern to the motor deficit; usually involves all modalities, decreased stereognosis, graphesthesia
- Right homonymous hemianopsia
- Aphasia, fluent and nonfluent
- Alexia
- Agraphia
- Acalculia
- Apraxia

Stroke in right hemisphere (ie, nondominant)

- Left hemiparesis
- Left-sided sensory loss
- Left homonymous hemianopia
- Neglect of the left side of environment
- Anosognosia (paresis ignored)
- Asomatognosia (not recognizing body parts and sides)
- Loss of prosody of speech
- Flat affect

Middle cerebral artery

- hemiparesis opposite to the affected side (specially distally)

Initially reduced muscle tone then spastic , Babinski's sign usually (+) from the beginning

- Hemisensory loss opposite to the affected side (greater intensity distal)
- homonymous hemianopsia
- If dominant hemisphere affected: aphasia, alexia, agraphia, acalculia, apraxia

Anterior cerebral artery

Main trunk :

Hemiparesis opposite to the affected side, mainly in the lower limb
Paresis of eyesight
Urinary incontinence
Behavioral disorders, retardation

Heubner's artery (vascularization of the anterior part of the capsula interna) – central facial paresis, and paresis of upper limb, aphasia (if dominant hemisphere)

Posterior circulation

Posterior cerebral artery (PCA) occlusion

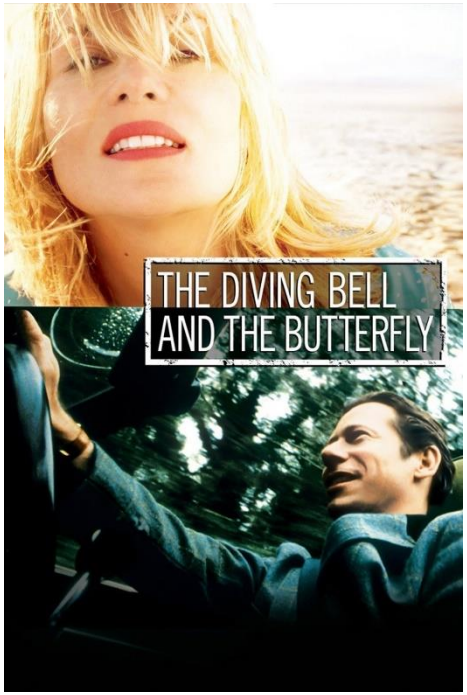
- The most common finding is occipital lobe infarction leading to contralateral hemianopsia with macular sparing

- Clinical symptoms associated with occlusion of the PCA vary depending on the location of the occlusion and may include the thalamic syndrome, thalamic perforate syndrome, Weber syndrome, cortical blindness, color blindness, failure to see to-and-fro movements, verbal dyslexia, and hallucinations.

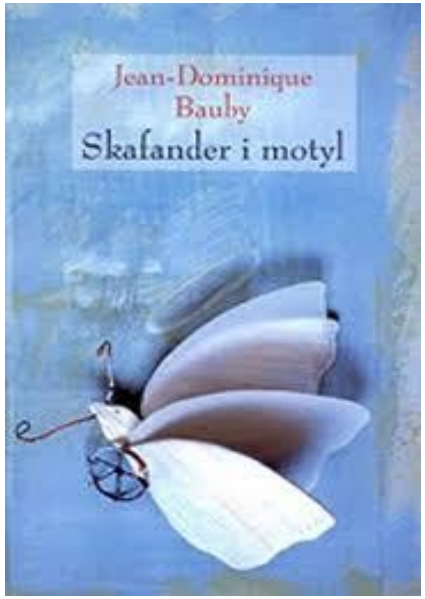
Basilar artery oclusion

- Brain stem stroke

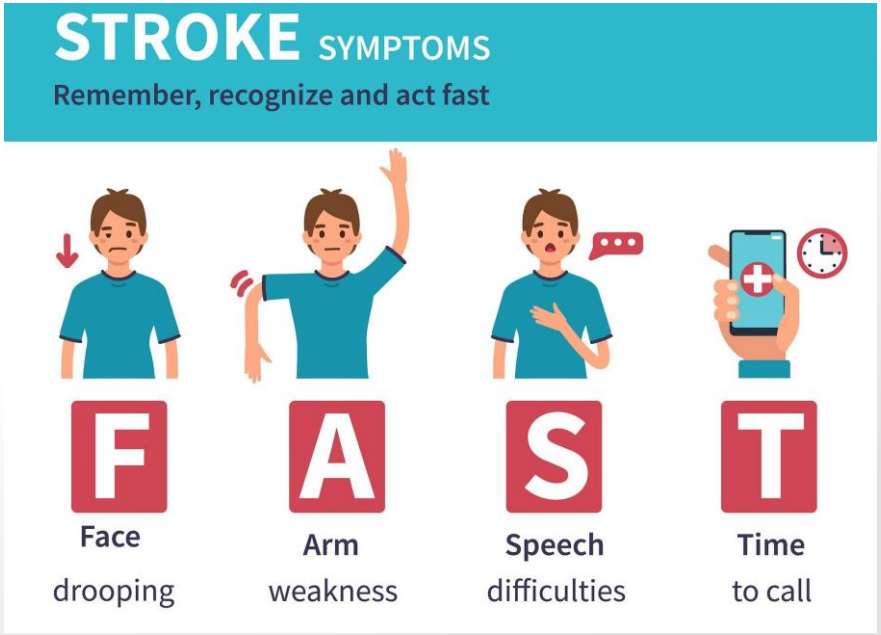
➢ **Locked - in syndrome:**



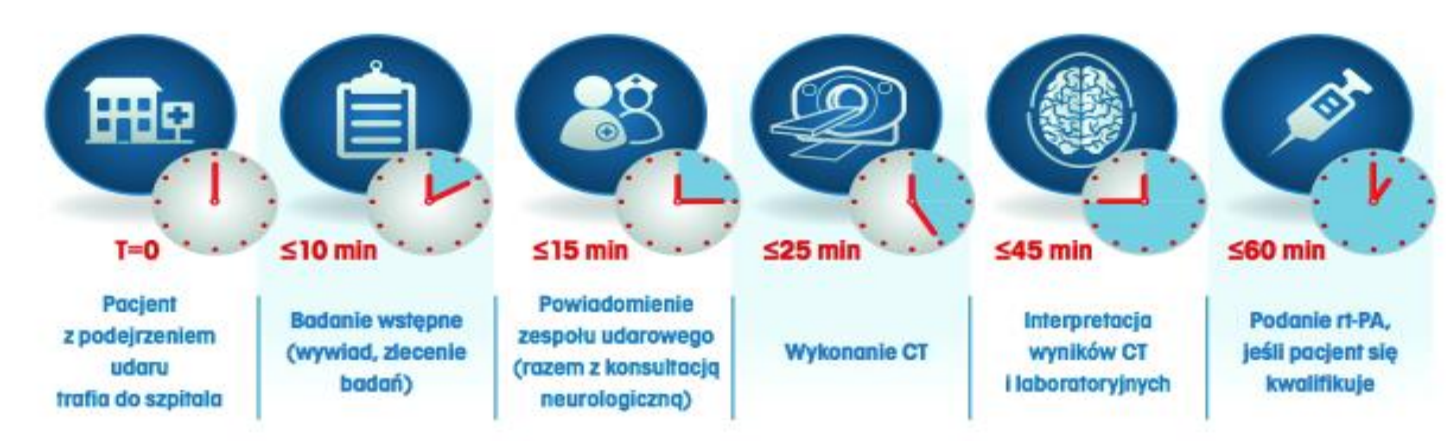
Quadriplegia and **paralysis** that is global except for vertical **eye movements** and blinking.
A coma-like condition (**pseudocoma**) where the patient only can respond or communicate with others by **eye movements**.



Ischemic stroke treatment

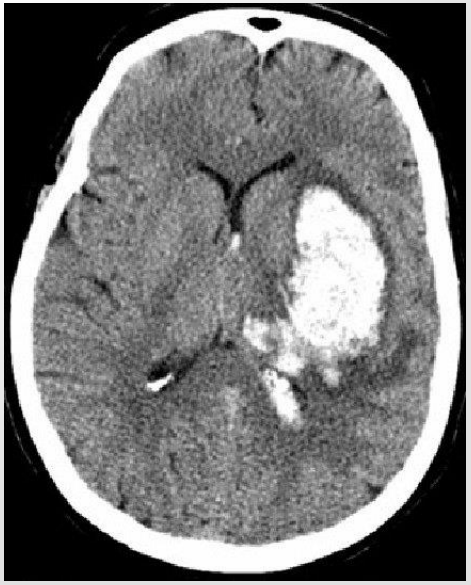


- „GOLDEN HOUR „



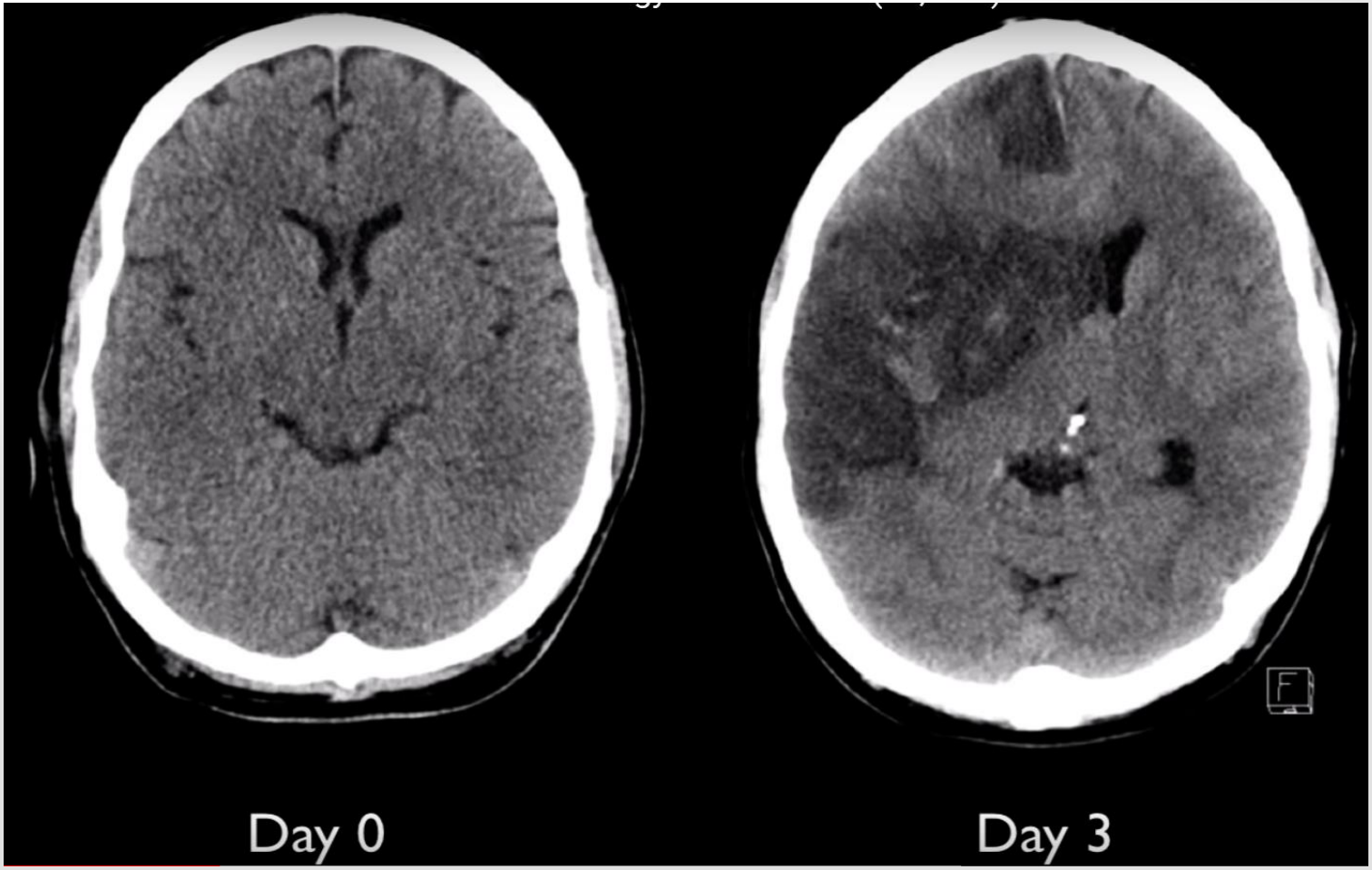
DIAGNOSIS OF STROKE

- Clinical symptoms
- Computer tomograhy (CT) urgently !!!!! (CT can exlude hemorrhage or tumour)



- Auscultation of cervical arteries
- Doppler of cervical and vertebral arteries
- Laboratory samples (morphology, parameters of the coagulation system , lipids, glucose, electrolites, creatynine)
- Cardiologic tests (ECG, echocardiography)

Ischemic stroke – CT changes over time



ISCHEMIC STROKE TREATMENT

- Trombolisis (rt- PA recombinant tissue plasminogen activator) – Alteplase intravenously only if < 4,5 hours since the first symptoms of stroke
- Ony is some centers - Mechanical trombectomy - till 6 hours after first symptoms of stroke
- Anti-aggregation : ASA 150-300 mg/day
- Hypo- and hyperglycemy treatment,
- Blood pressure (should be decreased carefully),
- Anticonvulsants
- Electrolites disturbances treatment (hyponatraemia is very often in stroke !),
- Features of increased intracranial pressure - Furosemid, Mannitol
- Anti-infective
- Cardiological treatment

Early rehabilitation !!!

Stroke treatment

- Secondary prevention :

N*ovel oral anticoagulants* – NOAC :

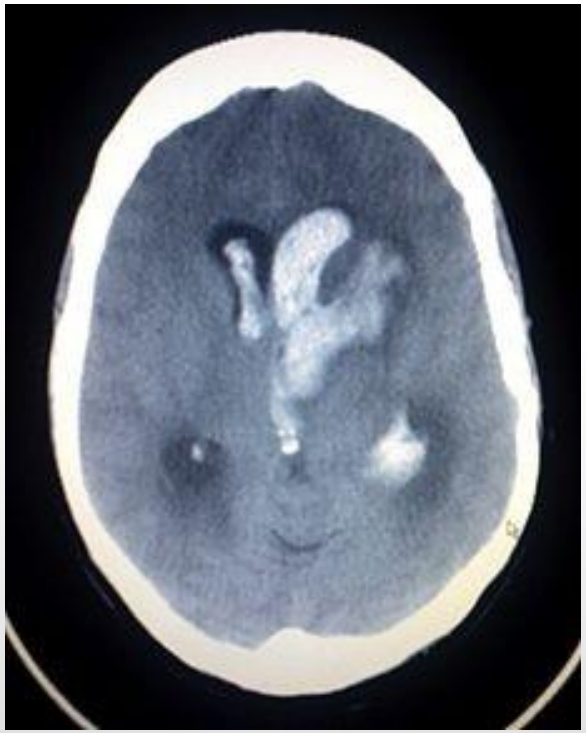
dabigatran (Pradaxa), riwaroksaban (Xarelto), apiksaban (Eliquis),

edoksaban(Lixiana)

- Treatment of risk factors: Statins in hypercholesterolaemy, arterial hypetension treatment , hyperglicemy treatment

- Endarterektomy of common carotid artery – when stenosis is > 70 %

Intracerebral hemorrhage (ICH)



Causes:

- spontaneous intracerebral hemorrhage (in pt with hypertension)
- head trauma
- rupture of an aneurysm or arteriovenous malformation (AVM)
- bleeding within a tumor
- amyloid angiopathy

Risk factors for ICH include:

- hypertension (high blood pressure)
- Diabetes mellitus
- Cigarette smoking
- Excessive alcohol consumption
- Severe migraine

Signs and symptoms : severe headache, focal neurological signs, loss of consciousness, seizures, vomiting

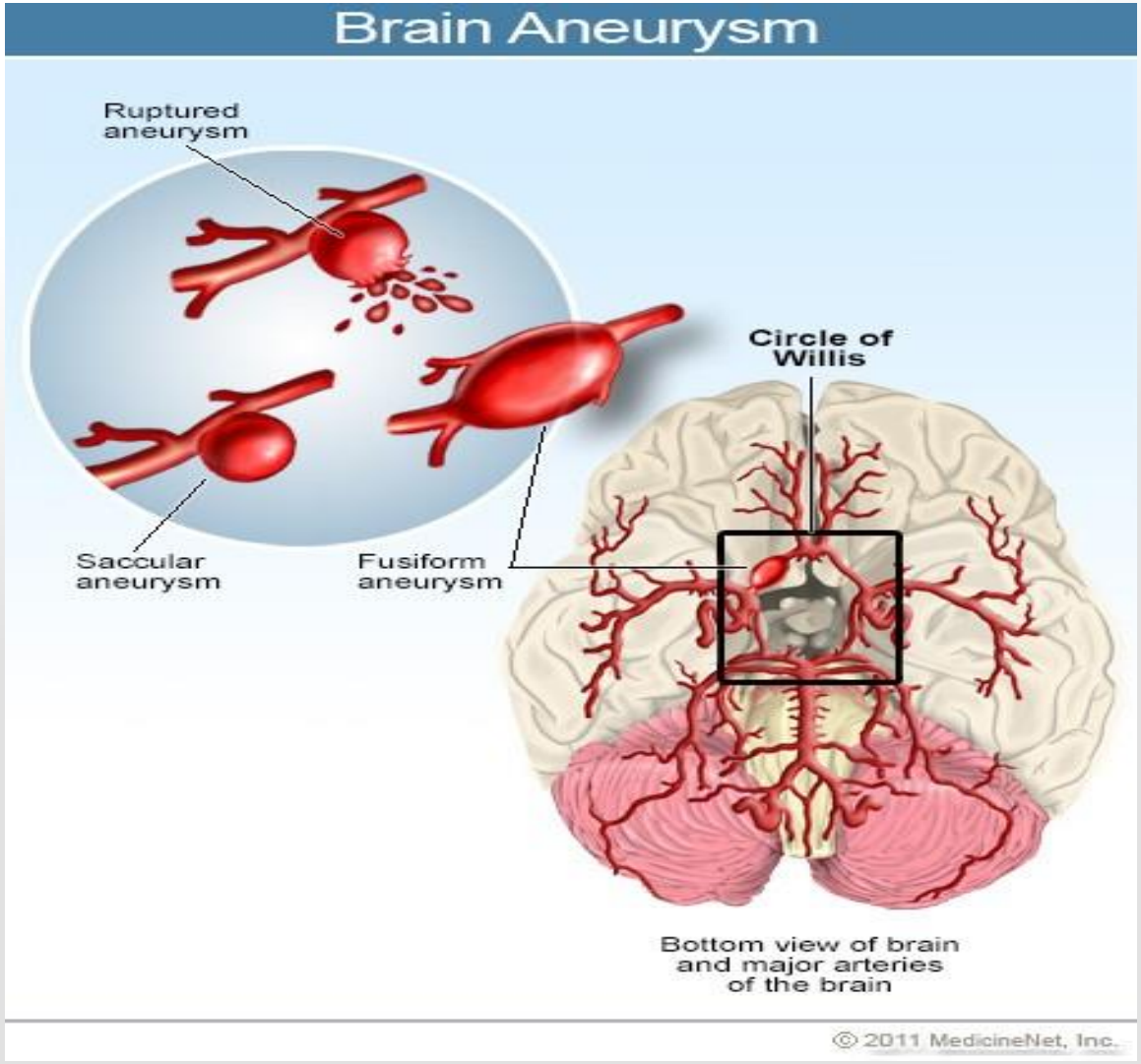
Treatment – symptomatic treatment, neurosurgery

Subarachnoid haemorrhage (SAH)

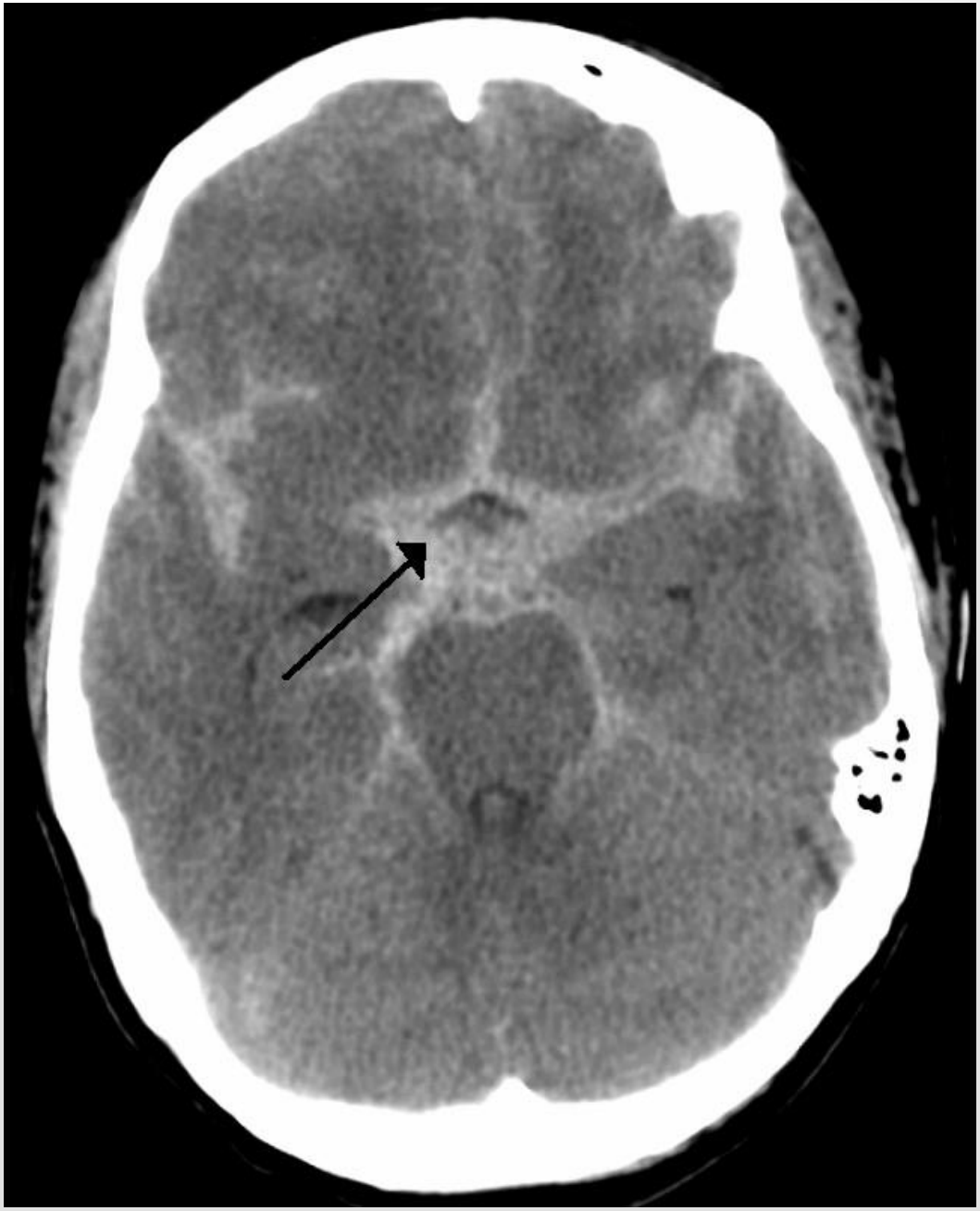
- Bleeding into the subarachnoid space — the area between the arachnoid membrane and the pia mater surrounding the brain.

- This may occur spontaneously, usually from a ruptured cerebral aneurysm, or may result from head injury

Cerebral aneurysm - a weakness in the wall of one of the arteries in the brain that becomes enlarged.
They tend to be located in the circle of Willis and its branches.



Subarachnoid haemorrhage (SAH) in CT



Subarachnoid haemorrhage (SAH)

Signs and symptoms : a **severe headache** with a rapid onset ("thunderclap headache"), a headache described as "like being kicked in the head", or the "worst ever", developing over seconds to minutes.
This headache often pulsates towards the occiput (the back of the head).
Meningism, vomiting, confusion or a lowered level of consciousness, and sometimes seizures

Diagnosis: CT / MR of the head, angiography

lumbar puncture - mandatory in people with suspected SAH if imaging is negative (red blood cells, xanthochromia at least >12 hours after the headache)

Management of Subarachnoid haemorrhage (SAH)

- involves general measures to stabilize the patient while also using specific investigations and treatment
- prevention of rebleeding by obliterating the bleeding source (neurosurgery)
- prevention of a phenomenon known as vasospasm: nimodipine - calcium channel blocker
- prevention and treatment of complications: seizures - antiepileptic drugs, electrolyte disturbances like hyponatremia, hydrocephalus

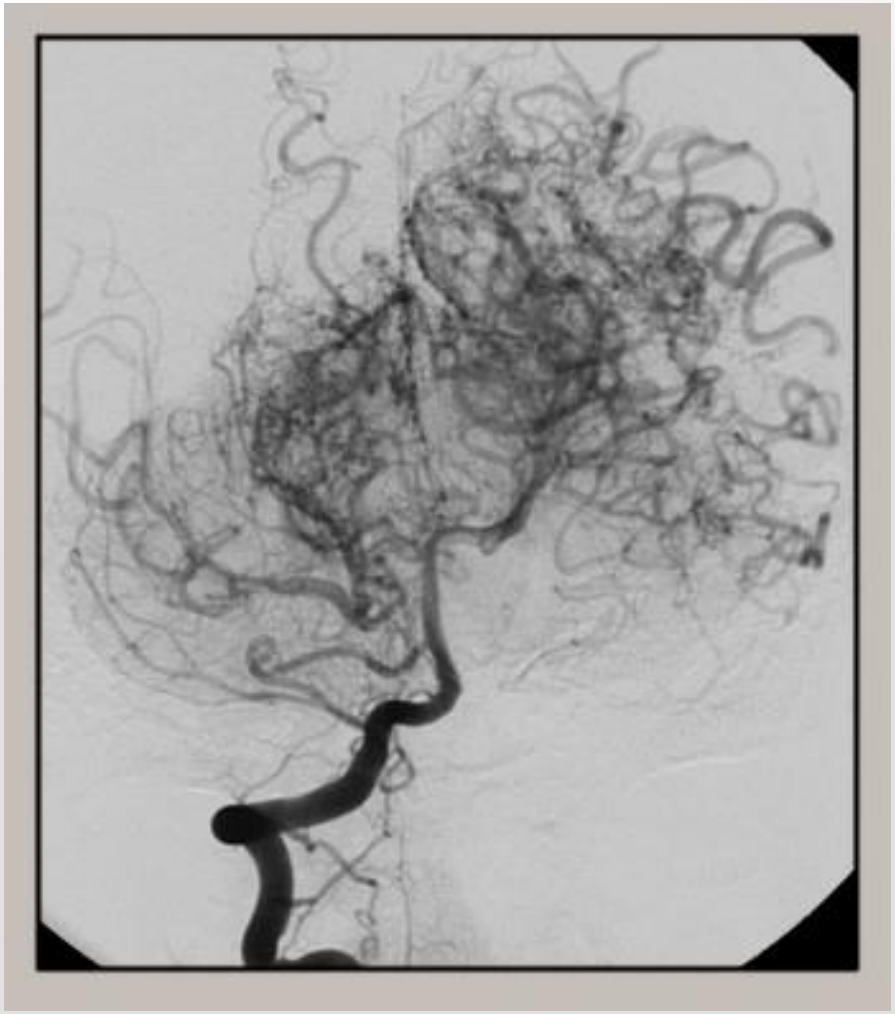
Moyamoya syndrome :

- is a disease (congenital in Japanese) or acquired (after radiotherapy, in arterial hypertension, after vasculitis) in which certain arteries in the brain are constricted.
- A collater circulation develops around the blocked vessels to compensate for the blockage, but the collateral vessels are small, weak, and prone to hemorrhage, aneurysm and thrombosis.

  On conventional X-ray angiography, these collateral vessels have the appearance of a "puff of smoke" (described as "もやもや(moyamoya)" in Japanese).
- Moyamoya disease tends to affect adults in the third to fourth decade of life (women> men) .
- In children it tends to cause strokes or seizures.
- In adults it tends to cause strokes or bleeding. The clinical features are strokes, recurrent transient ischemic attacks (TIAs), sensorimotor paralysis (numbness and paralysis of the extremities), convulsions and/or migraine-like headache.

- Treatment: ASA, surgical

Angiography - collateral vessels have the appearance of a "puff of smoke" described as "moyamoya" in Japanese



Cerebral vein and dural sinus thrombosis, CVT

- Cerebral vein and dural sinus thrombosis (CVT) is rare.

- It is estimated to cause less than 1% of all strokes - Venous stroke (0,5 - 1 % of all types of strokes)

- In many cases, the disease is not recognized or is diagnosed too late, and then its consequences are very serious.

- Thrombosis of the upper sagittal sinus (62%) most often occurs, followed by transverse sinus (41-45%), straight sinus (18%), and superficial cortical veins (the results of the research are divergent - 6-17%), internal jugular vein (12%), deep vein system (11%), cavernous sinus (3%), cerebellar veins (2%).

Causes:
- congenital thrombophilia;
- especially in young patients it is important to consider potential risk factors (pregnancy, hormonal contraception, autoimmune diseases, infections) in the diagnostic process.

Cerebral vein and dural sinus thrombosis, CVT

SIGNS AND SYMPTOMS

The most common symptom of CVT is **headache**, which is present in approximately 95% of cases. Headache in CVT is most often described as blinding, sometimes well localized, less often pulsating.

In 30-50% of cases, there are **features of increased intracranial pressure** (headache, vomiting, edema of the optic nerves, paresis of the VI nerve, unilateral or bilateral).

Focal neurological symptoms occur in 30-80% and may include paresis, speech disorders, impaired consciousness, amblyopia, ataxia, paresis of the cranial nerves.

In 10-15% of patients the first symptom is **convulsions**.

In the course of the disease, seizures occur much more often, in as much as 10-76% of cases.

Deep cerebral vein thrombosis or massive straight sinus thrombosis are usually associated with **disturbances of consciousness**, rather quantitative (10-63%), less often qualitative (15-20%).

A typical symptom of cavernous sinus thrombosis is **painful proptosis of the eyeball and swelling of the face** resulting from obstructed venous outflow.

Other symptoms of CVT are: earache, toothache, facial pain, swelling of the behind the ear, fever, meningeal symptoms.

Cerebral vein and dural sinus thrombosis, CVT

DIAGNOSIS

- TK/ MR, angio TK/ angio MR, venography,

- Diagnosis for trombophilia:

•Lupus anticoagulant,
•Anticardiolipin antibodies,
•The concentration of protein C and S (the test is performed 2-4 weeks after the end of anticoagulant treatment),
•Antithrombin III (the test is performed 2-4 weeks after the end of treatment with an anticoagulant),
•Homocysteine (a strong independent risk factor present in 27-43% of patients with CVT),
•Factor V Leiden mutations,
•Prothrombin gene mutations (G20210),
•Factor VIII

TREATMENT: antycoagulants : Heparin, vitamin K antagonists (acenokumarol, warfaryna), antibacterial and antiviral drugs, trombolisis, drugs to reduce elevated intracranial pressure, sometimes neurosurgery.

Cerebral vein and dural sinus thrombosis (CVT)

- In 20% of cases, routine computed tomography without a contrast agent allows to detect hyperintense thrombus in the sinus or vein projection (dense clot sign).
- In the case of cortical vein thrombosis, the so-called chord symptom (that is, a linear, hyperintensive change corresponding to a blood clot in the lumen of a venous vessel)