Intracranial hypertension

Intracranial pressure

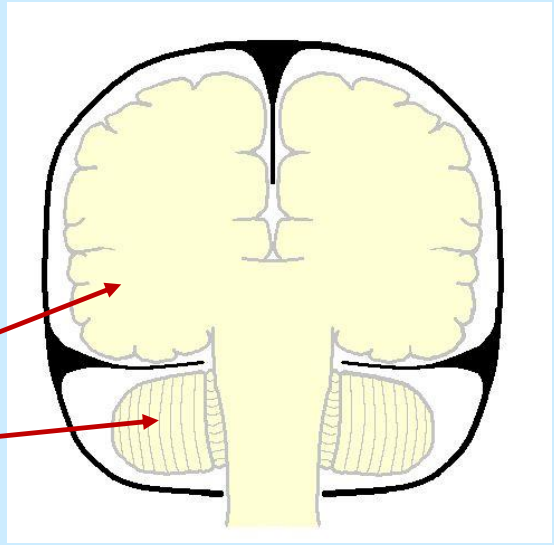
Normal intracranial pressure in horizontal position

= 7-15 mm Hg

(values over 40 mm Hg = intracranial hypertension)

Intracranial space - supratentorial space

- infratentorial space



Total content of the cranial cavity (brain 80 %, cerebrospinal fluid 10 %, blood 10 %) has a constant volume. Increase of any of the intracranial structures or occurrence of a new structure (neoplasm) leads to reduction of volume of other compounds and increase of intracranial pressure (Monro-Kellie‘s hypothesis).

Processes increasing their volume in the cranial cavity = **expansive intracranial processes**

- intracranial tumors
- brain edema
- hematomas
- hydrocephalus

Intracranial hypertension syndrome

- headache – worsens in horizontal position
- vomiting, sometimes without nausea
- disorders of consciousness (dominant at fats developing intracranial hypertension)
- focal symptoms depending on localization of the primary pathological process
- papilledema
- bulging fontanelle in infants

Intracranial hypertension syndrome

- one of the main mechanisms of the secondary brain injury
- decreases perfusion pressure (CPP = MAP – ICP)
  → brain hypoperfusion
  → reduction of vein drainage
  → hypoxia, acidosis
  → brain edema → potentiation of intracranial hypertension
  → blockade of CSF circulation → potentiation of intracranial hypertension

Brain herniations (conus)

- expansive processes push the brain tissue into a relatively free space

Brain herniations

1. Subfalcial (lateral) conus

- in the case unilateral expansive process in the hemisphere → usually no focal symptoms caused directly by the conus

2. <u>Tentorial conus</u>

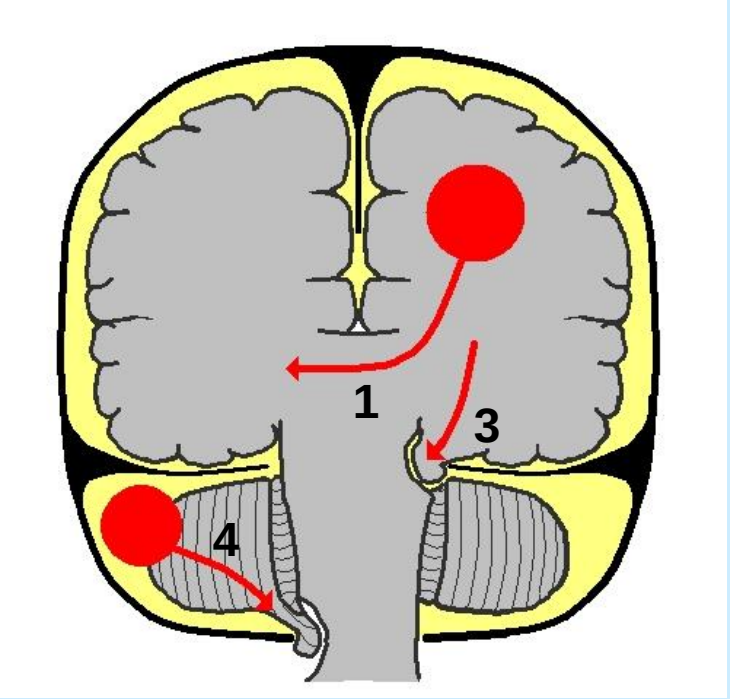
- in the tentorial foramen
- in symmetric supratentorial expansion → descendent
  → compression of the brainstem and n. III → deterioration of consciousness, ipsilateral mydriasis, no photoreaction, oculomotor disorder
- in symmetric infratentorial expansion → ascendant
  → nausea, consciousness deterioration

3. <u>Lateral tentorial (temporal, uncal) conus</u>

- unilateral tentorial conus
- in unilateral supratentorial expansion → unilateral focal symptoms (contralat. paresis n. III)

4. <u>Occipital (tonsillar) conus</u>

- through the foramen occipitale magnum
- in infratentorial expansive processes or in long-lasting supratentorial expansion → injury of the cerebellum (tonsil) and compression of the medulla oblongata (vital centers)



Intracranial hypertension compensation

Autoregulation of brain blood flow

↑ systemic BP, alkalosis, hypocapnia → constriction of brain vessels

↓ systemic BP, acidosis, hypercapnia → dilatation of brain vessels

Cushing‘s reflex:

ischemia (brain stem hypoxia) → stimulation of sympathetic system → ↑ heart rate, ↑ heart contraction, peripheral vasoconstriction → ↑ BP → ↑ perfusion pressure → ↑ brain perfusion

↑ BP → baroreceptor activation → ↓ heart rate (but hypertension persists)

→ bradycardia and ↑ BP at intracranial hemorrhage

Compensatory mechanisms (reserve)

- ↓ CSF volume, ↓ blood volume in the intracranial vessels (mainly veins)

Intracranial hypertension compensation

1. **Full compensation**
   - ↓ production and ↑ resorption of CSF

2. **Partial compensation**
   - ↑ systemic BP → maintenance of perfusion pressure
   - ↓ volume of blood in the intracranial vessels

3. **Decompensation**
   - subsequent ↑ of systemic BP, ↓ heart rate
   - hypoxia and hypercapnia → brain vessel dilatation → next ↑ of intracranial pressure

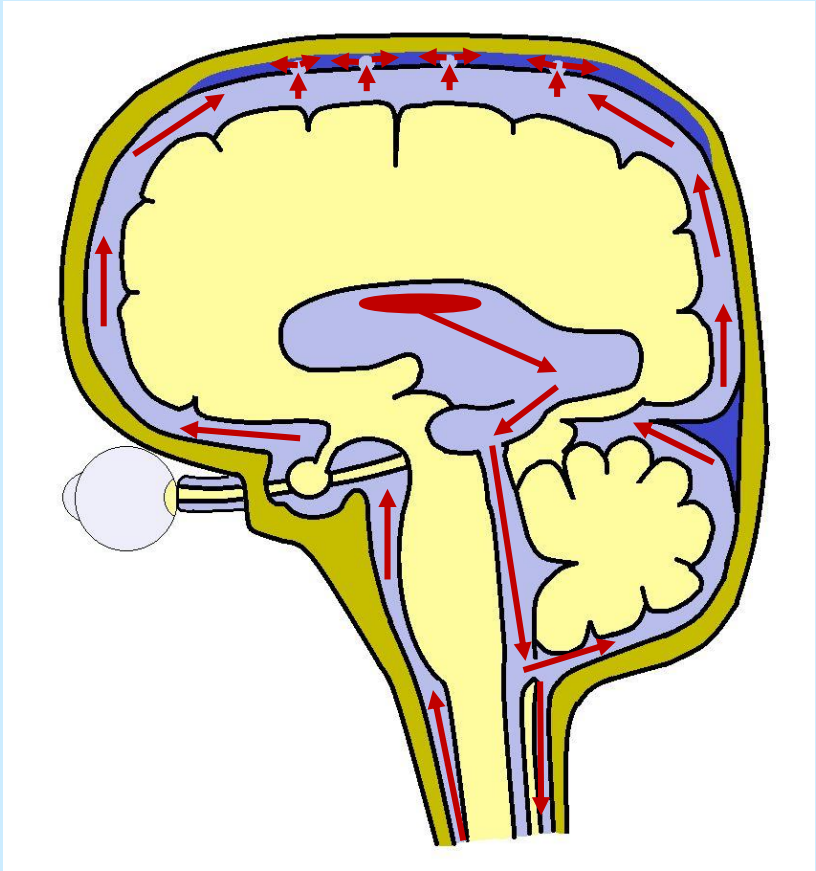
Intracranial hypotension

- CSF loss (lumbal puncture, penetrating head injuries, fistula)
- dehydration
- idiopathic

Manifestations:
- similar to intracranial hypertension, but relief in horizontal position
- headache, nausea, vomiting
- less dangerous

Disorders of cerebrospinal fluid circulation

Volume: 100-180 ml
Production: choroid plexus in the lateral ventricles
Cirkulation: lateral ventricles → foramina Monroe → ventricle III → aqueductus Sylvii → ventricle IV
→ spinal cord central channel
→ foramen Magendi and Luschkae → cisterna magna → subarachnoidal space
Resorption: villi arachnoideales
Complete exchange within 5-7 hours



Hydrocephalus

= increase of CSF volume with extension of the brain ventricles and potentially also subarachnoid space

X hydrocephalus e vacuo = enlargement of CSF space due to brain atrophy

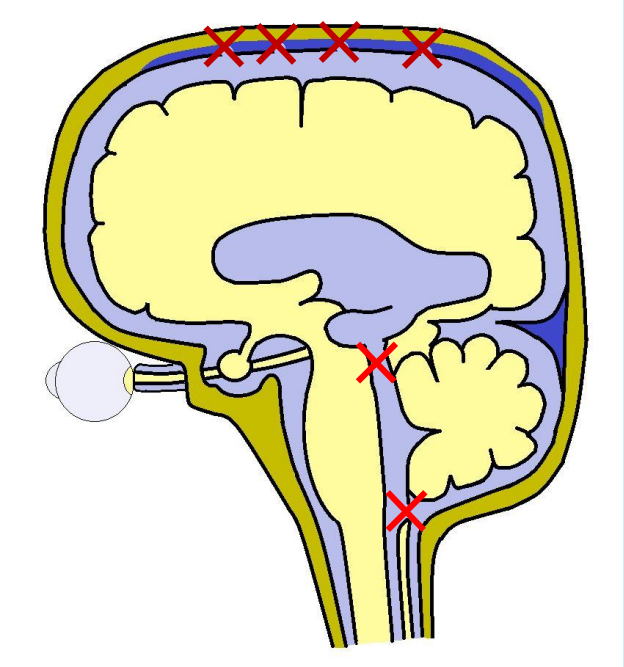
Mechanism of development:

- reduction of CSF resorption, in rare cases increased CSF production

Communicating hydrocephalus

= CSF accumulation also in subarachnoid space

- defective CSF resorption in villi arachnoideales, deteriorated circulation in subarachnoid space due to meningeal adhesions after purulent meningitis or bleeding
- in rare cases CSF hypersecretion by a choroid plexus papilloma



Non-communicating hydrocephalus

= dilation of brain ventricles

- obstruction of communication between the ventricles and subarachnoid space
- obstruction by an tumor, after inflammation, bleeding, developmental defect

Hydrocephalus manifestations

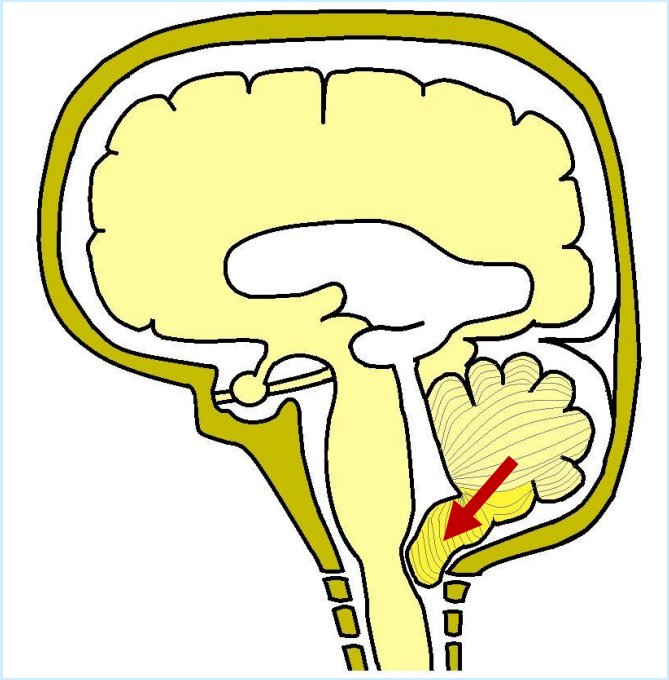
- **acute hydrocephalus** – symptoms of intracranial hypertension, focal symptoms related to the cause and localization of the obstruction
- **chronic hydrocephalus** – gait disorders, incontinency, dementia
  In children (< 2 years) – head enlargement

Normotensive hydrocephalus

- normal intracranial pressure, only pulses of intracranial hypertension
- gait disorders, incontinency, dementia
- unknown pathogenesis, sometimes a consequence of an injury, bleeding, infection, but often unknown etiology

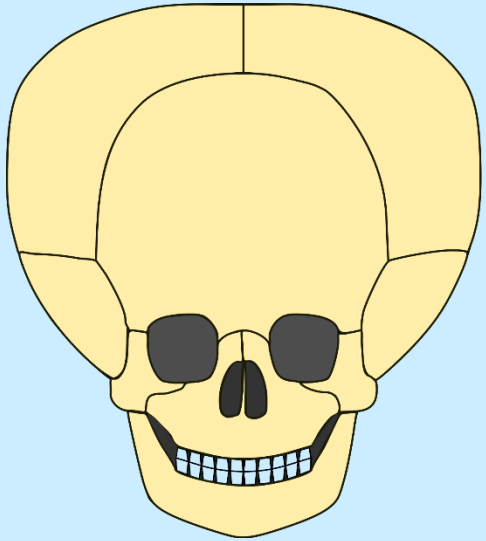
Hydrocephalus in children

- Causes like in adults + developmental malformation
  E.g. Arnold-Chiari malformation = deviation of the cerebellum and oblongata into the spinal cord channel (+ other malformations in the posterior fossa)
  → compression of CSF communications



Arnold-Chiari malformation I

- In children below 2 years
  increase of head circumference



Brain edema

Brain edema

= general pathological response of the brain to various insults

- accumulation of water and Na^+
- generalized or focal
- accompanies:
  - hypoxia
  - brain infarction
  - bleeding
  - inflammation
  - brain tumors
  - brain trauma
  - intoxication
  - metabolic disorders
  - severe epileptic seizures
  - of water and mineral metabolism disorders

Model od brain edema:

- administration of distilled water and ADH (not only brain edema but also edema of other organs)

Brain edema classification:

1) vasogenic – extracellular

- disorder of vessel wall permeability – disorders of blood-brain barrier
  - brain infarction, brain trauma, tumor surrounding (pathologic angiogenesis, ischemia due to compression of vessels by growing tumor), brain bleeding, inflammation

  → invasion of plasmatic proteins into the interstitium → water sucking
- mainly in the white matter

2) cytotoxic - intracellular

- lack of energy (from any reason) → reduction of Na^+-K^+-ATPase activity → ↑ of intracellular Na^+ and water
- brain ischemia (lack of energy), purulent meningitis (arachidonic acid released by inflammatory reaction, damage of cell membrane by bacterial toxins), intoxication (deterioration of cell membrane and metabolism), metabolic disorders (endogenous toxins – e.g. ammonia at hepatic failure)

3) Interstitial - extracellular

- in obstructive hydrocephalus, increase of hydrostatic pressure in the brain ventricles → CSF permeation into the periventricular brain tissue

4) Combination of 1-3

Brain edema

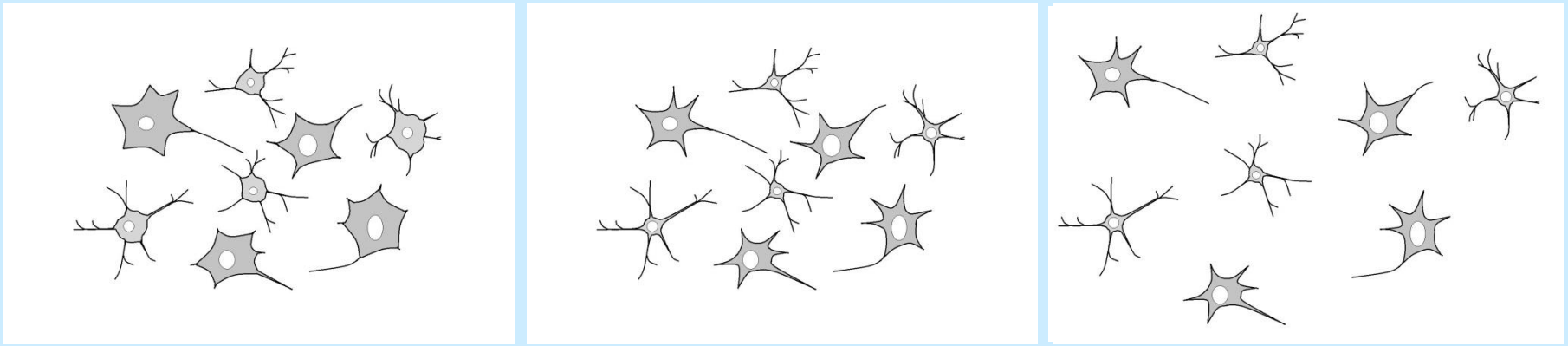
→ intracranial hypertension

→ brain herniations

→ decrease of perfusion pressure

→ in extracellular edema increase of extracellular space → prolongation of diffusion trajectory for oxygen, nutrients, catabolites and signaling molecules

intracellular edema | normal state | extracellular edema



Therapy:

- fast dehydration (restriction of fluid intake, diuretics, i.v. infusion of hypertonic solutions – e.g. 40% glucose, manitol)
- glucocorticoids
- decompression craniotomy

Blood-brain barrier disorders (HEB)

HEB = endothelium of brain capillaries (tight junction), basal membrane, astrocyte projections (astrocytic feet), pericytes (type of microglia)

HEB permeability

- most substances cross the BBB due to concentration gradient – dependent on plasmatic level
- lipid soluble substances pass BBB easy (O_2, CO_2)
- specific transporters (glucose, amino acids)
- water passes easy → osmotic equilibrium between the brain and blood
- ethanol, heroin, nicotine pass easy
- ions need channels or transport mechanism

Slow penetration of ions and some other substances with high osmotic activity and good permeability for water could lead to brain edema after rapid decrease of plasma osmolality (fast correction of diabetic hyperosmolar coma, dialysis).

Lower efficiency normally in periventricular areas, neurohypophysis

Disorders: tumors, injury, ischemia, hemorrhage, inflammation...

Intracranial hemorrhage

Epidural hemorrhage

- Into epidural space form meningeal arteries and veins, venous sinuses
- Due to head injury

Subdural hemorrhage

- After head injury, often with skull fracture, venous bleeding
- Acute - hours-days after the injury
- Subacute – more days
- Chronic – in aged people, often after mild injury

Subarachnoid hemorrhage

- Rupture of an aneurysm, often related to arterial hypertension

Intracerebral hemorrhage

- Arterial hypertension + atherosclerosis
- Usually aa. perforantes (bleeding into the thalamus, basal ggl., white matter - capsula interna)

THE END