COMA
&
INTENSIVE CARE

Jozef Firment, MD. PhD.,
Judita Capkova, MD. PhD.

Department of
Anaesthesiology & Intensive Care Medicine
Šafárik University Faculty of Medicine, Košice

DEFINITION

- Victim is unresponsive to verbal stimuli

Quantitative:

- GCS <8 points
- Somnolence – stupor – coma

Qualitative:

- *Vigilant coma (coma depassé, apalic sy)*

COMA - PATIENT EXAMINATION

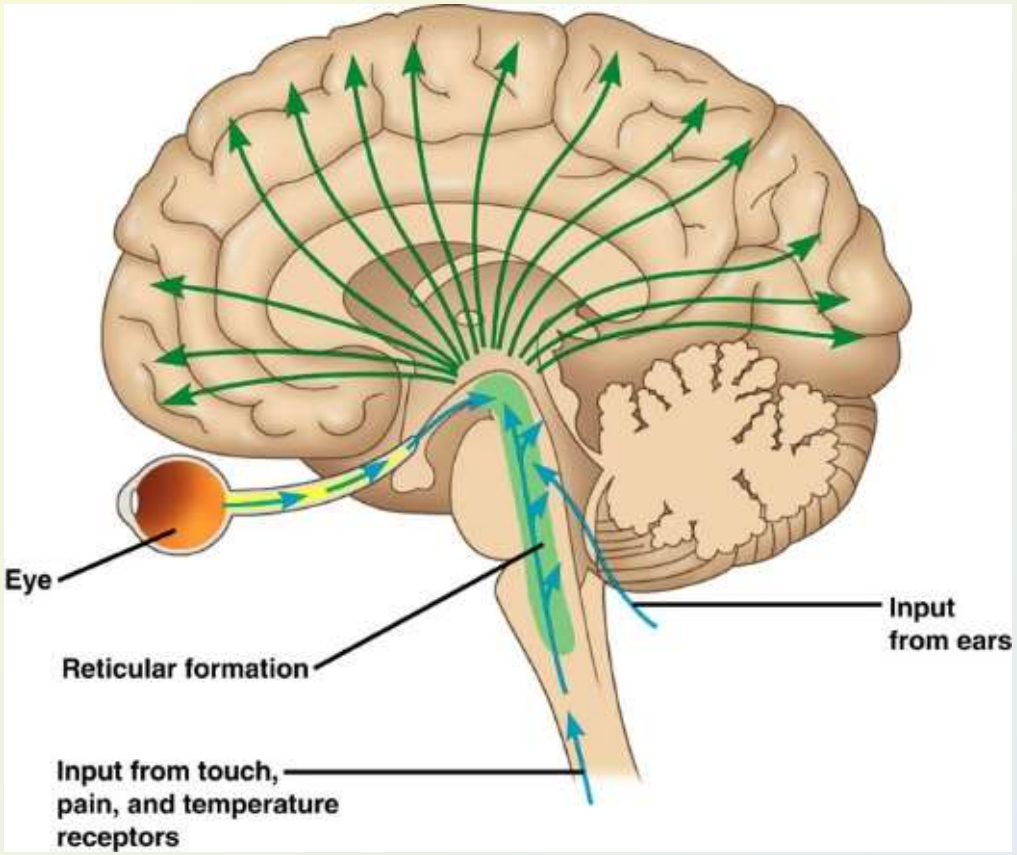
1. **Causalities - history**
2. **Clinical examinations**

- Patient **responded** to requests, contacts, painful stimuli.
- **Performance:** Injuries, foetor, convulsions, body position, ocular signs, head stiffness, lateralisation, fever, breathing, signs of organ failure... BP, P

3. **SpO_2**
4. **Laboratory** tests: Glycaemia, K, Na, Hb, ABG, BUN, creatinine, AST, ALT, osmolality, toxicology
5. Brain **CT**...

Activatory system of Reticular formation (RF)

- Transmission of several sensoric inputs
- Facilitating of **vigilance**



GLASGOW COMA SCALE

Glasgow Coma Scale		Score
Eye opening (E)	Spontaneous	4
	To voice	3
	To pain	2
	None	1
Verbal response (V)	Oriented	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1
Best motor response (M) *in the upper limbs*	Follows commands	6
	Localises pain	5
	Withdraws from pain	4
	Abnormal flexion	3
	Abnormal extension	2
	None	1

(E+M+V)

Score 3-8 points = severe head injury.

Score 9-12 points = moderate head injury.

Score 13-15 points = mild head injury.

Coma without focal (lateralizing) neurological signs

- Anoxia / hypoperfusion
- Metabolic: e.g. Hypo/-hyperglycaemia, acidosis/alkalosis, hepatic or renal failure
- Intoxications: e.g. alcohol, opioids, benzodiazepines,..
- Endocrine : hypothyreoidism
- Hypo- or hyperthermia
- Epilepsy
- Hypertensive encephalopathy...

Coma with focal (lateralizing) neurological signs

(due to brainstem or cerebral dysfunction)

- Vascular: cerebral haemorrhage or infarction
- Supra or infratentorial space-occupying lesion: tumour, haematoma, abscess

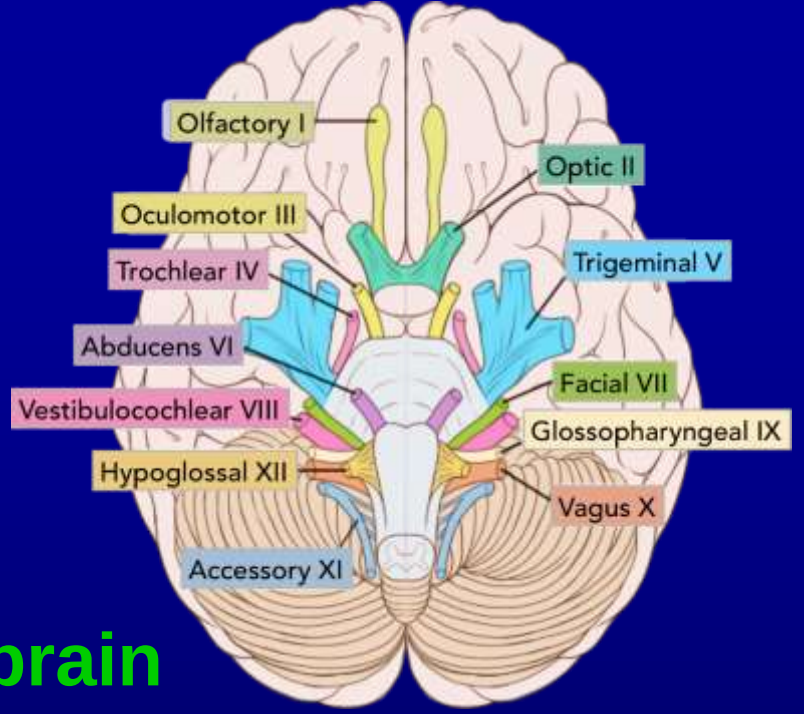
Coma with meningism

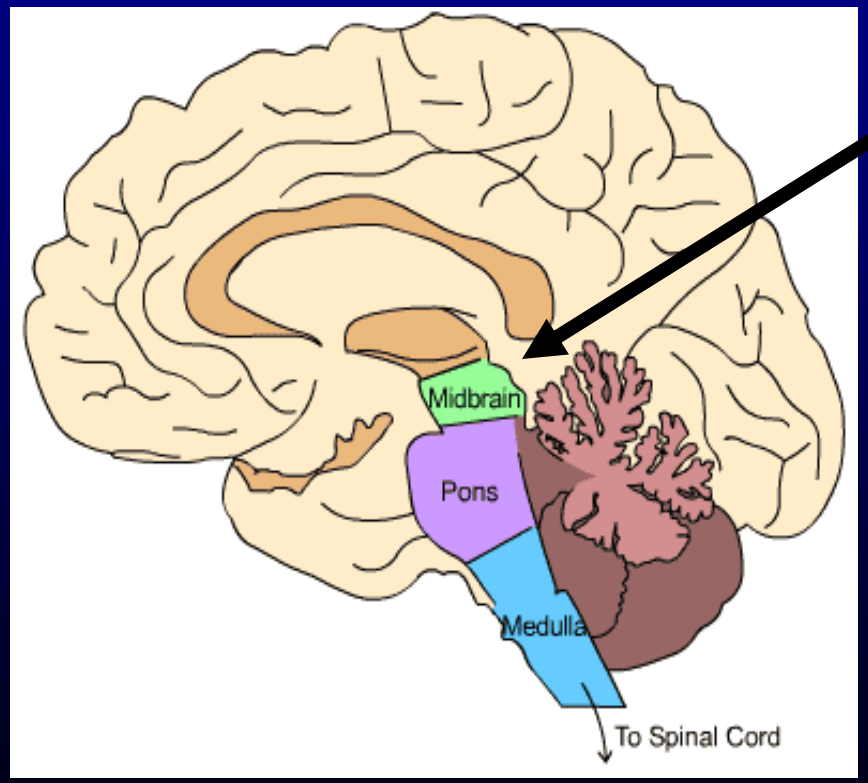
- Meningitis, encephalitis
- Subarachnoid haemorrhage

Test brainstem dysfunction

- Pupillary response
- Corneal reflex
- Spontaneous eye movements
- Oculocephalic response/Doll’s head manoeuvre
- Oculovestibular response
- Swallowing
- Respiratory pattern

Brain Stem



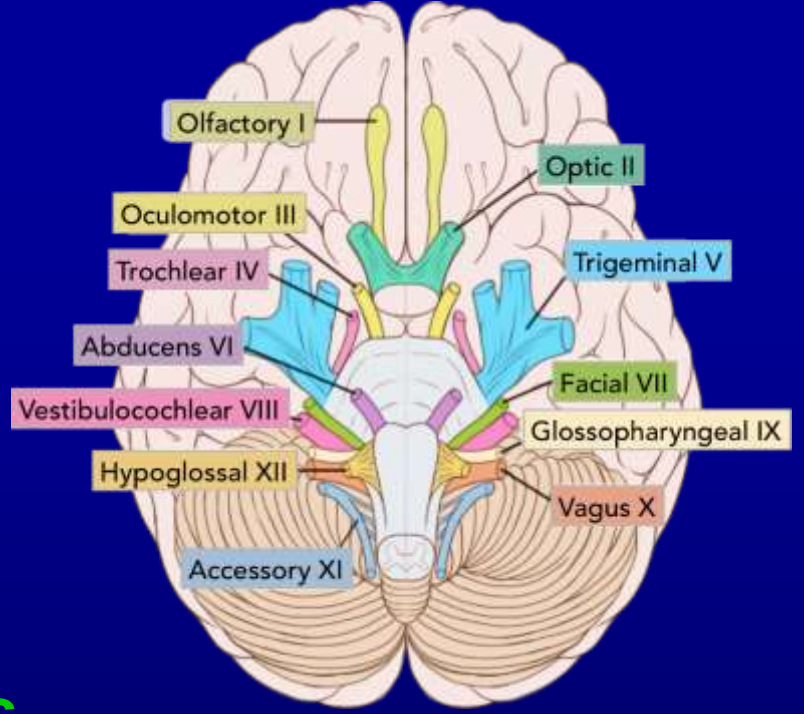


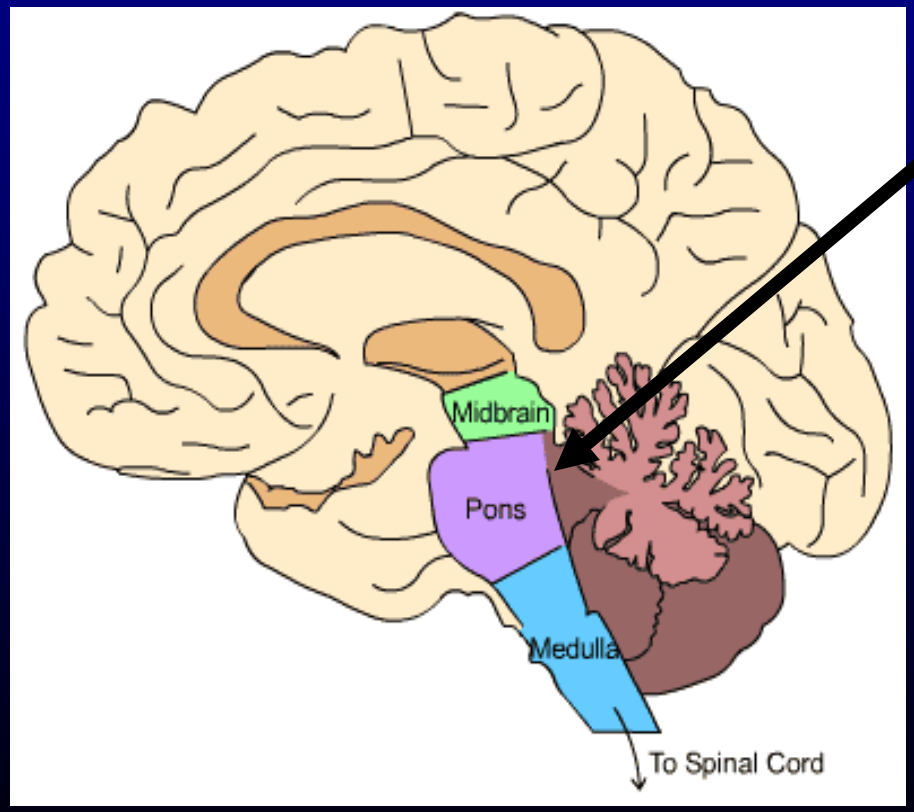
Midbrain

III brain nerve

- pupils
- eye movement

Brain Stem



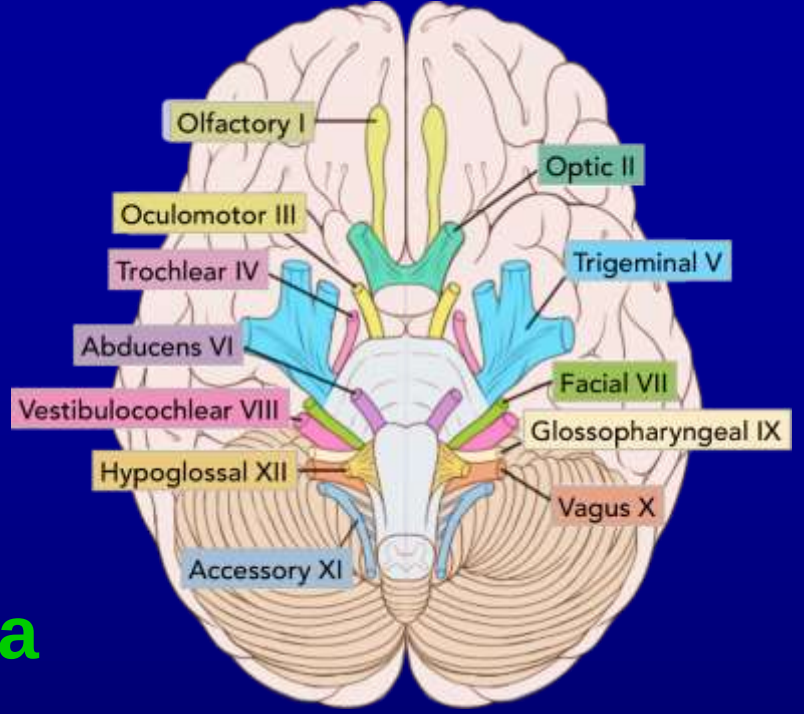


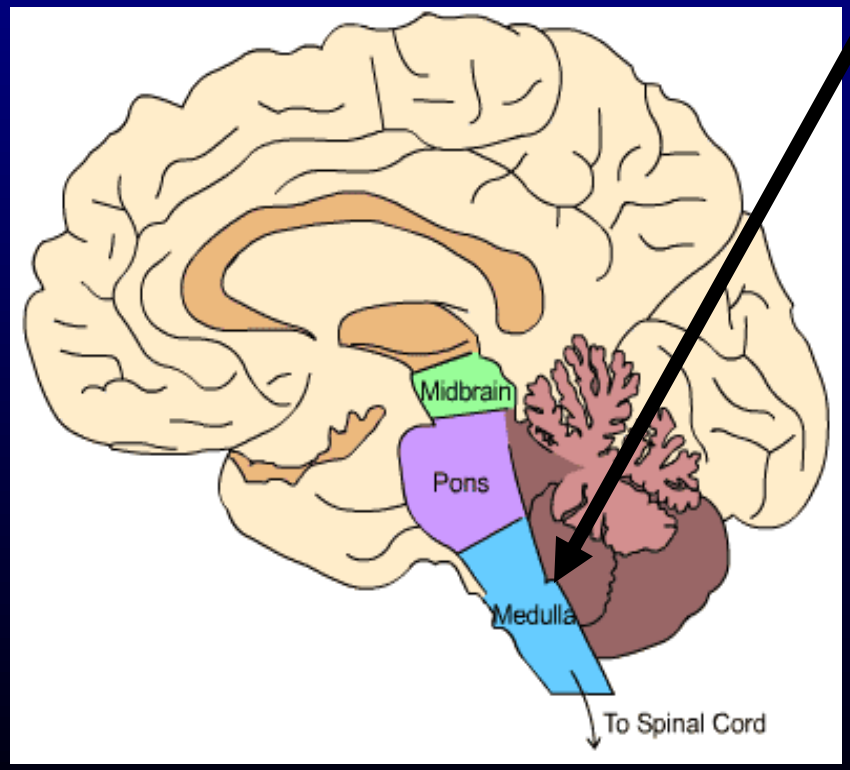
Pons

brain nerves IV, V, VI

- conjugate movement of eyeballs
- corneal r.

Brain Stem





Medulla

brain nerves IX, X

- Gag reflex
- Tracheal rr. (cough)

Respiratory centre!

Plan for further investigations:

1. Brainstem function intact:

urgent CT head scan :

- **lesions** (subdural haematoma,..),
- **normal** – lumbar puncture, CSF analysis

2. Brainstem function not intact:

- if **herniation syndrome** appears to be progressing **rapidly** - mannitol, HS, hyperventilation, surgeon

- if **herniation syndrome** appears to be progressing not so **rapidly** – mannitol, HS and CT

3. Consider giving thiamine, glucose (40 ml 40% glucose), naloxon, flumazenil

4. Examine patient:

- Core temperature, heart rate, rhythm, BP, respiratory pattern, breath, skin, heart, abdomen,
- Is there meningism?
- Asses GCS
- Look for evidence of brainstem dysfunction
- Are there lateralizing signs?

Head injury (HI)

- Primary brain injury: the neurones lost at the time of HI are lost forever (direct result of trauma)

- Causes of secondary brain injury:

Systemic:
- Hypoxaemia
- Hypotension
- Hypercarbia
- Severe hypocapnia
- Pyrexia..

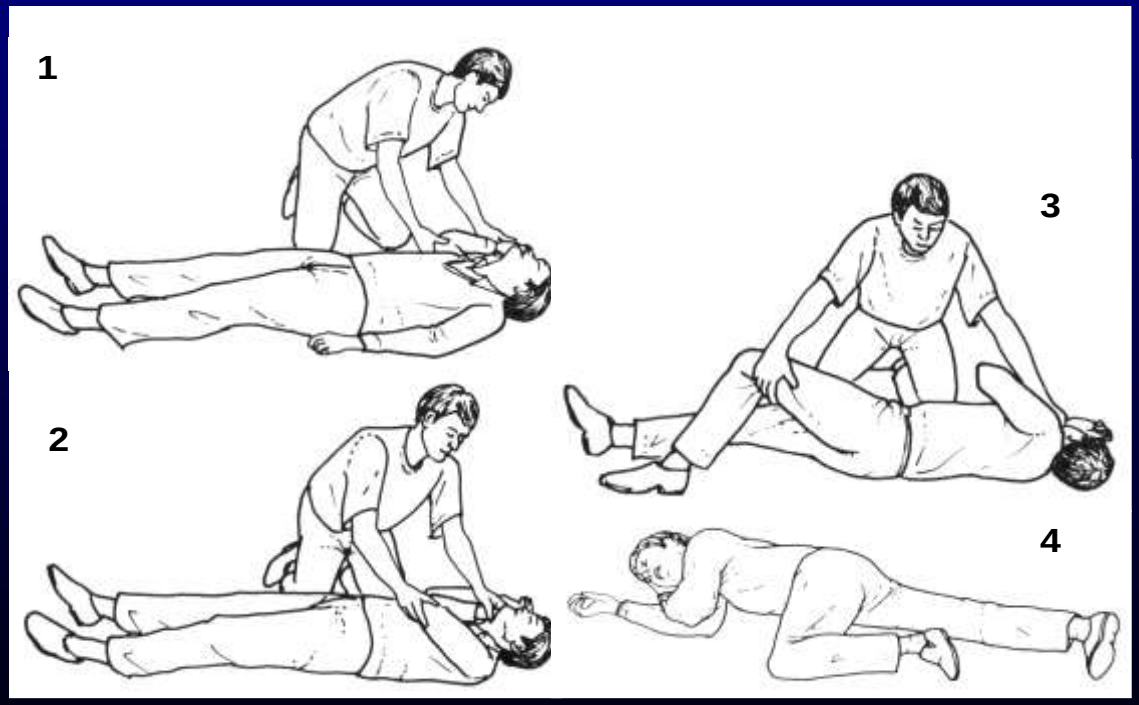
Intracranial:
- Haematoma (extradural, subdural, intracerebral)
- Brain swelling / oedema
- Raised ICP...

Prevention of secondary injury

is the aim of the treatment !

FIRST AID IN COMA

- **ABC** CPR
- **Recovery** (lateral) position



CEREBRAL OEDEMA

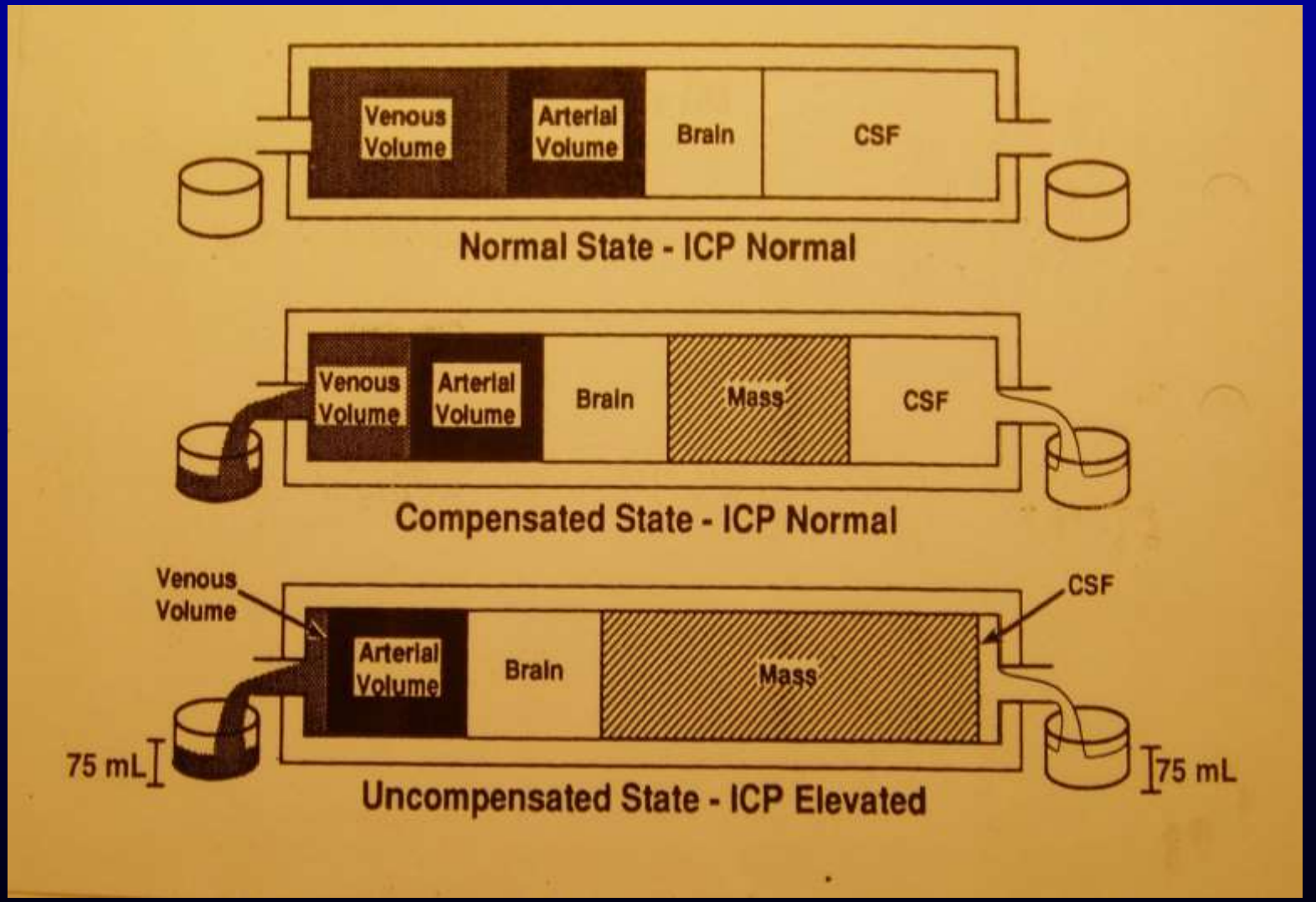
- Signifies an increase in the **brain water** content.
  There are three different types of cerebral oedema - vasogenic, cytotoxic, and interstitial (CBF).
- Increased cerebral **blood volume** (congestion).

	Vasogenic	*Cytotoxic*	*Interstitial*
Pathophysiology	BBB defect due to parenchymal necrosis	Toxic cell defects	Increased intraventricular pressure
Vessel permeability	Increased	Unchanged	Unchanged
Oedema fluid	Protein-rich	None	Low protein content
Morphology	Enlarged extracellular space in the white matter	Cell swelling	Enlarged extracellular space in the periventricular matter

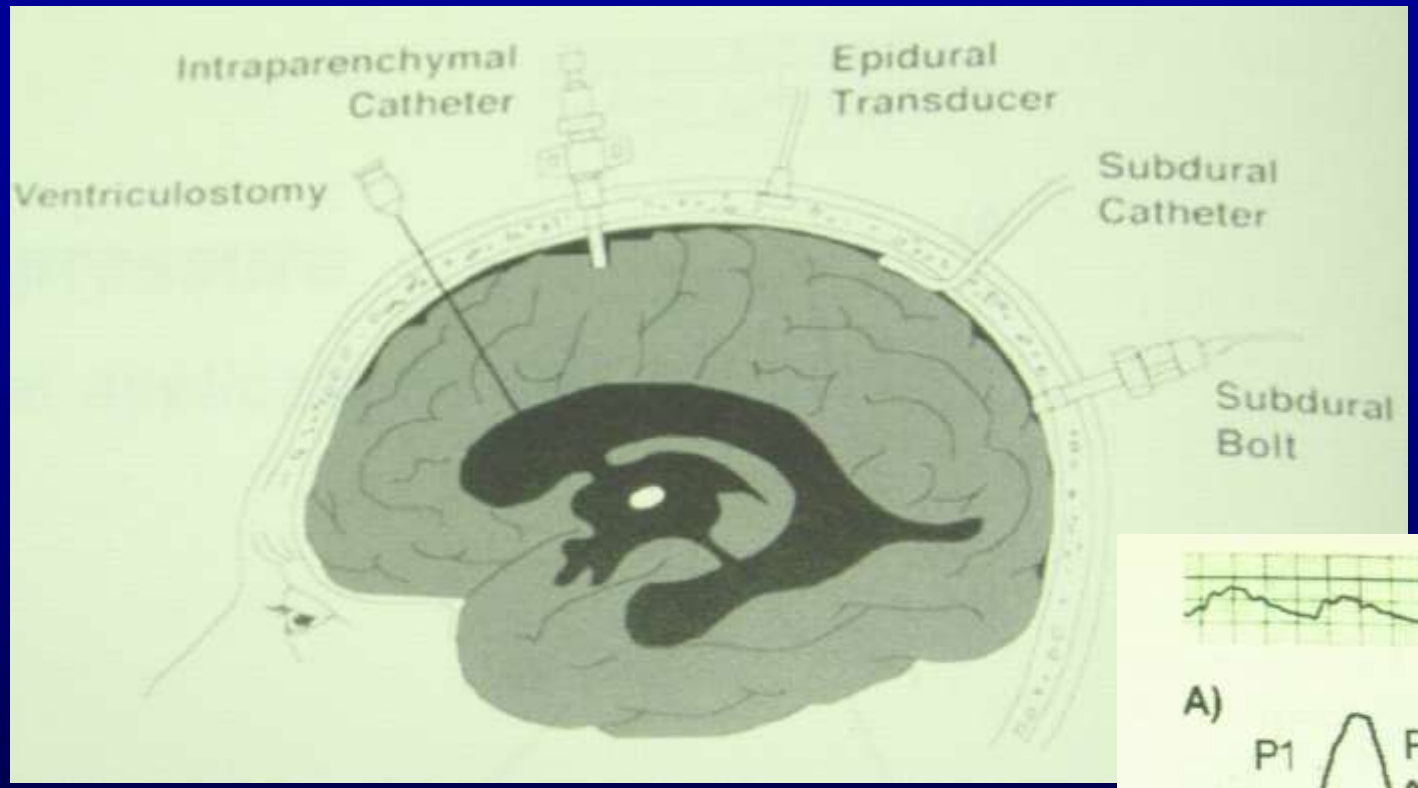
BBB – Blood Brain Barrier

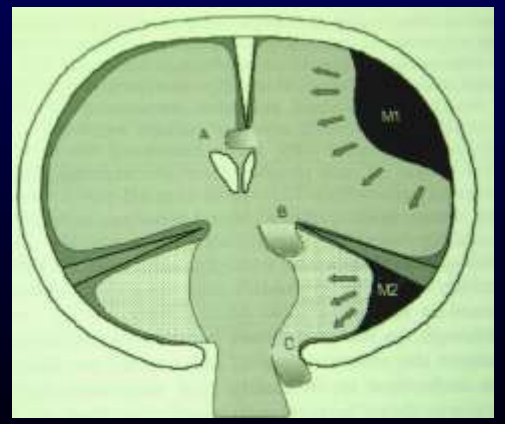
Patients with head injuries usually have a mixed type of oedema: vasogenic and cytotoxic.

INTRACRANIAL COMPENSATION FOR EXPANDING MASS



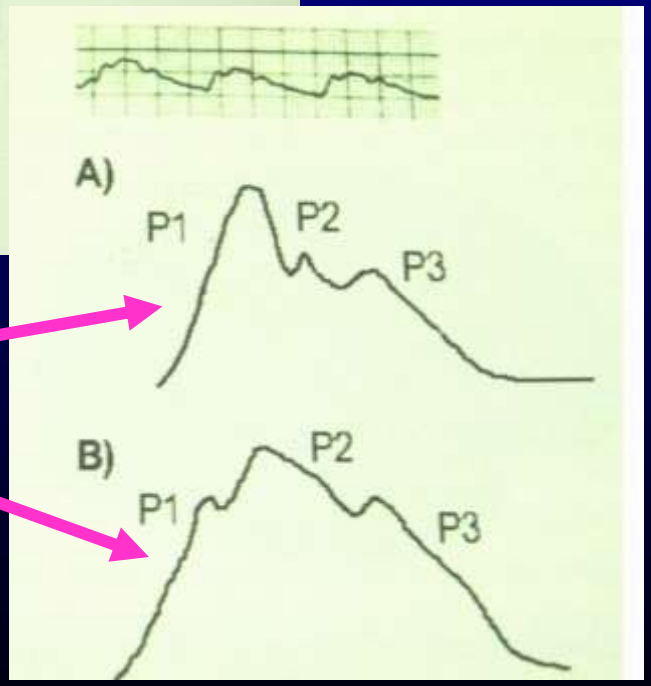
INTRACRANIAL PRESSURE





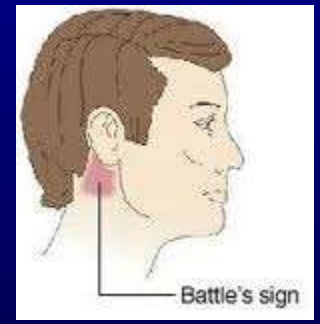
Normal curve shape

Low compliance



Early indications for head CT in adults TBI

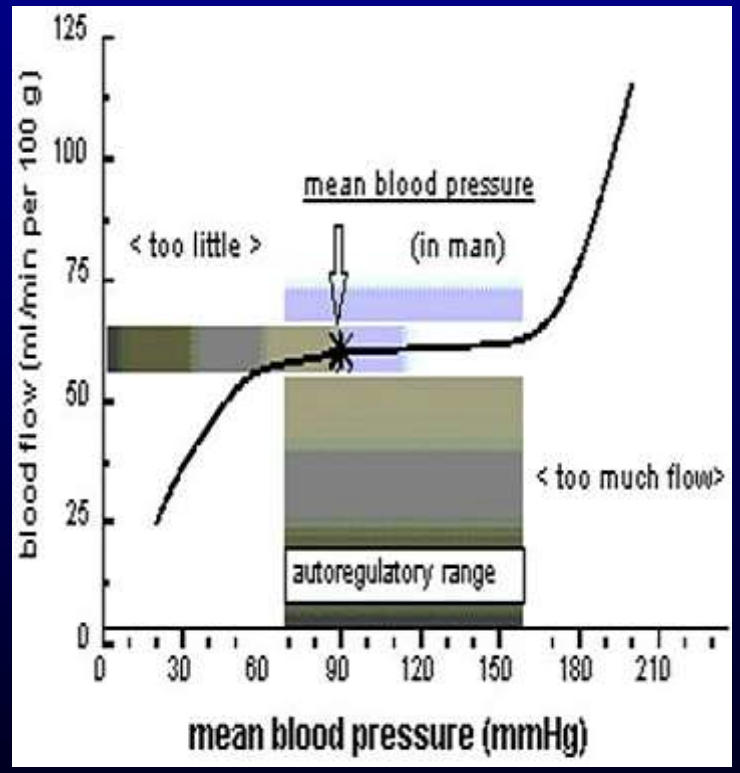
1. **GCS < 13** on initial assessment in the emergency department
2. **GCS < 15, 2 h** after the injury on assessment in the emergency department
3. Suspected open or depressed **skull fracture**
4. Any sign of **basal skull fracture** (haemotympanum, ‘panda’ eyes, cerebrospinal fluid leakage from the ear or nose, Battle’s sign)
5. Post-traumatic **seizure**
6. Focal neurological **deficit**
7. More than one episode of **vomiting**
8. **Amnesia** for events > 30 min before impact
9. Any patient who has experienced some **loss of consciousness** or amnesia since the injury **and**:
   - is aged 65 years or older
   - is at risk of coagulopathy (history of bleeding, clotting disorder, current treatment with warfarin)
   - there is a dangerous mechanism of injury (a pedestrian or cyclist struck by a motor vehicle, an occupant ejected from a motor vehicle or a fall from a height of > than 1 m or five stairs).



Protheroe RT, Gwinnutt CL: Early hospital care of severe traumatic brain injury. Anaesthesia, 2011, 66, 1035–1047

Aims of TBI treatment

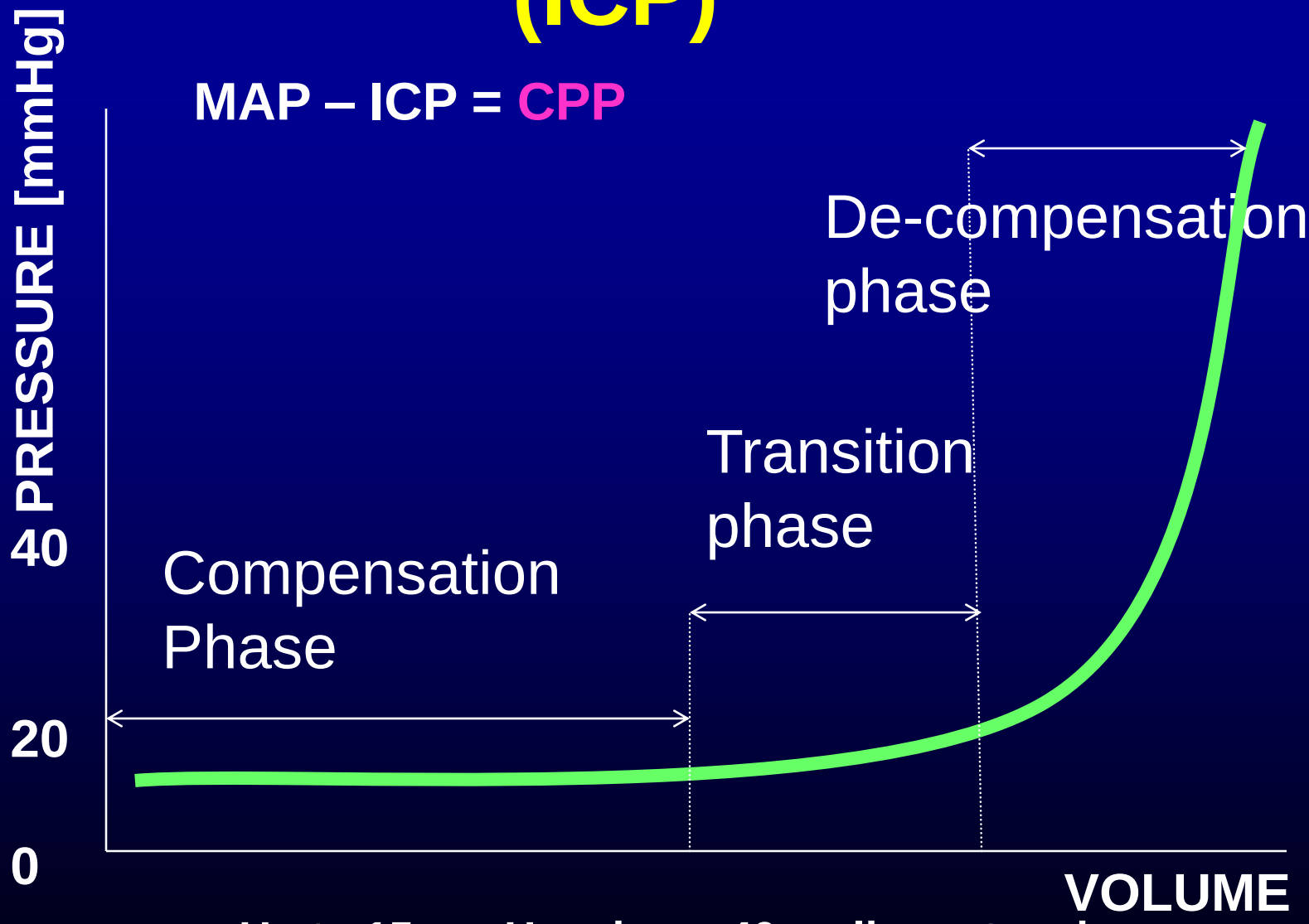
- To respect **primary** cerebral insult
- **Secondary** damage prophylaxis
- Cerebral oedema treatment
  (**ICP-targeted** therapy)
- Brain perfusion
  autoregulation disturbances
  (**CPP-targeted** therapy)
- ↑ **chance** for recovery



http://www.bioscience.org/2008/v13/af/2804/fulltext.asp?bframe=figures.htm&doi=yes

INTRACRANIAL PRESSURE (ICP)

MAP – ICP = CPP

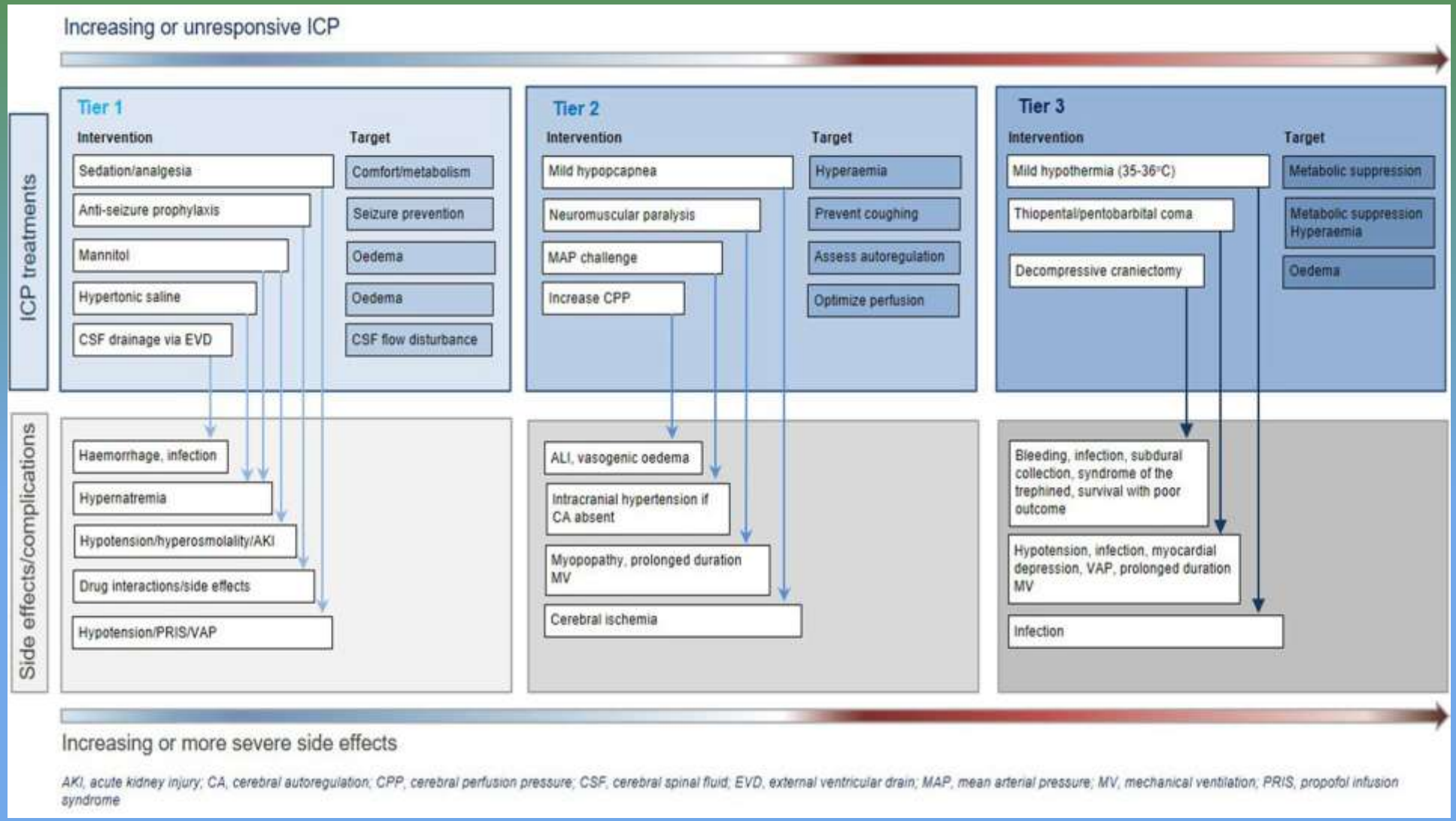


Up to 15 mmHg, above 40 malignant oedema

Raised ICP: immediate management

- Open the airway, intubation, mechanical ventilation, up to 6 hours keep P_aCO_2 4.0 – 5.3 kPa (30-40 mmHg)
- Correct hypotension: colloids, infusions of inotropes
- Treat seizures
- Take blood for glucose, U+Es, calcium, liver enzymes, albumin, clotting screen, FBC (full blood count)

Multilevel treatment of intracranial hypertension in TBI



Smith M, Maas AIR: An algorithm for patients with intracranial pressure monitoring: filling the gap between evidence and practice. Intensive Care Med, https://doi.org/10.1007/s00134-019-05818-4

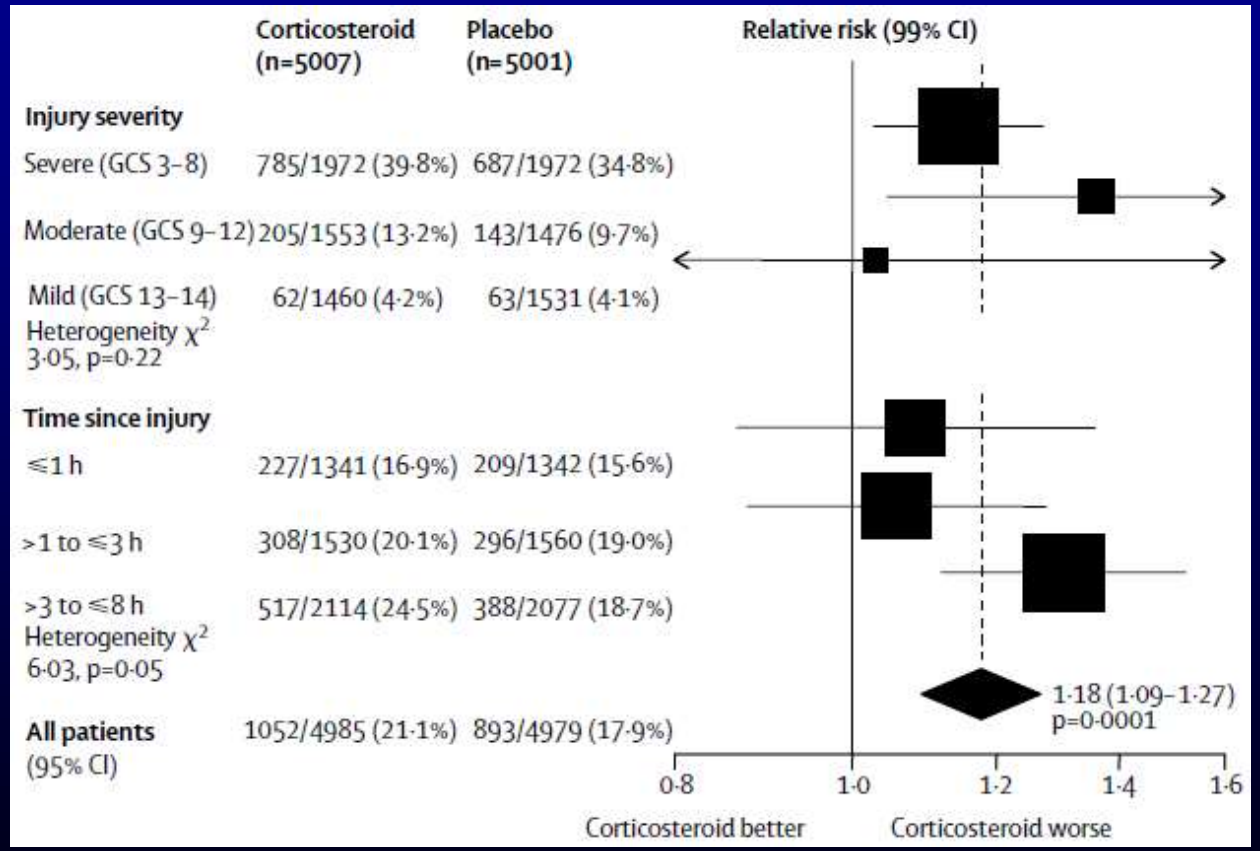
If MAP augmentation results in a reduction in ICP, confirming some degree of intact autoregulation.

TREATMENT OF TRAUMATIC BRAIN INJURY

- ABC CPR
- Ventilation ($paCO_2$, paO_2)
- Cerebral perfusion (BP, anaemia...)
- Head position (medial line,15-30º)
- Venous drainage from head
- Anti-oedematous therapy
- Mannitol 20% 0.25-1.0 g/kg bw
- Furosemide
- $PaCO_2$ 4.5 kPa
- $MgSO_4$
- Lidocain
- Hypertonic Saline (HS 3-25%)
- Convulsions, cough (sedation, analgesia...)
- Thiopental, propofol (artificial sleep, analgo-sedation, deep sedation)
- Decompressive craniectomy

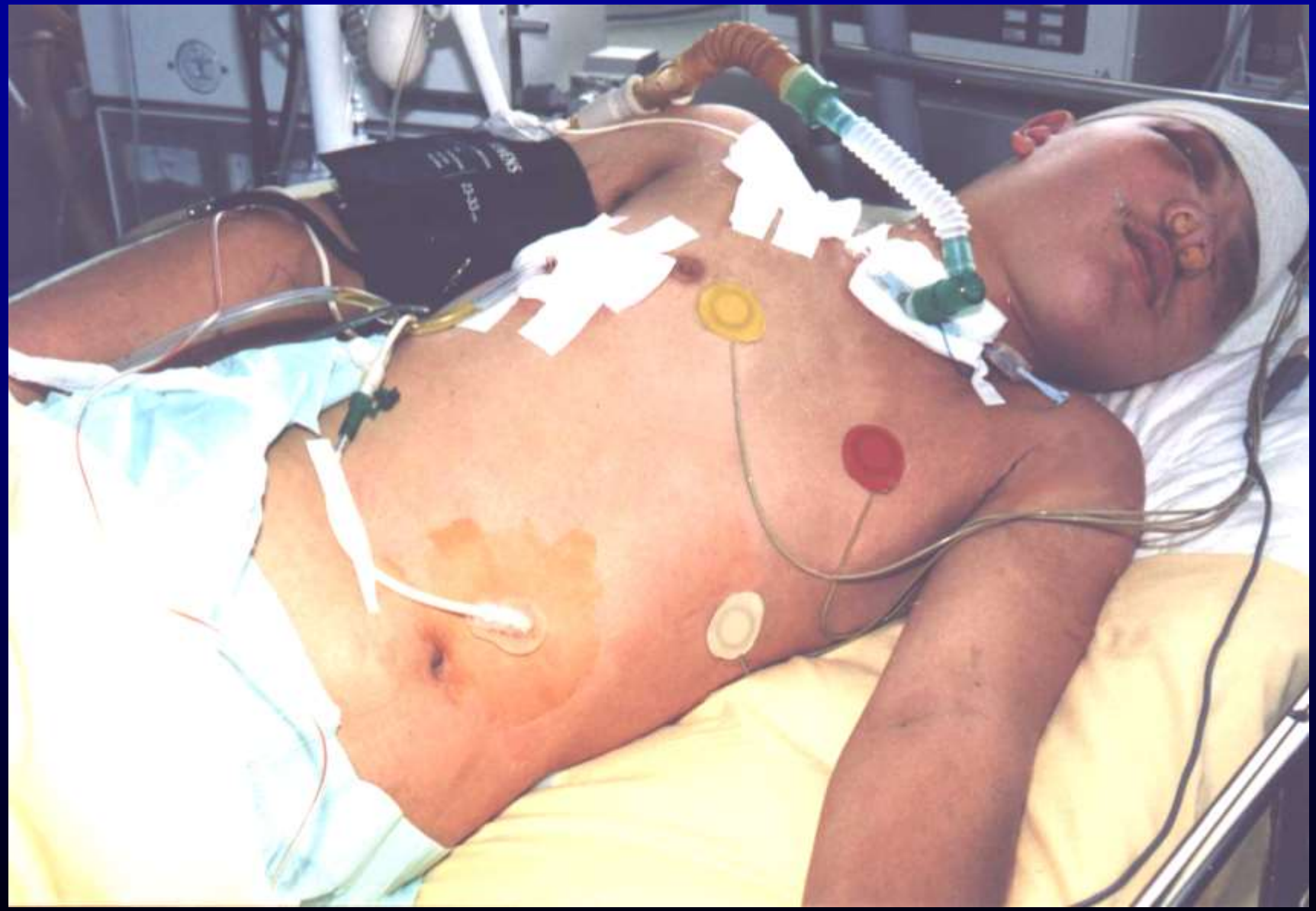
CRASH Trial – NO corticosteroids!

- **Risk of death** from all causes within 2 weeks was **higher in the group allocated corticosteroids** (1052 [21·1%] vs 893 [17·9%] deaths; relative risk 1·18 [95% CI 1·09–1·27]; p=0·0001).
- The relative increase in deaths due to corticosteroids **did not differ by injury severity** (p=0·22) or time since injury (p=0·05).



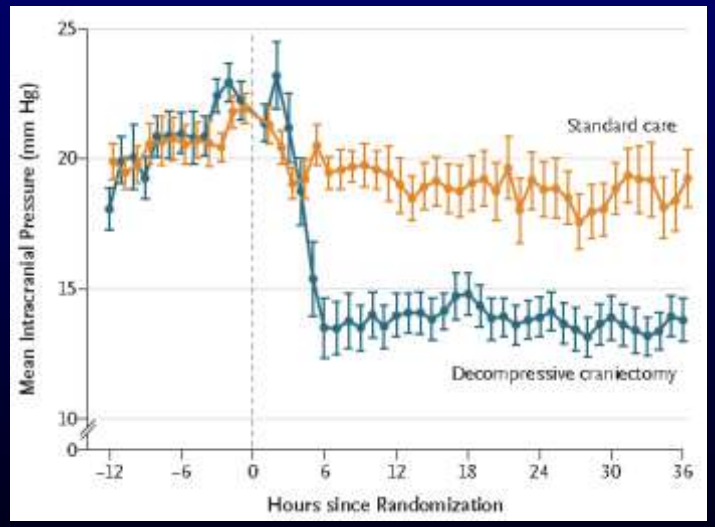
CRASH Trial Collaborators. Effect of intravenous corticosteroids on death within 14 days in 10 008 adults with clinically significant head injury (MRC CRASH trial): randomised placebo-controlled trial. Lancet 2004;364:1321-8.

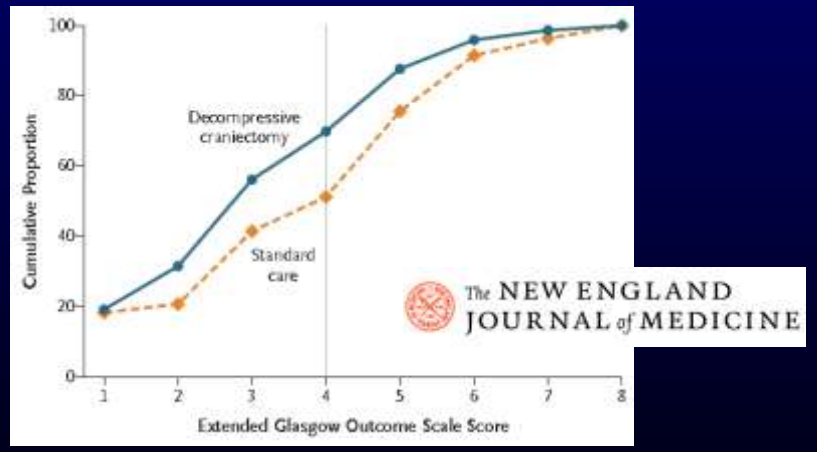
TBI, maxillofacial injury, haemothorax
Tracheostomy – artificial ventilation,
PEG, thoracic drainage



Decopmressive craniectomy

- In adults with severe diffuse traumatic brain injury and refractory intracranial hypertension,
- early bifrontotemporoparietal decompressive craniectomy decreased intracranial pressure and the length of stay in the ICU
- but was associated with more unfavourable outcomes.





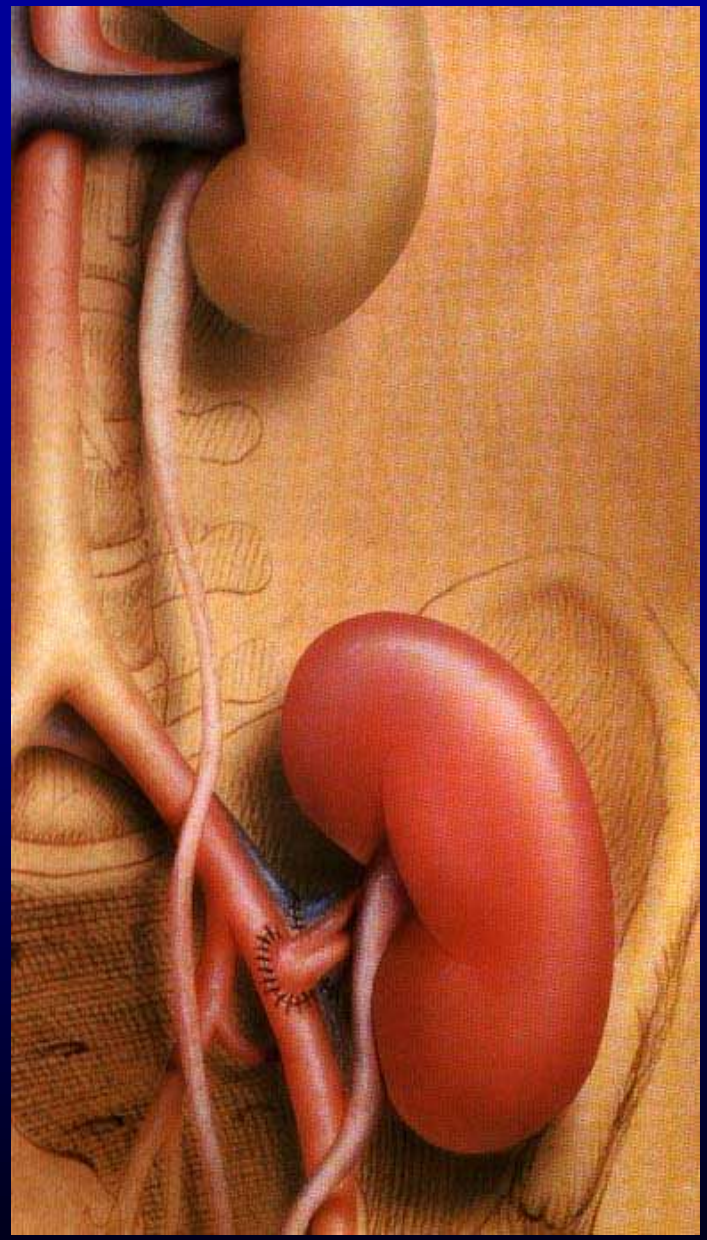
Cooper DJ et al. Decompressive Craniectomy in Diffuse Traumatic Brain Injury. N Engl J Med 2011; 364: 1493-1502

ORGAN DONATION

Jozef Firment MD PhD

Department of
Anaesthesiology & Intensive Care Medicine
Šafárik University Faculty of Medicine,
Košice

Please: „Don't take your organs to heaven ..., heaven knows we need them here" on Earth.



Brain death

- Is irreversible loss of the capacity for consciousness combined with irreversible loss of capacity to breathe

Preconditions

- No doubt that pt. has structural brain damage which has been diagnosed
- Pt must be in apnoeic coma (on mechanical ventilator)
- No possibility of drug intoxication, no significant metabolic, endocrine, electrolyte disturbance

CLINICAL BENCHMARKS FOR BRAIN DEATH

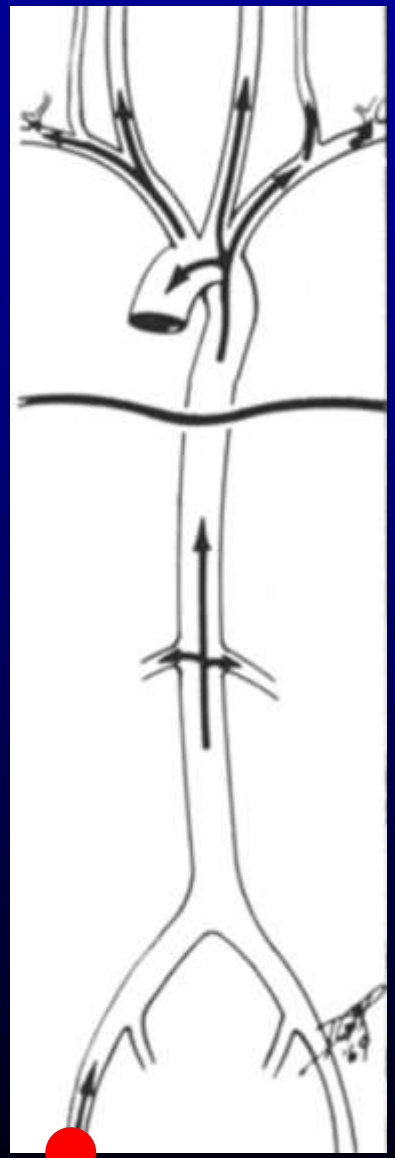
1. **Coma,** GCS = 3, known irreversible **cerebral** lesion (Cave: intoxication, metabolic disturbances, hypothermia, myorelaxants). Absent functions of brain stem
2. **Apnoea test** – no respiratory movements when the ventilator is disconnected and P_aCO_2 reaches 6,6 kPa
3. Lack rr of **all 12 cerebral nerves**
4. No spontaneous muscle movements (**spinal** rr sometimes are present)
5. **Absent panangiographic cerebral perfusion!**
   Panangiography is confirmation test (no mandatory in Slovakia)

Testing for brain death

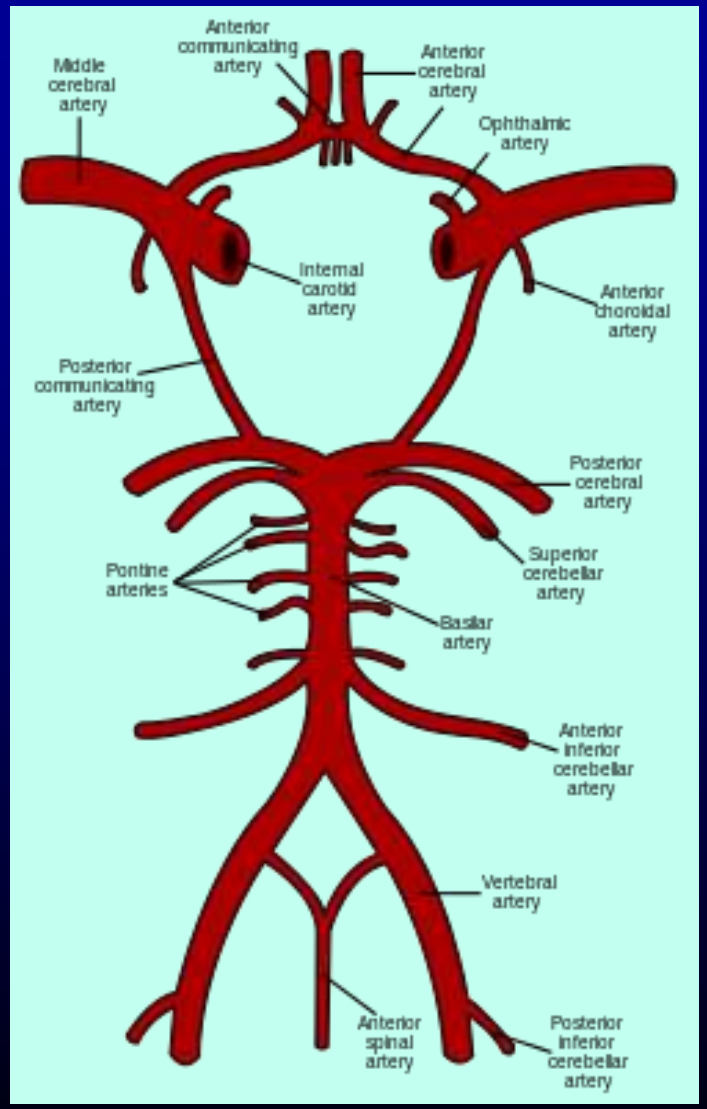
All brainstem reflexes must be absent (12 pairs)

- Pupils fixed and unresponsive to bright light
- Absent cornel reflexes
- Absent vestibulo-ocular reflexes
- No motor response within the cranial nerve distribution
- No reflex response to touching the pharynx, nor to a suction catheter passed into the trachea

Test for confirmation of brain death



Pan-angiography of cerebral vessels – after administration of contrast liquid into all 4 aa. - no perfusion



Brain death

- **Brain death** = **death of person**
- Brain death versus cardiac death
- Organ donation from **deceased donors (HBD - NHBD)**.
- Relatives organ donation from living donors.
- **Opting out**

  anyone who has not refused is a donor (Slovakia)
- **Opting in**

  anyone who has not given consent is not a donor

Brain death protocol

- 3-personal council
- Neurological status
- 12 h without sedation, relaxation
- Known brain lesion

- Disconnection from ventilator

DONOR MONITORING

- Continual EKG
- NIBP á 10 min, CVT á 4-6 h,
- Diuresis/h, fluid balance
- K, Na, ABG á 4 h, SpO_2, (E_TCO_2)
- Body temperature (core)
- BP measurement invasive

Tx PROGRAM

ORGANS

- Kidney
- Heart
- Liver
- Lungs
- Pancreas

TISSUES

- (Blood)
- (Bone marrow)
- Cornea
- Bones