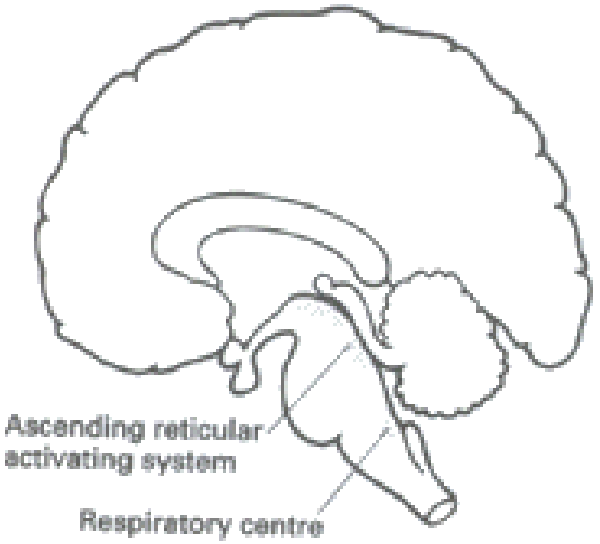
Consciousness

- **Vigilance**
- The ability to maintain attention and alertness over prolonged periods of time
- **Individual is fully responsive to stimuli, this is the condition of the person when awake.**
- **Activity of ARAS (ascending reticular activating system)**



Unconsciousness

A state of unawareness of self and environment. One shows no responsiveness to environmental stimuli but may respond to deep pain with involuntary movements.

Unconsciousness

- **Somnolencia –** ("drowsiness„) is a state of near-sleep, a strong desire for sleep, or sleeping for unusually long periods.
- **Sopor/stupor-** is an unresponsive state from which a person can be aroused only briefly and with vigorous, repeated attempts.
- **Coma-** is a profound state of unconsciousness.
  - a comatose patient cannot be awakened
  - fails to respond normally to pain or light
  - does not have sleep-wake cycles
  - does not take voluntary actions.
  - coma can last days, weeks, months, or indefinitely
  - the length of a coma cannot be accurately predicted or known
  - coma results from gross impairment of both cerebral hemispheres, and/or the ascending reticular activating system.

Unconsciousness

- **Deep unconsciousness – absent brain stem reflexes (corneal, pupillar, pharyngeal), tendom reflexes, muscle hypotonia, spontaneous breathing is absent**

- **Mild unconsciousness – brain stem reflexes +-, increases muscle tone, spontaneous breathing is present – different pathology**

Unconsciousness

- **Acute**

  a/ lesion in brain stem

  b/ metabolic reason

Unconsciousness

1. Consciousness
2. Breathing
3. Pupils
4. Position and movements of eyes
5. Muscle tone, motor functions
6. Brain stem reflexes

Glasgow coma scale

- **Maximum – 15 points**
- **More than 8 points – better prognosis**
- **Less than 7 points – worse prognosis**

Glassgow coma scale
GCS

- **This scale gives a simple measure of the degree of unconsciousness, but disregards other information that may be available**

- **This looks at eye activity, verbal and motor responses, and assigns points for each to give a composite score**
- **3 points- being deeply unconscious**
- **15 points- being fully conscious**

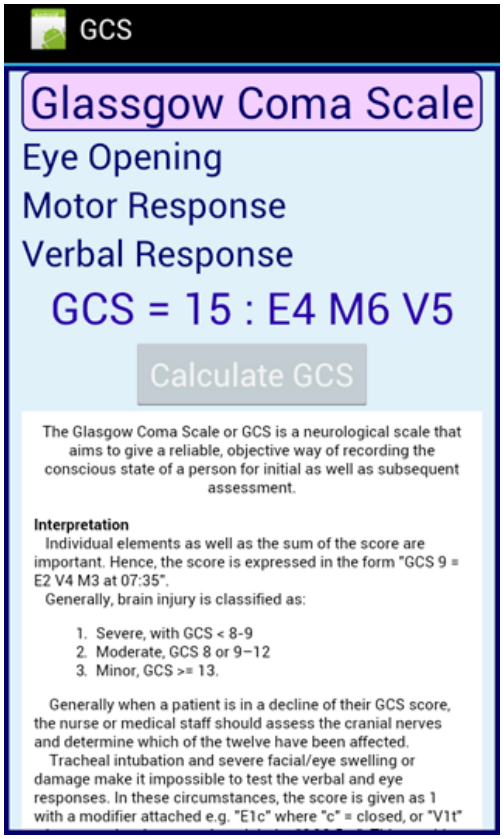
- **More than 8 points – better prognosis**

- **Less than 7 points – worsen prognosis**

Glasgow coma scale

Eye Opening Response

- Spontaneous--open with blinking at baseline **4 points**
- To verbal stimuli, command, speech **3 points**
- To pain only (not applied to face) **2 points**
- No response **1 point**



Glasgow coma scale

Verbal Response

- Oriented **5 points**
- Confused conversation, but able to answer questions **4 points**
- Inappropriate words **3 points**
- Incomprehensible speech **2 points**
- No response **1 point**

Glasgow coma scale

Motor Response

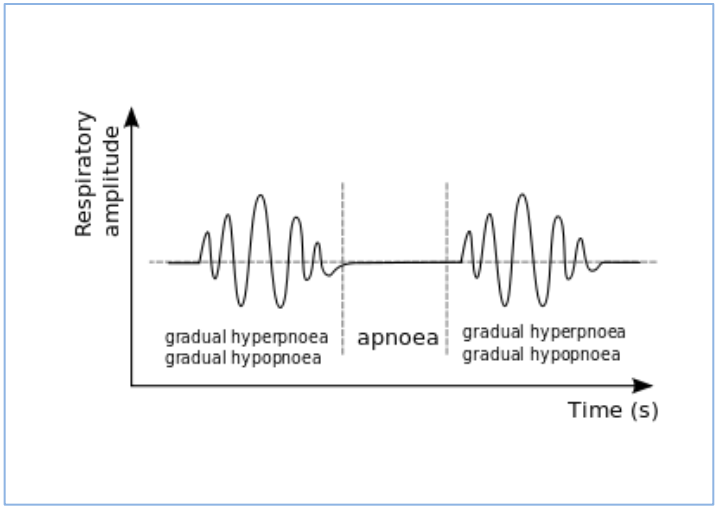
- Obeys commands for movement **6 points**
- Purposeful movement to painful stimulus **5 points**
- Withdraws in response to pain **4 points**
- Flexion in response to pain (decorticate posturing) **3 points**
- Extension response in response to pain (decerebrate posturing) **2 points**
- No response **1 point**

- ***GCS- is most useful in allowing the assessment of <u>changing levels</u> of consciousness, either improvement or deterioration.***

- ***A worsening of the GCS in a head injured patient indicates the need for urgent neurosurgical intervention.***

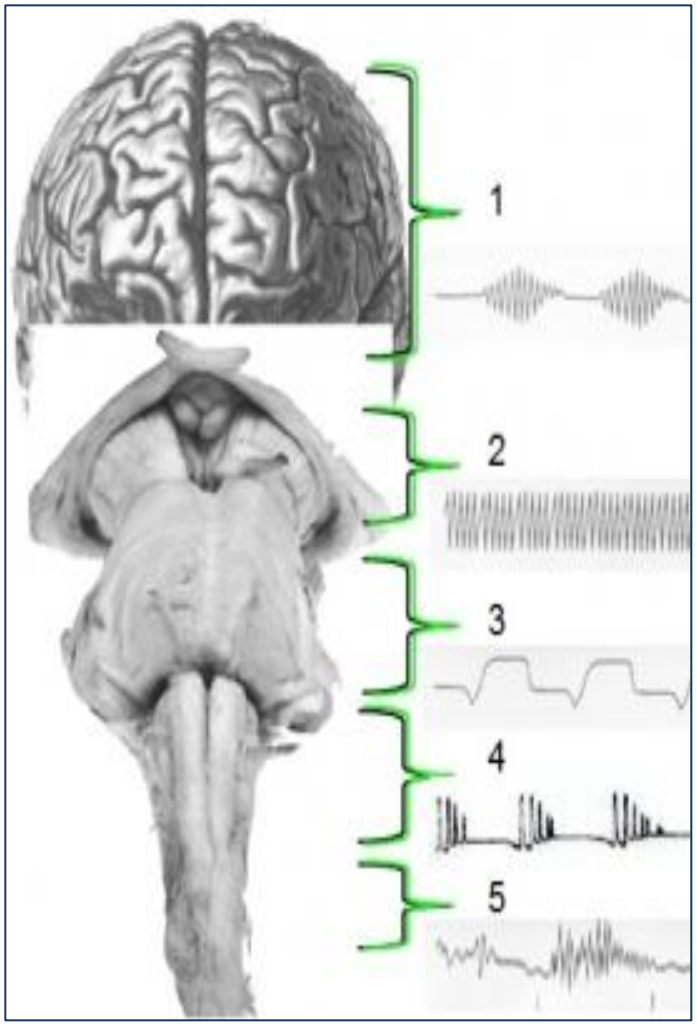
Breathing

- Frequency
- Quality
- Regularity



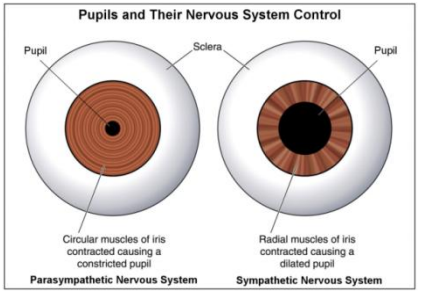
- **Posthyperventilation apnoe** – high CO_2 level stimulates breathing =HV
- HV leads to reduction of CO_2 = respiration centre depression = <u>apnoe</u>

Breathing

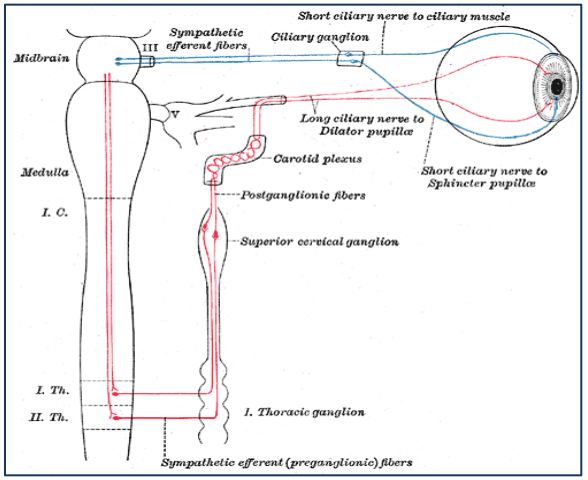


- Bilat. hemispheral and diencephalic lesions – Cheyne-Stokes breathing – changing of hyperpnoe and apnoe
- Mesencephalic lesions – central neurogenic hyperventilation
- Lesions in pons – apneusis
- Medulla oblongata lesions – ataxic breathing – irregular breathing

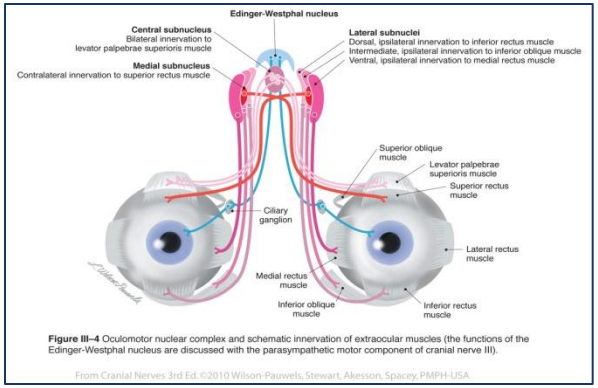
Pupils



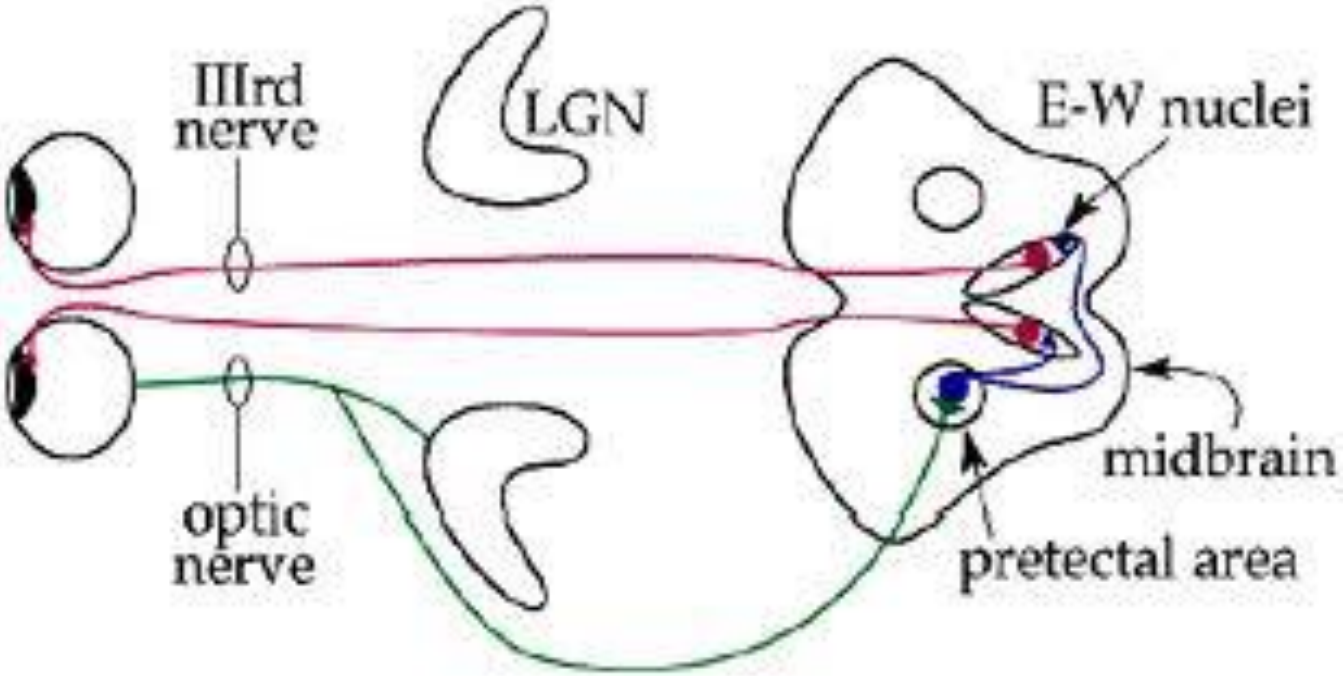
- **Sympatic inervation:** hypotalamus, homolateral brain stem, C- spinal cord, upper segments of Th-spinal cord, preganglionic fibers, 3 Th-roots to cervical ganglia, with a. ophtalmica to nc. dilatator pupillae



- **Parasympatic inervation:** W-E nuclei (III), ggl. ciliare, m. sphincter pupillae



Pupils


IIIrd nerve
LGN
E-W nuclei
midbrain
optic nerve
pretectal area

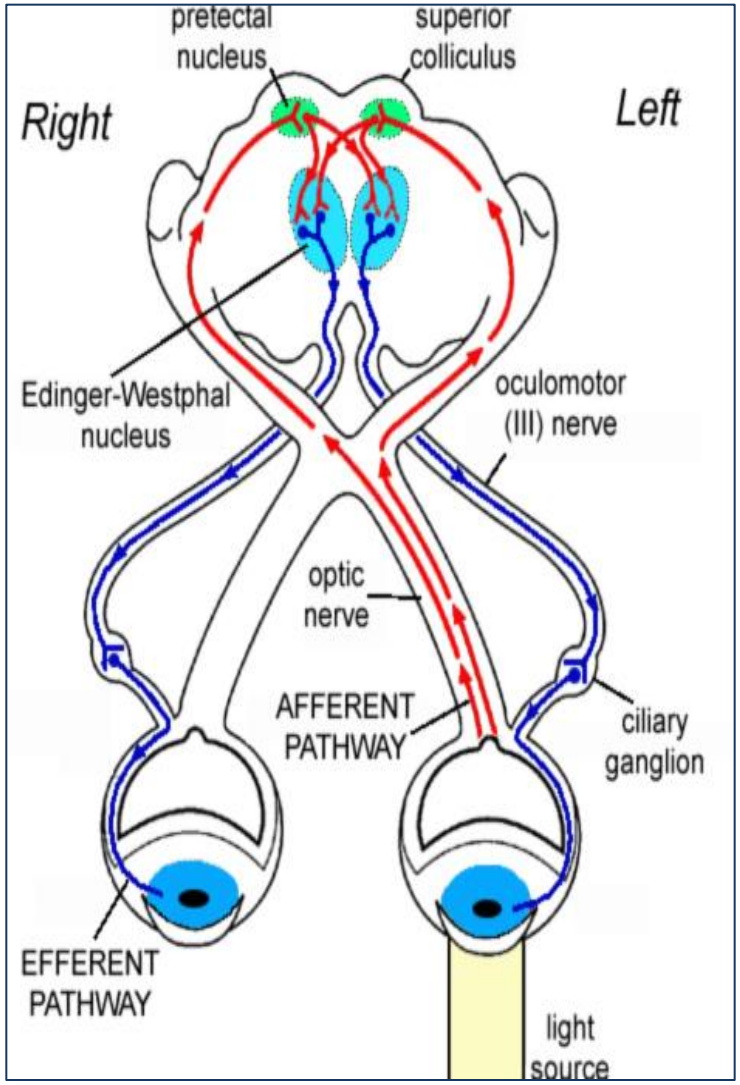
Pupils

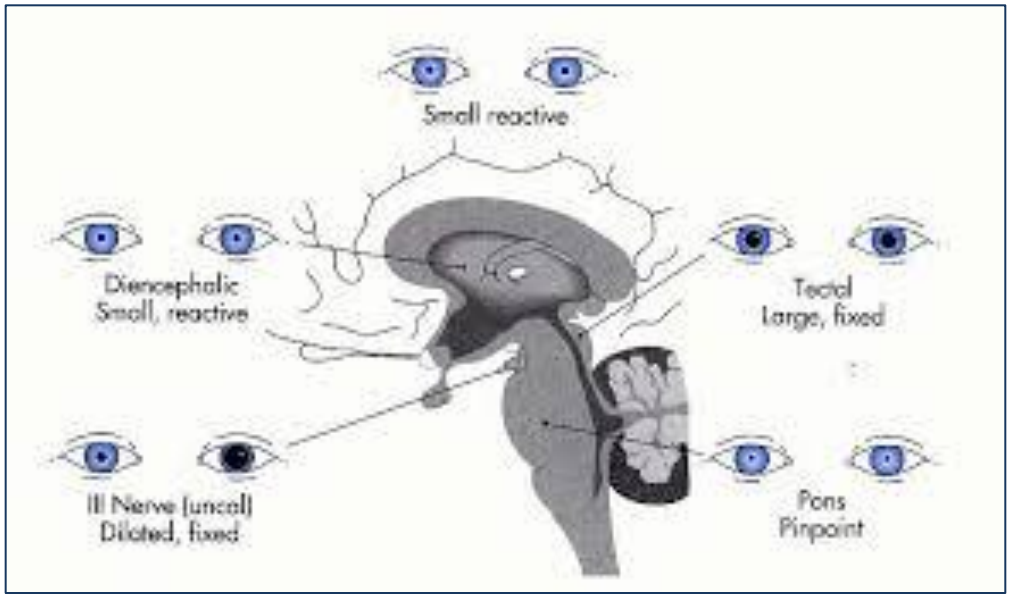
- **Pupillar reflex**

  retina, n.II., corpus geniculatum laterale, area pretectalis, bilat. nn. W-E, mm. sphincter pupillae

- **Ciliospinal reflex**
- **(afferent C2, C3, efferent sympathetic fibers)**

  during nociceptive stimulation on the face, neck, trunk (by pitching the neck) bilat. dilatation of pupils is more than 1 mm





- **Hemisphers – without change**
- **Hypotalamic lesions – homolat. miosis, FR +, (S)**
- **Tectal lesions (diencephalo.mezencephal.)-large, circle, 5-6 mm, FR -**
- **Mezencephalic lesions – medium size, frequently asym., FR: 0, ciliospin. R. + (PS,S)**
- **Uncal lesions:kálne herniácie – dilatation of homolat. pupil**
- **Pontinne lesions – very small pupils (S, iritation PS)**
- **Spinal cord lesions – wide, dilatated pupils from hypoxia**

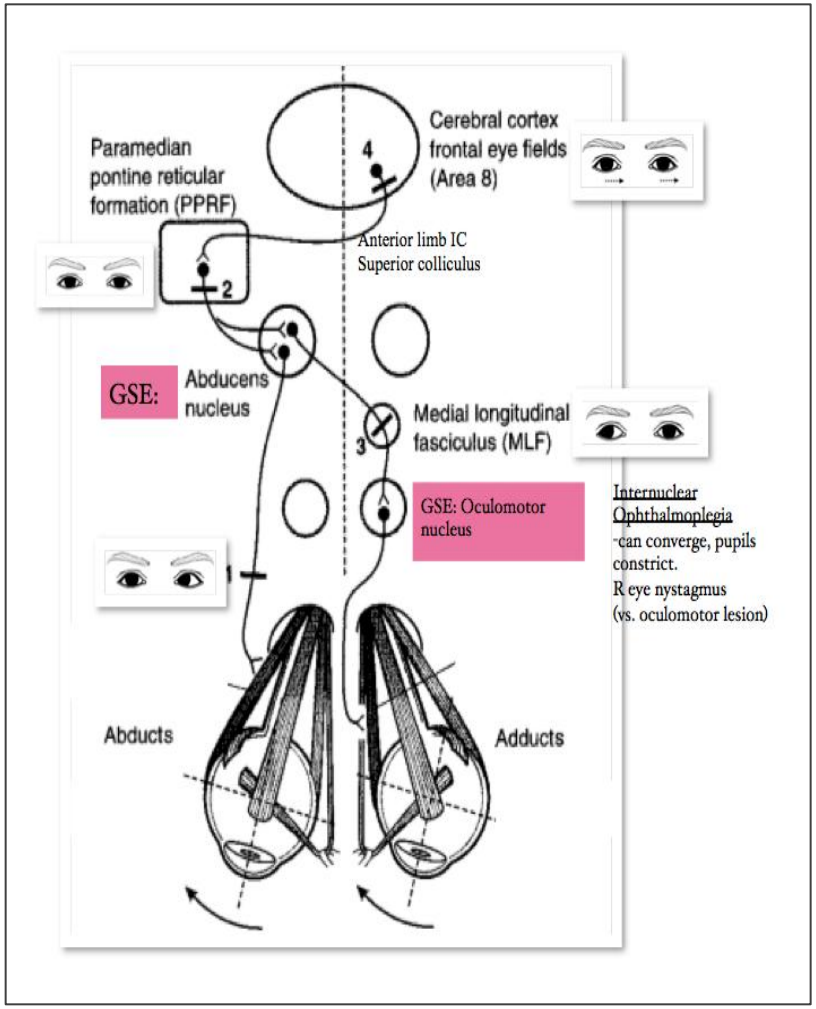
	Right	Left
Oculomotor Nerve Compression Dilated, nonreactive (fixed) pupil due to either cerebral edema or uncal herniation at the ipsilateral side of the dilated pupil.		
Bilateral Diencephalic Damage Small, reactive pupils indicative of bilateral, sympathetic pathway injury at the thalamus and hypothalamus. This can be present in metabolic coma.		
Horner's Syndrome Small, reactive pupil (miosis) at the affected side with lid ptosis. Patient will also exhibit anhidrosis on the forehead on the same side. It can be caused by trauma to the neck, carotid artery dissection, or a lesion at the lateral medulla or ventrolateral cervical spinal cord.		
Pontine Damage Small, nonreactive pupils. This can be due to pontine damage due to ischemia or hemorrhage. Bilateral pinpoint pupils could also be representative of opiate overdose.		
Bilateral Dilated Unreactive Pupils Pupils are dilated and fixed secondary to severe anoxia and indicative of severe brain injury and imminent death.		

Oculomotor response

- Position of eyes
- Movements of eyes
- Reflex movements

Position and movements of eyes

- **Hemisfers - F, O - paralyse of conjugate horizontal movements – looking at lesion**
- **Brain stem – fasciculus medialis, nc. paraabducens – paralyse of conjugate horizontal movements to brain stem lesion – looking from lesion, on health side**
- **Mezencephalon – paralyse of conjugate vertical movements**



Motor functions

- **Muscle tone, position of extremities, voluntary moevements, movements on pain stimuli**

- **Reactions to pain impulse**

  cortico-subcortical level – flexion of UE, reaction of mimic muscles – intact sensitive pathway

Motor system evaluation

- **Muscle tonus, postural limb tonus, spontaneous movements, motor reaction with algic stimuli**
- **Algic stimulus- reaction:**

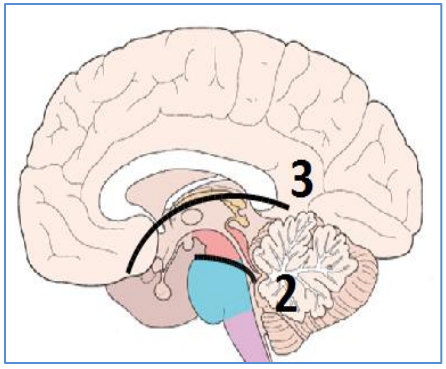
  cortico-subcortical level: UL flexion, mimic response – sensory pathways are intact, corticospinal tract is maderately damaged

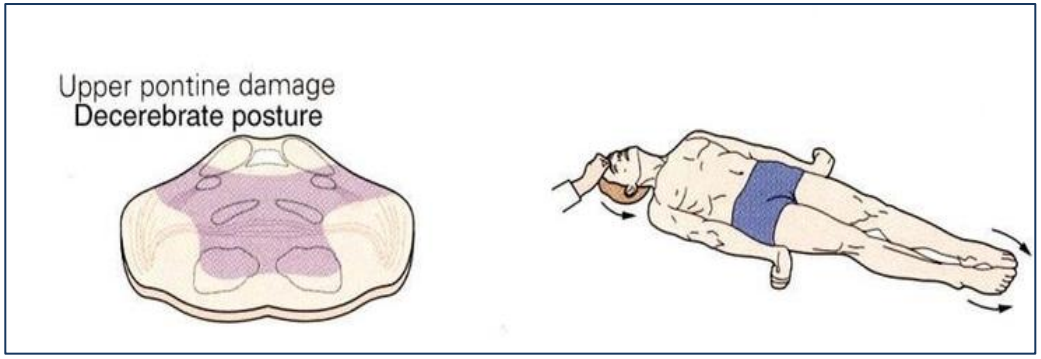
Motor functions

- **Diencephalon lesion – no flexion and mimic response**
- **Lesion above nc. ruber – Wernicke-Mann position**
- **Lesion below corpora quadrigemina (above vestibular nuclei, below nc.ruber) – elimination of cerebral function - decerebrate rigidity**
- **→ rigid extension of the limbs**

Decerebrate rigidity

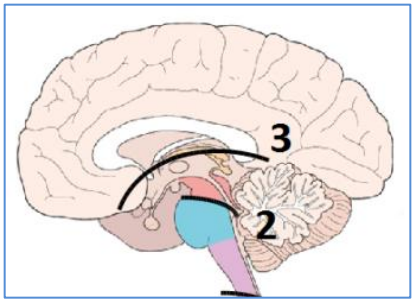
- **Brainstem compression by oedematic brain**
- **Brainstem lesion**
- **Metabolic reason – sometimes**

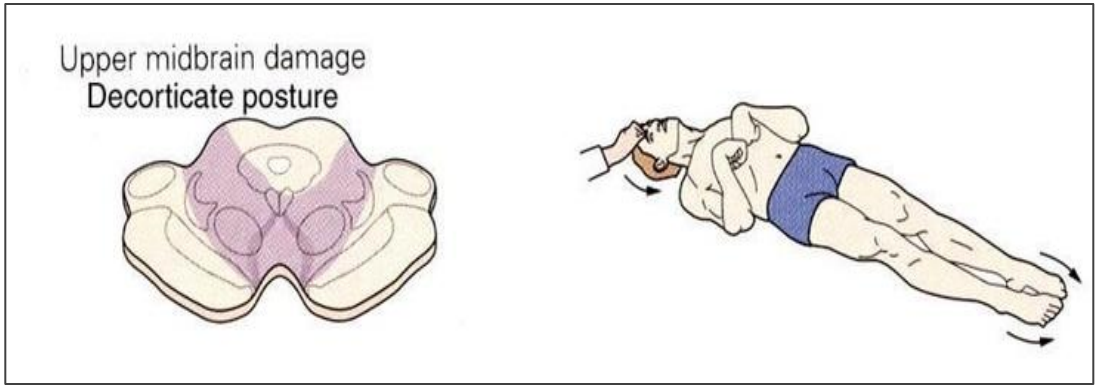




Decorticate posture

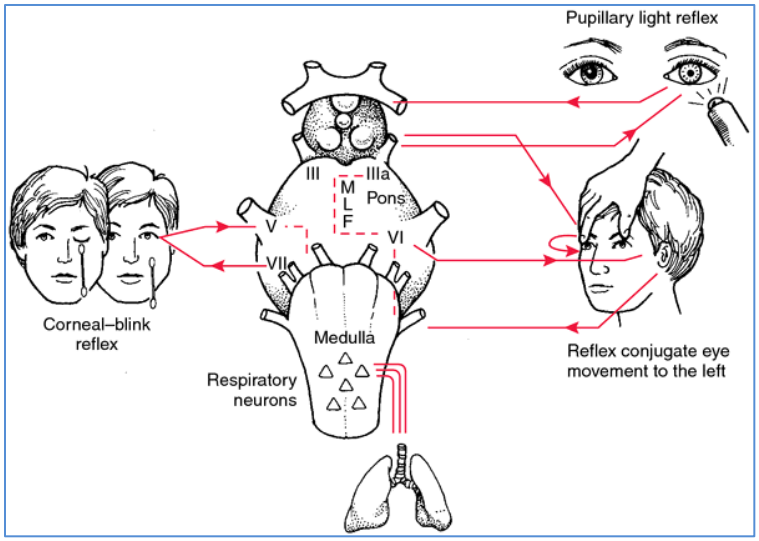
- **A posture caused by diffuse and severe cortical dysfunction, seen in a deep coma, where primitive reflex posturing prevails after the loss of higher cortical control; DP is characterized by fisted hands, arms flexed on the chest, extended legs**





Brainstem reflexes

- Pupilary reflex (Mes)
- Corneal reflex (Pons)
- Oculocephalic reflex
  - horizontal
  - vertical
- Vestibuloocular reflex
- Oculocardial reflex (MO)
- Gag and cough reflexes

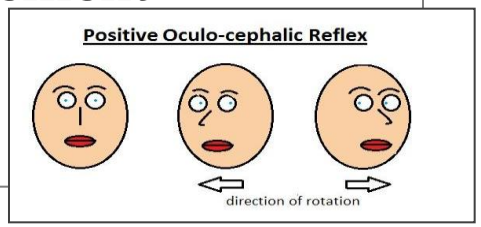


Oculocefalic reflex

Head movement is associated with eye bulbs conjugal /gaze movement í

Normal: eyes move in opposite direction with head movement

Pathology: dysconjugal or absent movement



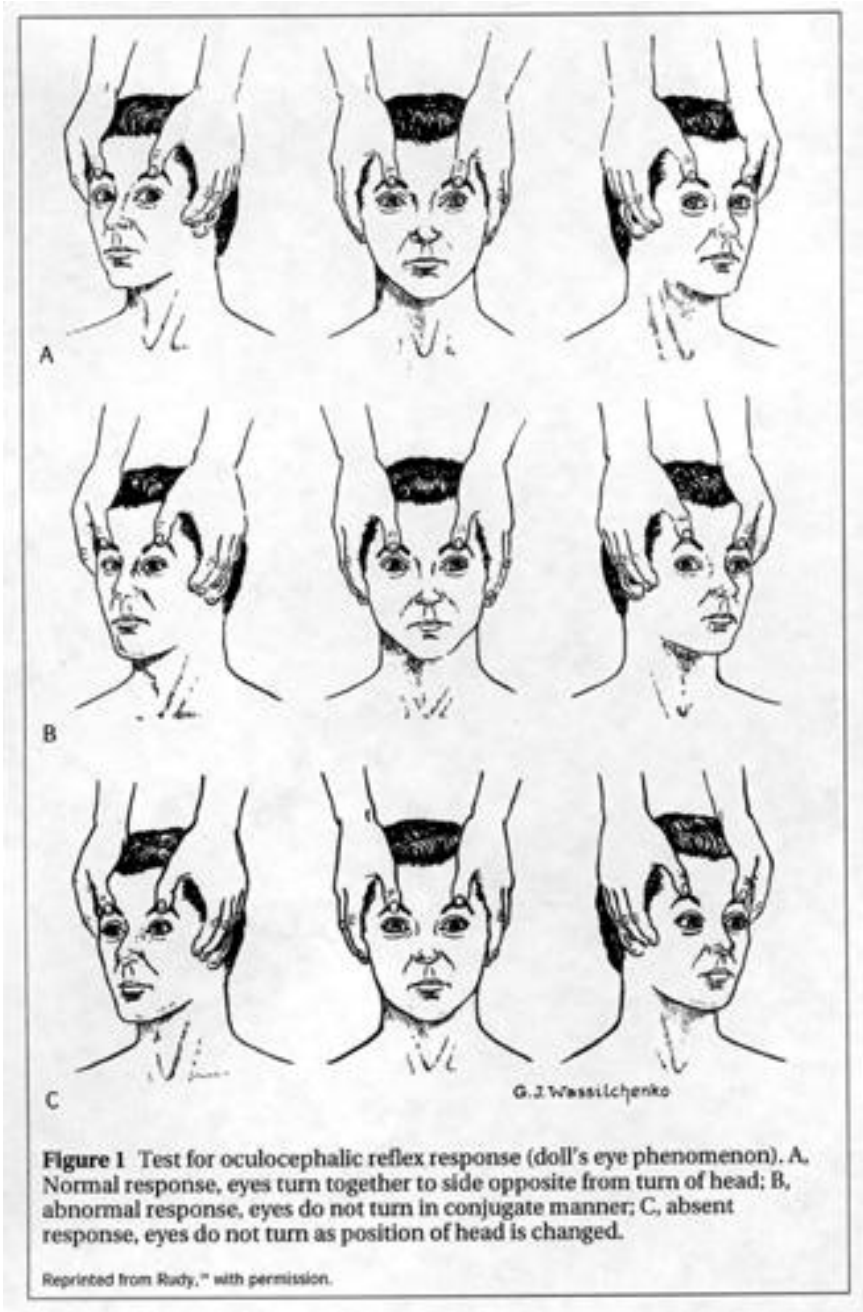


Figure 1 Test for oculocephalic reflex response (doll's eye phenomenon). A, Normal response, eyes turn together to side opposite from turn of head; B, abnormal response, eyes do not turn in conjugate manner; C, absent response, eyes do not turn as position of head is changed.

Reprinted from Rudy,[*] with permission.

Vestibuloocular reflex

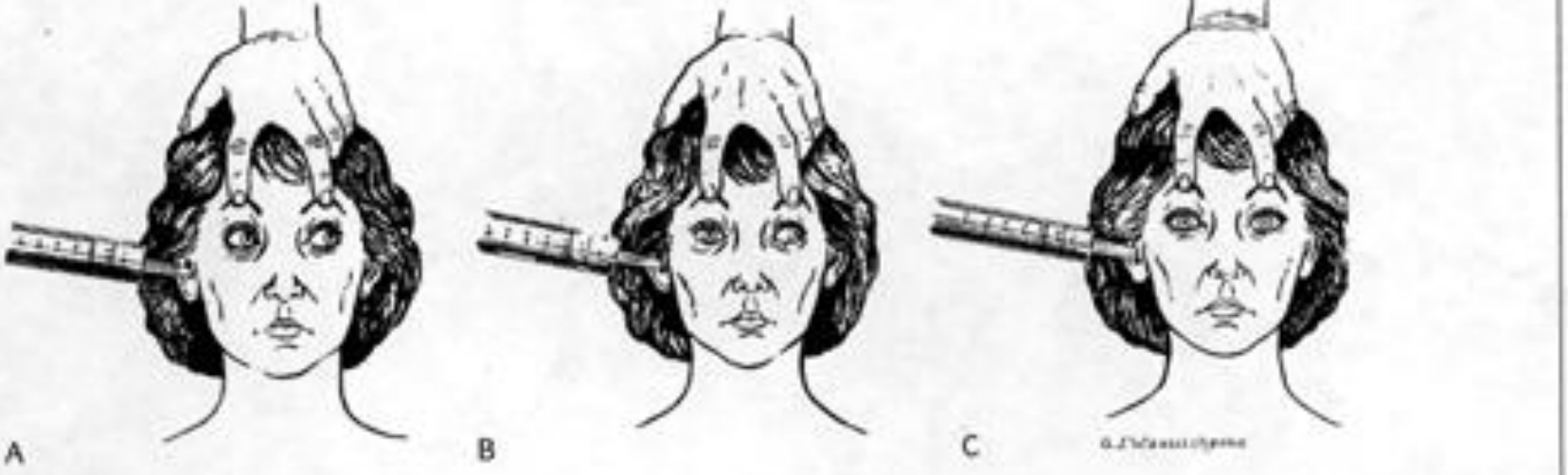


Figure 2 Test for vestibulo-ocular reflex response (caloric ice water test). A, Normal response, conjugate eye movements. B, abnormal response, dysconjugate asymmetric eye movements; C, absent response, no eye movements.

Reprinted from Rudy,[14] with permission.

Oculocardial reflex

- Activation:

  bulbus compression
- Afferent pathway: trigeminal nerve (n.V.)
- Efferent pathway: vagal nerve (n.X.)
- Result: bradykardia

Gag reflex

- **Gag reflex** is a normal reflex in humans that prevents the passage of anything from the throat, except during normal swalloving
- also named as *pharyngeal reflex*
- Touching the soft palate results in a very strong gag reflex, or vomit.

Dif.dg. of unconsciousness

- **Metabolic coma**

  pupils - isocoria, present FR, without abnormity

  - ocular movements - floating eye movements

  - motor pattern – symetric pathology

- **Lesion of the brain**

  pupils - asymmetric FR – 0

  ocular movements - lesion of conjugate movements, position of he eyeballs,

  motor pattern – symetric pathology

Coma- etiology

Focal brain dysfunction

- brain tumour, MTS
- vascular events –ischemia, haemorrhage
- demyelination
- infection, such as cerebral abcess
- focal head injury (subdural or epidural haemorrhage

Diffuse brain dysfunction

- infection, such as meningitis or encephalitis
- epilepsy
- hypoxia and hypercarbia
- drugs, poisoning and overdoses (including alcohol)
- metabolic/endocrine causes, such as diabetic coma, hepatic or renal failure, hypothyroidism, severe electrolyte disturbances
- hypotension, or hypertensive crisis
- diffuse head injury
- subarachnoid haemorrhage
- hypothermia, hyperthermia

Causes of unconsciousness

Focal

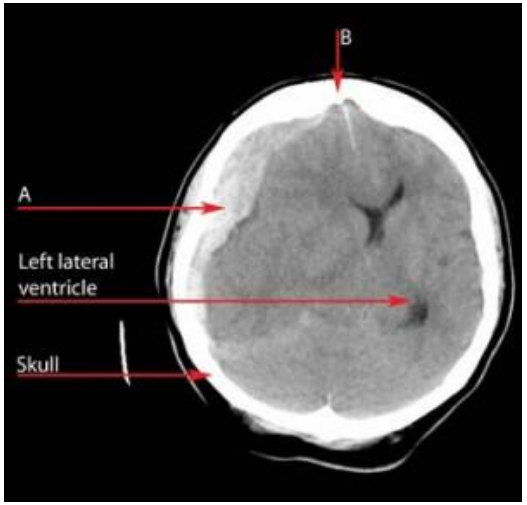
A/ Brain haemorrhage

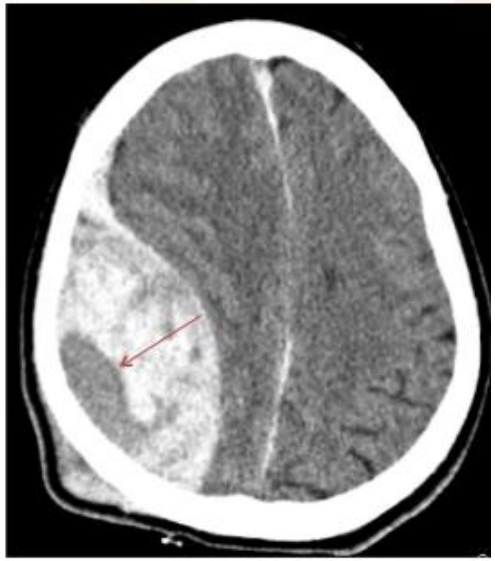
B/ Brain infarkt

C/ Brain absces

D/ Brain contusion, subdural or epidural haematoma

E/ Brain tumor





Causes of unconsciousness

- **Without local lesion**

A/ Intoxication – alkohol, drugs

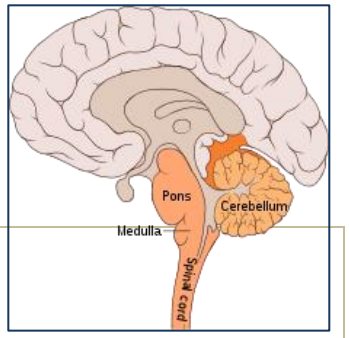
B/ metabolic causes – anoxia, diabetes, acidosis, renal and liver coma, hypoglykemia, addison crisis

C/ severe infections – pneumonia, ...

D/ circulatory colaps

E/ eklampsy

Unconsciousness



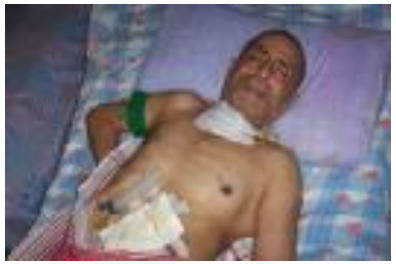
- **Vegetative state**
- **Patient is awake, follow people with eyes**
- **Primitive responses, reflex movements**
- **Present brain stem reflexes**
- **Quadrusymptomatology**
- **Disturbances of the rhytm of vigility and sleeping**

Persistent /chronic vegetative state

- is a condition of patients with severe brain damage in whom coma has progressed to a **state of wakefulness without detectable awareness**

- **Arousal is present**, but the ability to interact with the environment is no.

- **mental function is absent**



Persistent vegetative state (VS)

- Eye opening can be spontaneous or in response to stimulation
- General responses to pain exist, such as increased heart rate, increased respiration, posturing, or sweating
- Sleep-wakes cycles, respiratory functions, and digestive functions return
- There is no test to specifically diagnose Vegetative State; the diagnosis is made only by repetitive neurobehavioral assessments.
- Persistent Vegetative State (PVS) is a term used for a Vegetative State that has lasted for more than a month

Vegetative state

- **Improvement – always some residuum**

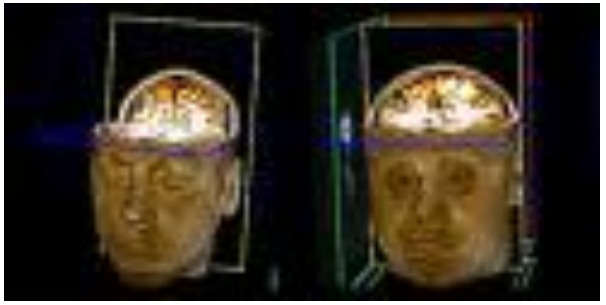
- **Progression- Coma de passe – brain death**
  - irreversible

Apalic syndrom, vegetative state

- Persistent
- Death

- Persistent
- Permanent
- Death

Apalic syndrom



- **Etiology: diffuse severe cerebral damage**
- **Absence of cortical functions - (EEG)**
- **Decortical posturing**
- **Retained –head rotation, oral automatisms-tongue clicking, lips licking**
- **Supranuclear oculomotor palsy**

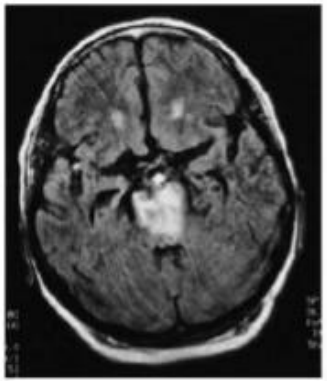
<u>Prognosis</u>: - improvement with residual symptoms

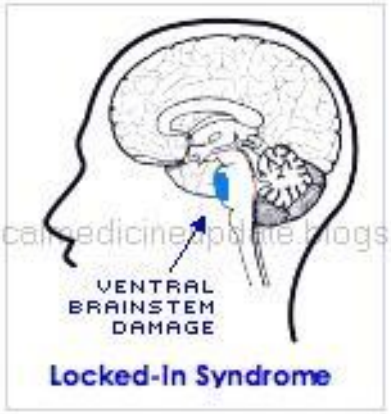
- persistent vegetative state

Dif.dg. of unconsciousness

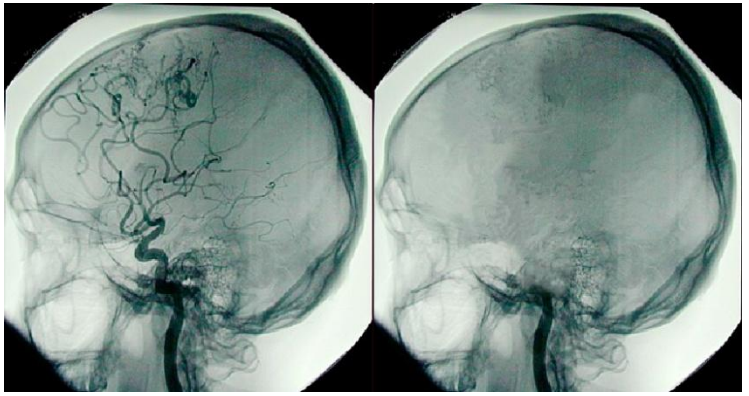
- **Locked-in-syndrom**

lesion in ventral pons, patient is fully awake, he cannot speak or move, he can answer by moving with eyes – vertical movements of eyeballs are preserved

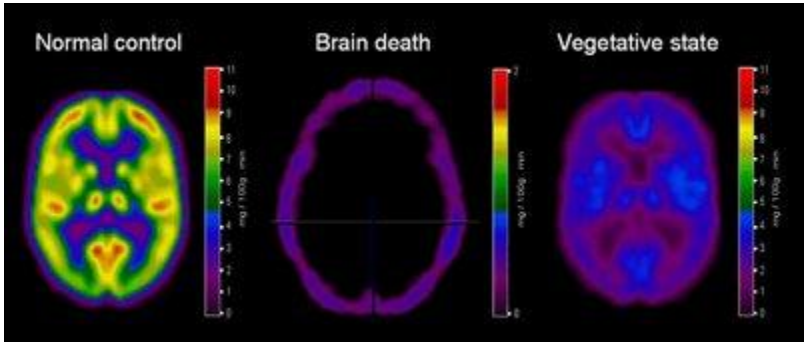




Brain death



- Irreversible lost of all brain functions
- Absence of spontaneou breathing and circulation (blood pressure)
- Autonomic cardial activity present
- Thermoregulation is absent
- Absence of all motor activities
- Fixed dilated pupils, FR-
- Muscle hypotonia, arreflexia



Coma de passe – brain death

- **Brain death** is defined **as irreversible cessation of all brain activity**
- **The determination of brain death depends on very definite clinical and laboratory findings:**

Clinically, a person is brain dead when all of the following conditions are met:

- **no spontaneous respirations**
- **pupils are dilated and fixed**
- **no response to noxious stimulation (painful stimulation provokes no eyeblink, no grimacing, no movements of any part of the body).**
- **all extremities are flaccid (there is no movement, no muscle tone and no reflex activity in any of the limbs - arms or legs).**
- **There are no signs of brain stem activity:**

There are no signs of brain stem activity:

- the eyeballs are fixed in the orbits + mydriasis bilateral

- no corneal reflexes

- no response to caloric testing (exposing the tympanic membrane of the ear to ice cold water fails to produce movement of the eyes)

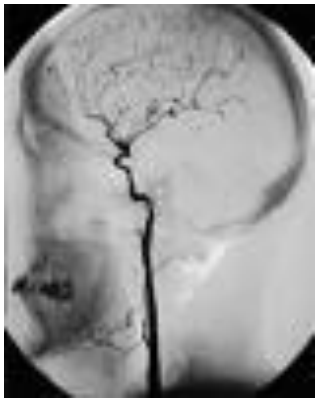
- no gag reflex or cough reflex

- **If all of the clinical criteria of "brain death" have been met, a person cannot be declared "brain dead" until the physician has made sure that :**

  - no opiate drugs (ex. codeine, morphine, cocaine, heroin) and no barbiturate drugs (ex. phenobarbital, secobarbital, nembutal, amytal) have been administered in the previous 24 hours

Brain death has been confirmed by one of the following diagnostic studies:

- **Cerebral AG - showing no penetration of dye into the arteries of the brain.**

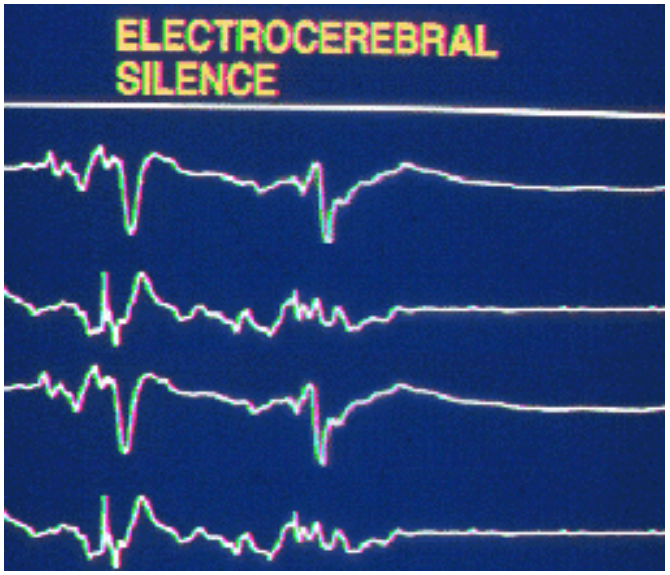


- **Two EEG's – not in SR,**
- **showing no electrical activity coming from the brain, i.e., flat or isoelectric tracings**

EEG

- **The EEG measures brain voltage in microvolts. It is so sensitive that the static electricity in a person's clothes will give a squiggle on the EEG (a false positive).**

- **All positive responses suggest brain function. The patient in the deepest coma will show some EEG electroactivity, while the brain-dead patient will not.**



- **Prognosis:**

- **A "dead" brain has never been known to recover**

- **Modern medicine can maintain bodily functions (except brain function) for long periods of time before heart and kidneys failed**

Cerebral herniation

- a deadly side effect of very high intracranial pressure
- occurs when the brain shifts across structures within the skull Because herniation puts extreme pressure on parts of the brain, it is often fatal

- The brain can shift by structures as

  - the falx cerebri - cingular herniation

  - the tentorium cerebelli- uncal herniation

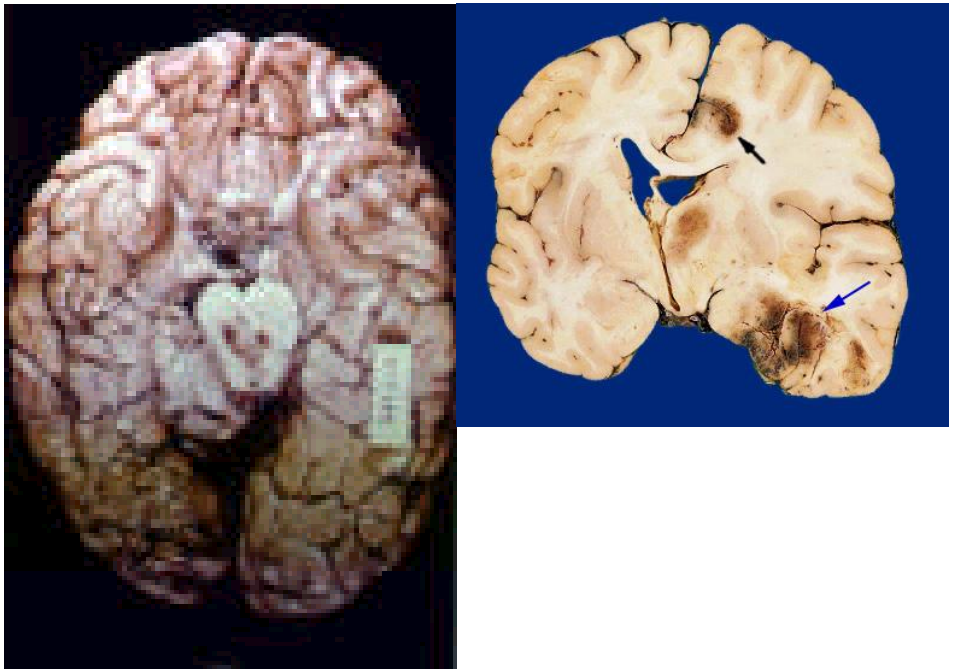
  - through the foramen magnum in the base of the skull - occipital herniation

Intracranial hypertension syndrome ICH

- Headache
- Vomitus
- Dizziness
- Impressions of brain at the skull (X-ray)
- Oedema of the optic nerve papila
- Brain oedema - CT

Brain herniations

- Temporal



- Occiptal

Brain herniations