EVALUATION OF COMATOSE PATIENT

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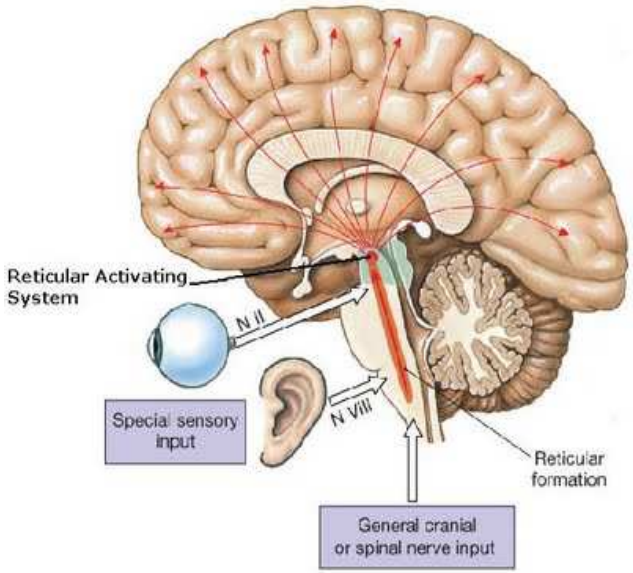
Consciousness

- Two components of conscious behavior:
  - **Vigilance (arousal):** appearance of wakefulness
  - **Awareness (content):** the sum of cognitive and affective function
- Awareness depends on arousal, but normal arousal does not guarantee normal awareness

Neuroanatomy

- Where is Vigilance localized?
  - Ascending Reticular Activating System (ARAS) in pons and midbrain (mesencefalo)
  - receives input from numerous somatic afferents
  - projects to midline thalamic nuclei (which are in circuit with cortical structures) and the limbic system



Consciousness

- Consciousness requires:
  - An intact pontine *ARAS*
  - An intact cerebral hemisphere, or at least part of a *hemisphere*
- Coma requires dysfunction of either the:
  - Pontine ARAS or
  - Bihemispheric cerebral dysfunction

COMA

- Definition of Coma: unarousable and unresponsiveness in which the subjects lie with eyes closed

- Other older terms: obtundation, stupor
  - fallen out of favour because of imprecision
  - descriptive methods favoured

What is Coma?

- No awareness or vigilance
- Lasts > 1-6 h (differential diagnosis with syncope and concussion)
- No spontaneous speech or movement, eyes shut
- No eye opening to verbal command
- Noxious stimuli: vocalisation absent or limited
- Noxious stimuli: motor activity absent or abnormal or reflexive (not purposeful or defensive)

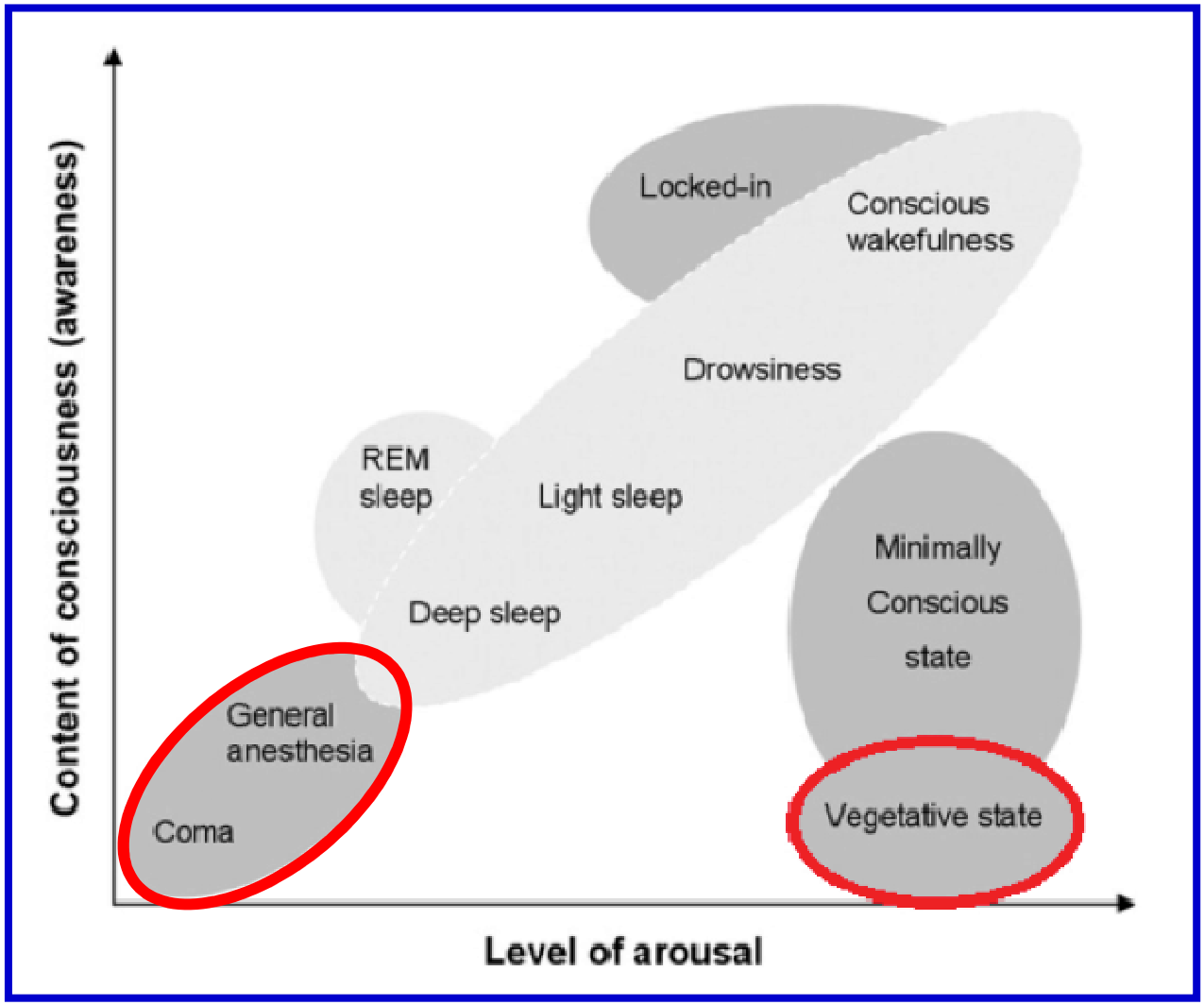
COMA AND TRANSIENT LOSS OF CONSCIOUSNESS

Loss or alteration in consciousness is a very common clinical disorder. This can be transient lasting seconds or minutes as occurs in syncope and seizures or more prolonged as occurs in coma. *Coma is by definition a state of impaired consciousness during which the patient is unrousable by external stimuli.* In states of coma the patient remains in a sleep like state with no purposeful movements or response to any external stimuli. These can be measured by the Glasgow Coma Scale which defines coma as a GCS ≤ 8/15. Coma can be caused by disorders that affect either

States of altered consciousness

Confusion	disturbed consciousness and impairment of higher cerebral function
Delirium	confusion with motor restlessness, and transient hallucinations and delusions
Stupor	conscious but rousable only with intense stimulation
Coma	unrousable unresponsiveness (GCS ≤ 8/15)
Vegetative state	loss of consciousness with preservation of brain stem function
Death	loss of consciousness and capacity to breathe spontaneously: irreversible

AWARENESS



VIGILANCE

Epidemiology of Coma

- Plum and Posner 1982
  - **500 consecutive cases of coma:**
    - 101 supra-tentorial (44 IC Hemorrhage)
    - 65 sub-tentorial lesions (40 brainstem – infarcts)
    - 326 diffuse or metabolic brain dysfunction
      - 149 drug intoxication

Pathogenesis of Coma

1. **Supratentorial lesions**: cause coma by either widespread bilateral disease, increased intracranial pressure or herniation.
2. **Infratentorial lesions**: involve the ARAS, usually with associated brainstem signs
3. **Metabolic coma**: causes diffuse hemispheric involvement and depression of ARAS, usually without focal findings
4. **Psychogenic**

Pathogenesis of Coma

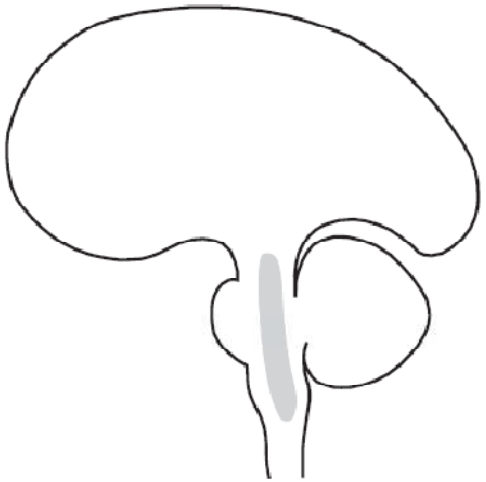


Figure 9.1 The reticular activating system

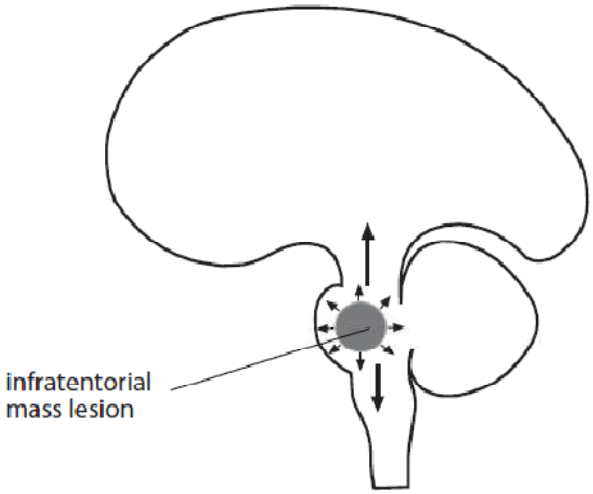


Figure 9.2 Sites that produce loss of consciousness

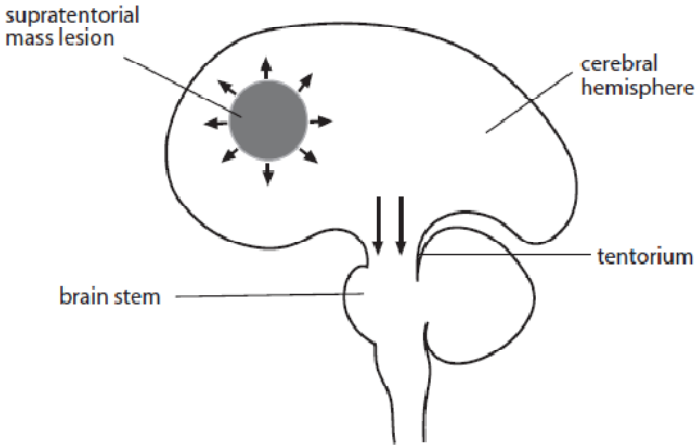


Figure 9.3 Sites that produce loss of consciousness

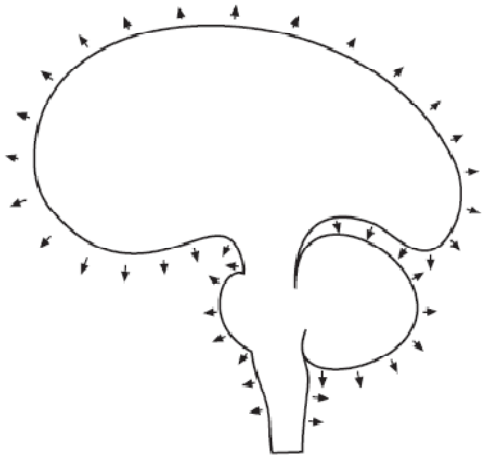
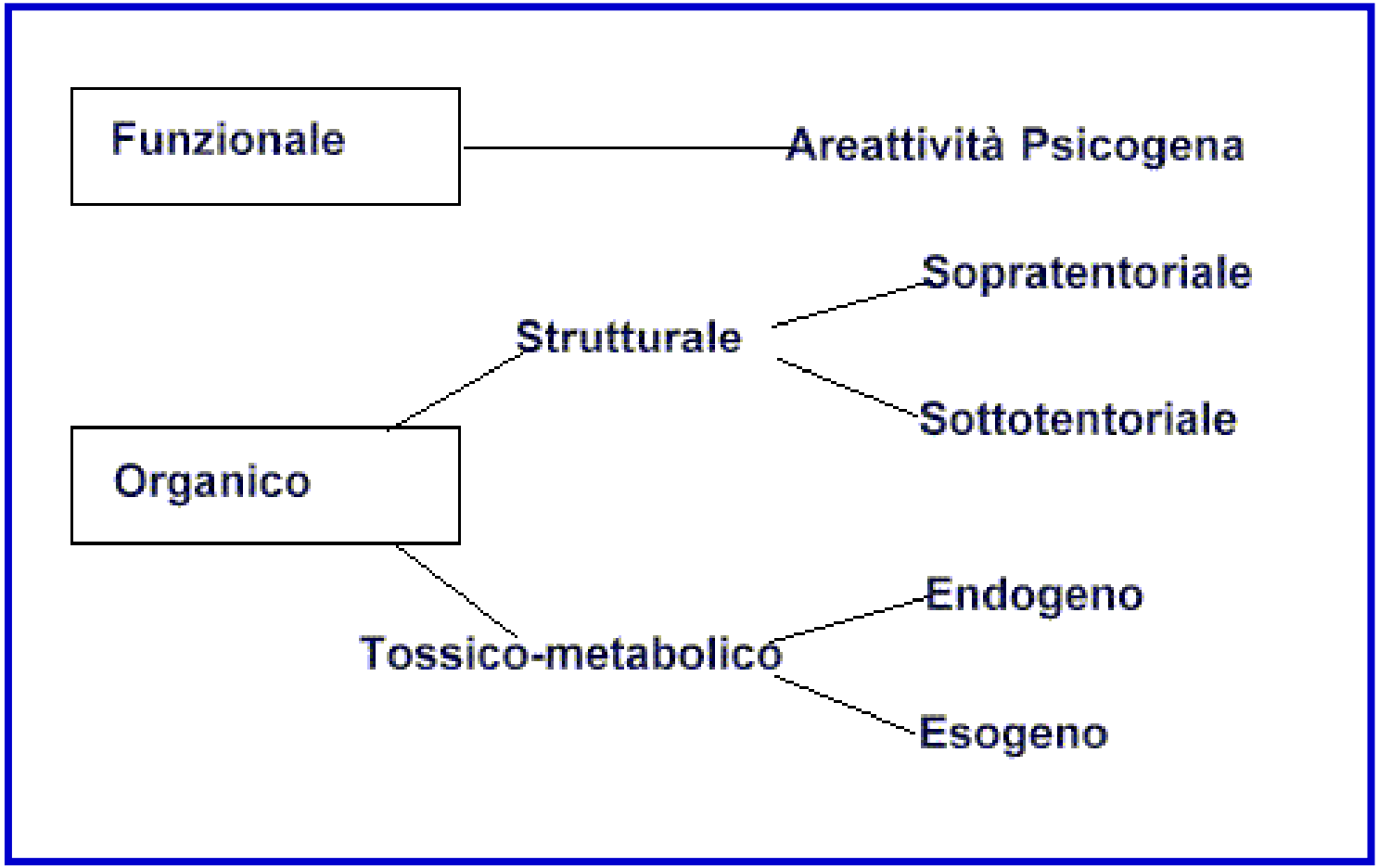


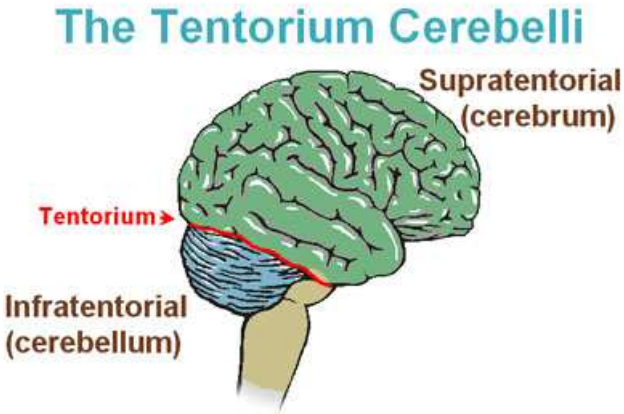
Figure 9.4 Encephalopathy diffuse

Pathogenesis of Coma



1. Supratentorial Mass Lesions

a. Hematoma
b. Neoplasm
c. Abscess
d. Contusion
e. Vascular Accidents



Subdural Hematoma

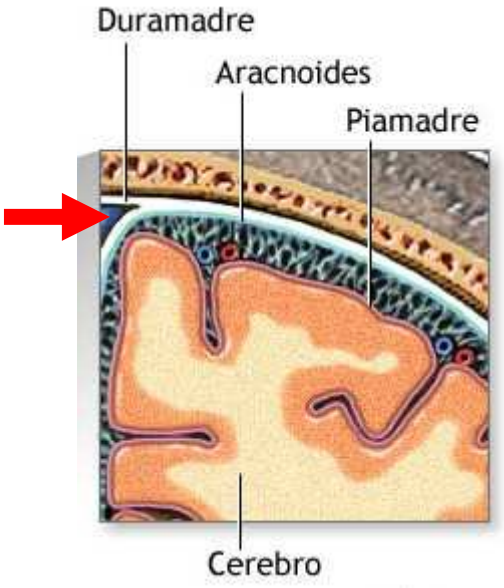
• **Subdural hematoma** is a traumatic brain injury in which blood (usually of vein origin) gathers ***between the Dura Mater and the Arachnoid***. Subdural hemorrhages may cause an increase in intracranial pressure, which can cause compression of and damage to brain tissue (mass effect).

• Subdural hematomas are divided into: ***ACUTE, SUBACUTE, and CHRONIC,*** depending on their speed of onset. ***Acute subdural hematomas*** that are due to trauma are ***the most lethal of all head injuries*** and have a high mortality if they are not rapidly treated with surgical decompression.

Subdural Hematoma

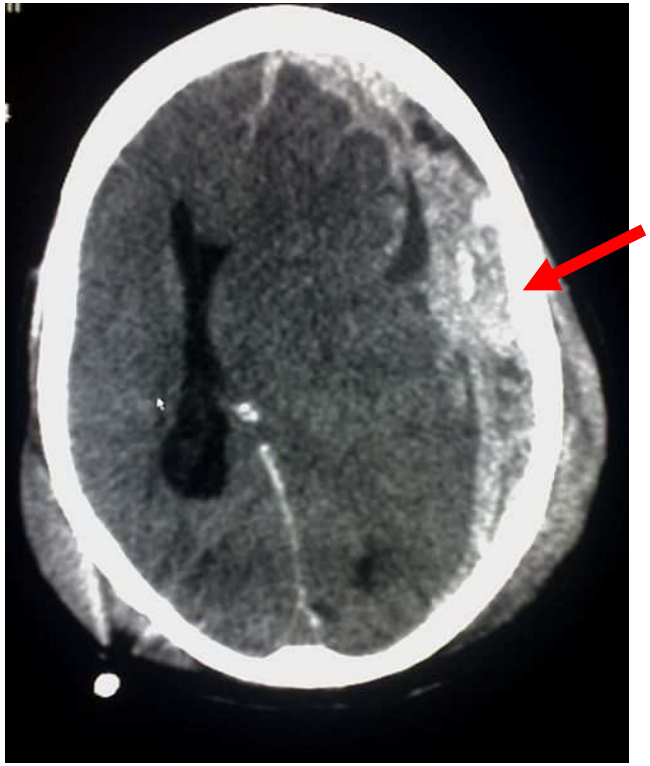
• Factors increasing the risk of a subdural hematoma include:

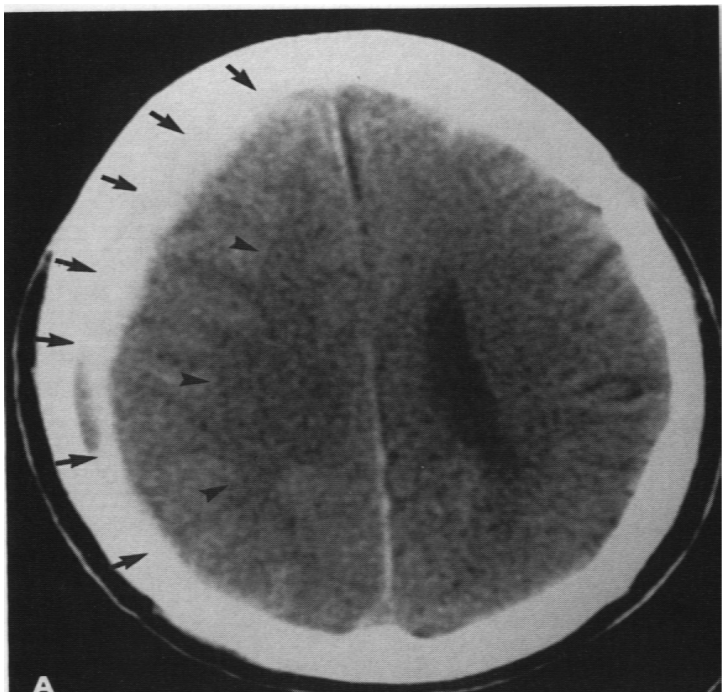
- very young age
- very old age (***search for previous falls in olders***)
- **anticoagulants (warfarin)**
- **aspirin and other antiplatelet drugs**
- alcohol abuse
- dementia



Subdural hematoma diagnosis

CT scan or MRI imaging



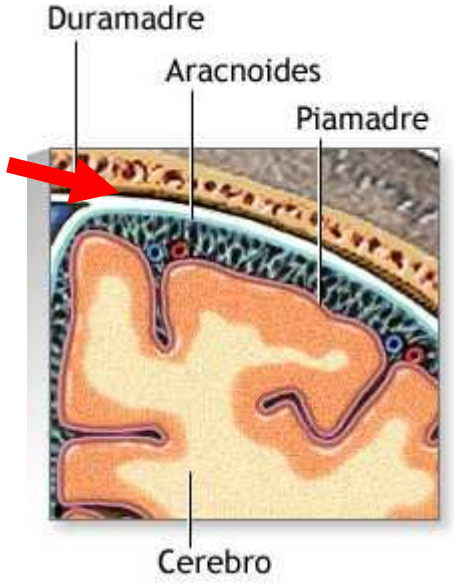


Acute Epidural Hematoma

• **Epidural or extradural hematoma** is a type of traumatic brain injury in which a buildup of blood occurs ***between the Dura Mater and the Skull.***

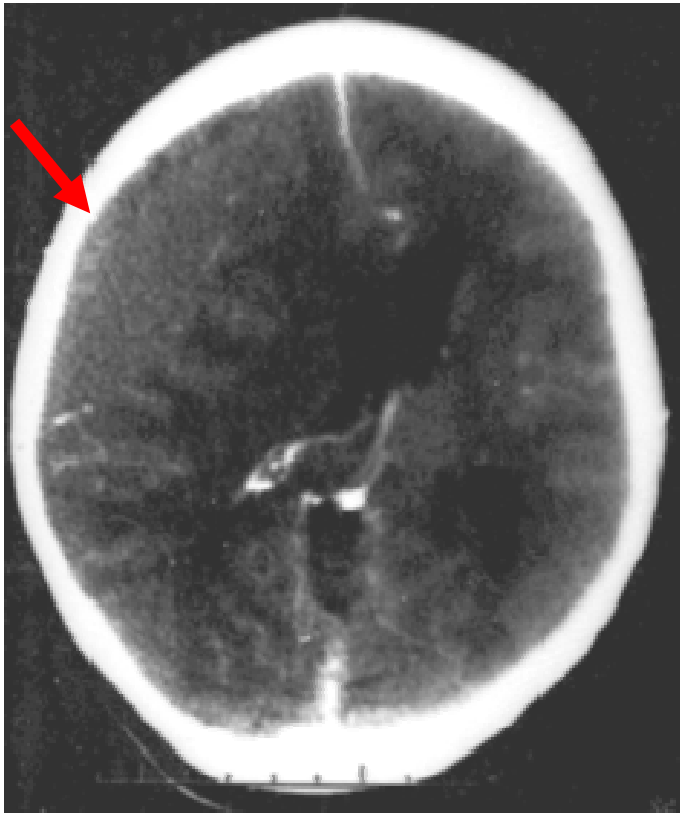
• Epidural bleeding ***is rapid because it is usually from arteries,*** which are high pressure. Epidural bleeds from arteries can grow until they reach their peak size at six to eight hours post injury, spilling from 25 to 75 cc of blood into the intracranial space.

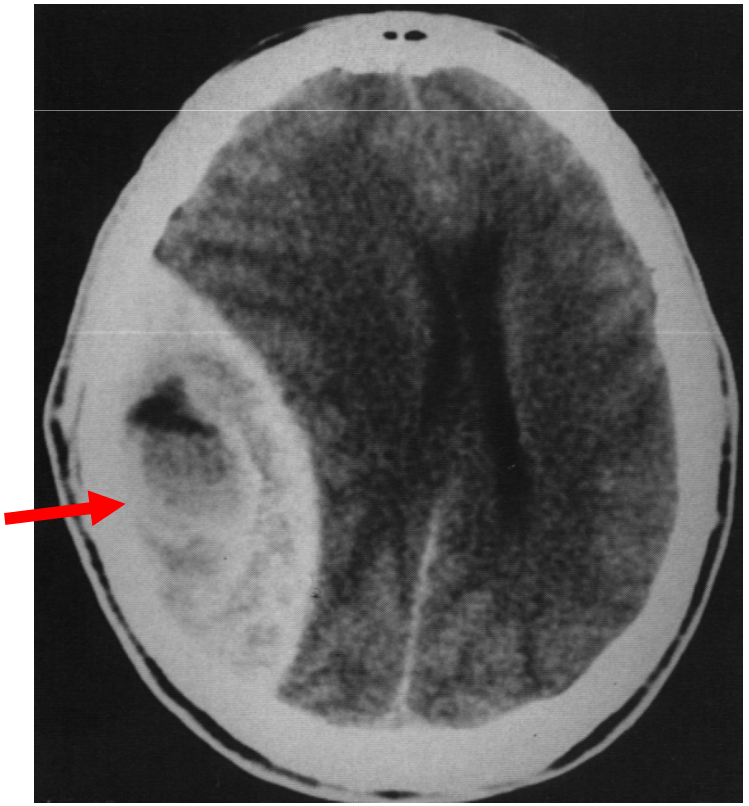
• As the hematoma expands, it strips the dura from the inside of the skull, causing an *intense headache*.



Acute Epidural Hematoma

CT scan or MRI imaging





Cerebral Abscess

• **Brain abscess** is an abscess within the brain tissue caused by collection of infected material, coming from local (*e.g. ear infection, dental abscess, infection of paranasal sinuses, infection of the mastoid air cells of the temporal bone, epidural asbcess*) or remote (*lung, heart, kidney*, etc.) infectious sources.

• The infection may also be introduced through a skull fracture following a head trauma or surgical procedures.

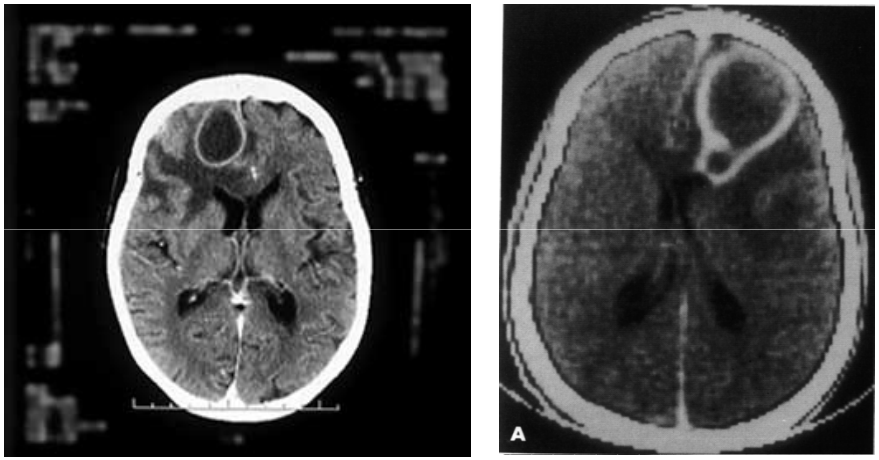
Cerebral Abscess

• The symptoms of brain abscess are caused by increased intracranial pressure due to a space-occupying lesion:

• confusion, ***Coma***
• ***Headache,*** vomiting
• focal neurologic signs

plus

• infection: fever, fatigue



The most frequent presenting symptoms are: headache, drowsiness, confusion, seizures, hemiparesis or speech difficulties together with fever with a rapidly progressive course.

Supratentorial Mass Lesions
Pathophysiology

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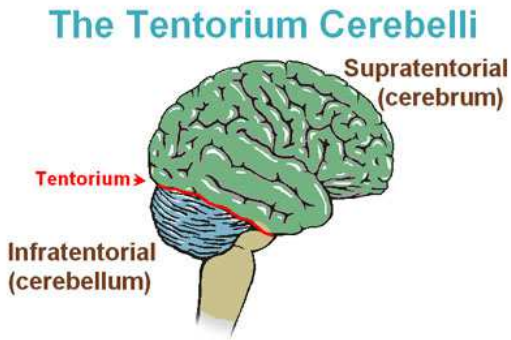
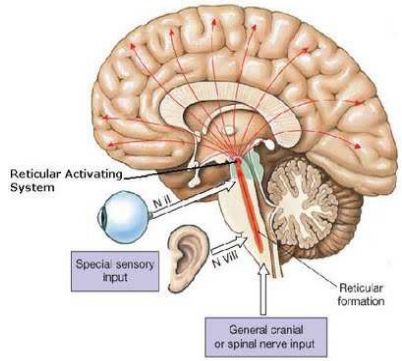
- The altered consciousness is based on:

  - *Increased intracranial pressure*
  - *Herniation*
  - *Diffuse bilateral lesions*

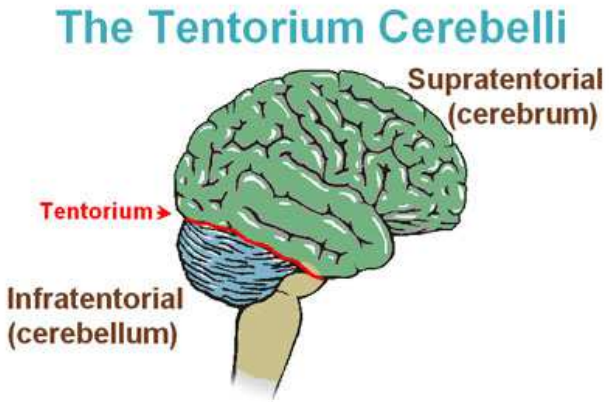


2. Infratentorial Lesions

- Cause coma by affecting ascending reticular activating system **(ARAS)** in pons.
- Brainstem nuclei and tracts usually are involved with resultant focal brainstem findings.



Infratentorial Lesions



a. Vascular accidents
b. Trauma
c. Neoplasm
d. Cerebellar hemorrhage
e. Demyelinating disease
f. Central pontine myelinolysis (e.g. too rapid correction of hyponatremia)

Infratentorial Lesions: Differentiating Features

- History of preceding ***brainstem dysfunction*** or ***sudden onset*** of coma
- ***Localizing brainstem signs*** precede or accompany onset of coma and always include oculo-vestibular abnormality
- ***Cranial nerve palsies*** usually present
- ***"Bizarre" respiratory patterns*** are common, usually present at onset of coma

3. Metabolic Coma

- Drugs
- Anoxia
- Epilepsy: *may be sub-clinical*
- CO
- SIRS
- Hypoxia
- Hypercapnia
- Glucose ↓ or ↑↑
- Sodium ↑ ↓
- Liver ko
- Kidneys ko
- Hypothermia
- Hypertension
- Hypotension
- Calcaemia ↑
- Wernicke’s
- Thyroid, adrenal, pituitary ko

Drug poisoning and Coma

FARMACO	SEGNI E SINTOMI
Acetaminofene	Anoressia, elevati livelli di enzimi epatici, ittero, letargia, insufficienza epatica, nausea e vomito, pallore
Benzodiazepine	Amnesia anterograda, atassia, coma, confusione, sonnolenza, letargia, sedazione
Beta-bloccanti	Acidosi, bradicardia, broncospasmo, coma, iper o ipo glicemia, iperpotassiemia, ipotensione, depressione respiratoria, convulsioni
Antagonisti del canale del calcio	Aritmie, bradicardia o tachicardia, coma, vertigini, ipotensione, letargia, convulsioni
Clonidina	Apnea, bradicardia, coma, iper o ipotensione, ipotermia, variazioni nello stato di coscienza, miosi
Oppiacei	Depressione del sistema nervoso centrale, stupore, letargia, coma, costipazione, nausea, vomito, vampate di calore, prurito, ipotensione, miosi, edema polmonare, depressione respiratoria, convulsioni
Salicilati	Alcalosi o acidosi metabolica, coma, diaforesi, disorientamento, squilibri elettrolitici (ipopotassiemia, iper o iponatriemia), iper o ipoglicemia, iperventilazione, nausea e vomito, insufficienza renale, acufeni e sordità, convulsioni
Sulfaniluree	Diminuzione dell'appetito, vertigini, ipoglicemia, letargia, astenia, coma
Antidepressivi triciclici	Confusione, delirio, coma, midriasi, secchezza delle fauci, ipotensione, tachicardia, convulsioni

Metabolic Coma: Differentiating Features

- Confusion and coma commonly ***precede*** motor signs
- Motor signs are usually ***symmetrical***
- ***Pupillary reactions*** are usually ***preserved***
- ***Asterixis, myoclonus, tremor, and seizures*** are common
- ***Acid-base imbalance*** with hyper- or hypoventilation ***is frequent***

Metabolic Coma

HYPOGLYCAEMIA

The diagnosis should be suspected if there are *feelings of hunger, sweating, nervousness* and *palpitations* coupled with episodes of *confusion, abnormal speech* or *unusual* behaviour in a patient not at risk for seizures or syncope. A blood glucose <2.5 mmols/L is considered to be hypoglycaemia. These attacks occur most frequently in diabetics taking oral hypoglycaemic agents or insulin. Other less common causes include during or after a period of prolonged exercise, fasting, liver disease and malignancies including hepatoma and insulinoma. The loss of consciousness in hypoglycaemia can be prolonged (>30 mins) and seizures may occur. If untreated this may proceed to coma and brain damage. The diagnosis is confirmed by measuring blood glucose during the episode and by its response to treatment. While a blood sugar level should always be checked in the unconscious patient in whom hypoglycaemia is suspected, intravenous glucose should be given without waiting for the results of a confirmatory blood sugar.

Coma “Mimics”

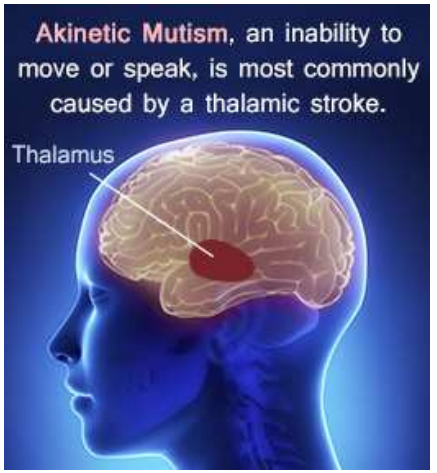
- Akinetic mutism
- ‘Locked-in’ syndrome
- Catatonia
- Conversion reactions

Coma “Mimics”

Psychogenic

This can be a manifestation of severe schizophrenia (catatonia), hysteria (conversion disorder) and malingering. These are all diagnoses of exclusion and should only be considered when other causes have been excluded and there is strong evidence in their favour. Neurological exam in these patients is invariably normal and most will exhibit resistance to eye opening and tensing or withdrawal to a painful stimulus. Investigations including caloric tests and EEGs if performed are normal. A return to full consciousness is usually the rule.

Akinetic Mutism



- Silent, immobile **but alert** appearing
- Usually due to lesion in bilateral mesial frontal lobes, bilateral thalamic lesions or lesions in peri-aqueductal grey (brainstem)

“Locked-In” Syndrome

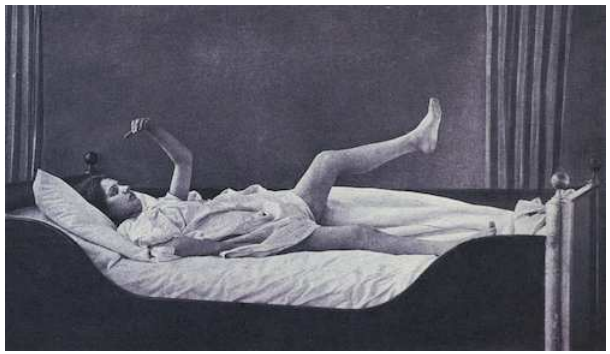
- Infarction of ***basis pontis*** (all descending motor fibers to body and face)
- Patient aware but cant’ move
- May spare eye-movements
- Often spare eye-opening
- EEG is normal or shows alpha activity

Catatonia

- **Catatonia** is a state of neurogenic motor immobility, and behavioral abnormality (stupor, mutism, posturing) associated with severe psychiatric disorders (e.g. schizophrenia)
  - can also be seen in organic brain disease such as encephalitis, toxic and drug-induced psychosis, and rarely in dementia

Conversion reactions

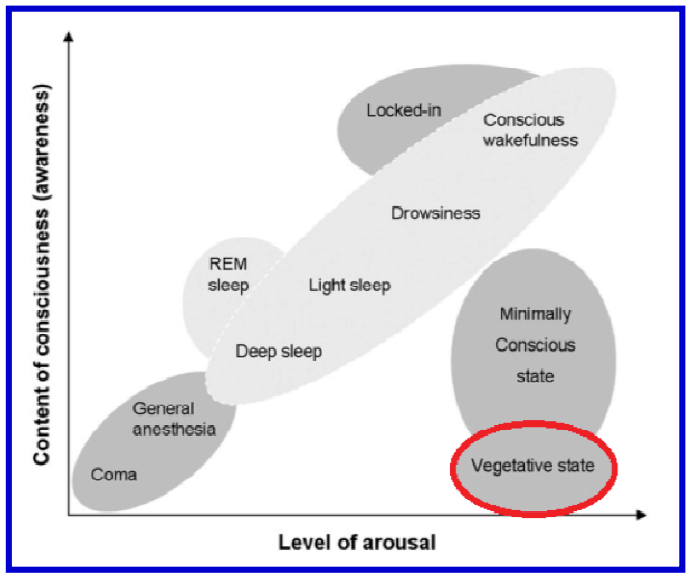
- Fairly rare (former called hysteria)
- The presence of nystagmus with cold water calorics indicates the patient is physiologically awake
- EEG used to confirm normal activity



Coma “Mimics”

Persistent vegetative state

Patient with this disorder are awake but not aware. Their eyes open and close normally and they have a sleep-wake cycle because of an intact brain stem but they show no purposeful response to any external stimuli. The cause is usually cerebral hypoxia or ischaemia or occasionally a structural lesion. Recovery is uncommon.



Coma: Clues from History

- Onset of symptoms (important):
  - *sudden onset*
  - *slow onset*
  - *fluctuations*

- Associated with neurologic symptoms
- Medications review

Approach to the Comatose Patient
Priorities

- **Airway**
- **Breathing**
- **Circulation**
- Identify and address life threatening inadequacies
- Evaluate for intracranial hypertension and imminent herniation and treat

Glasgow Coma Scale

Glasgow coma scale

The depth of coma can be measured by the **GCS**. This measures eye opening, best motor and verbal response, and is a reliable method for measuring and monitoring level of consciousness. It should be carried out and if necessary repeated on every comatose patient. When using the **GCS**, look carefully at the patient's face while assessing eye opening, and then check on the patient's ability to follow simple motor commands and listen to the patient's speech for content and orientation. If the patient is not responding to voice then test eye opening and limb movement response to deep pain by applying pressure to sternum or supra orbital ridge or nail beds. Record best eye opening, motor and verbal response as **E4, M6** and **V5.** Patients are considered comatose if the **GCS ≤ 8/15.**

Glasgow Coma Scale

- **Eye opening**
  - 4 - spontaneous
  - 3 - to speech
  - 2 - to pain
  - 1 - none
- **Verbal Response**
  - 5 - oriented
  - 4 - confused conversation
  - 3 - inappropriate words
  - 2 - incomprehensible sounds
  - 1 - none
- **Best Motor Response**
  - 6 - obeys
  - 5 - localizes
  - 4 - withdraws
  - 3 - abnormal flexion
  - 2 - abnormal extension
  - 1 - none

Max score: 15

(Coma <9)

Approach to the Comatose Patient
Priorities

- During ABC and secondary survey:
  - Start IV and obtain labs:
    - **ABG (EGA)**
    - **Whole clinical chemistry, ammonia, coagulation**
    - **Glucose stick**
    - **Toxin screens**
  - As soon as IV in and labs drawn, give:
    - Glucose
    - Consider **Thiamin (vit. B1)** (alcoholism)

Approach to the Comatose Patient
Priorities

Emergency investigations

Bloods	blood sugar malaria parasite film FBC HIV biochemistry (renal & LFTs)
Urine	sugar & ketones
X-rays	
Skull/neck	may show fracture
Chest	may show pneumonia, PCP, TB
CT head	may show blood & fracture & structural lesion

Approach to the Comatose Patient Priorities

- If CT scan / MRI is normal: probably metabolic coma
- Emergent causes of metabolic coma (even after ABC):
  - *Hypoglycemia*: give glucose
  - *Infections-sepsis*: consider antibiotics and acyclovir
    If diagnostic studies delayed, treat first
  - *Certain toxins:* antidepressants, salicylates, theophylline, alcohol (methanol) and ethylene glycol
  - *Subclinical status epilepticus* (EEG)

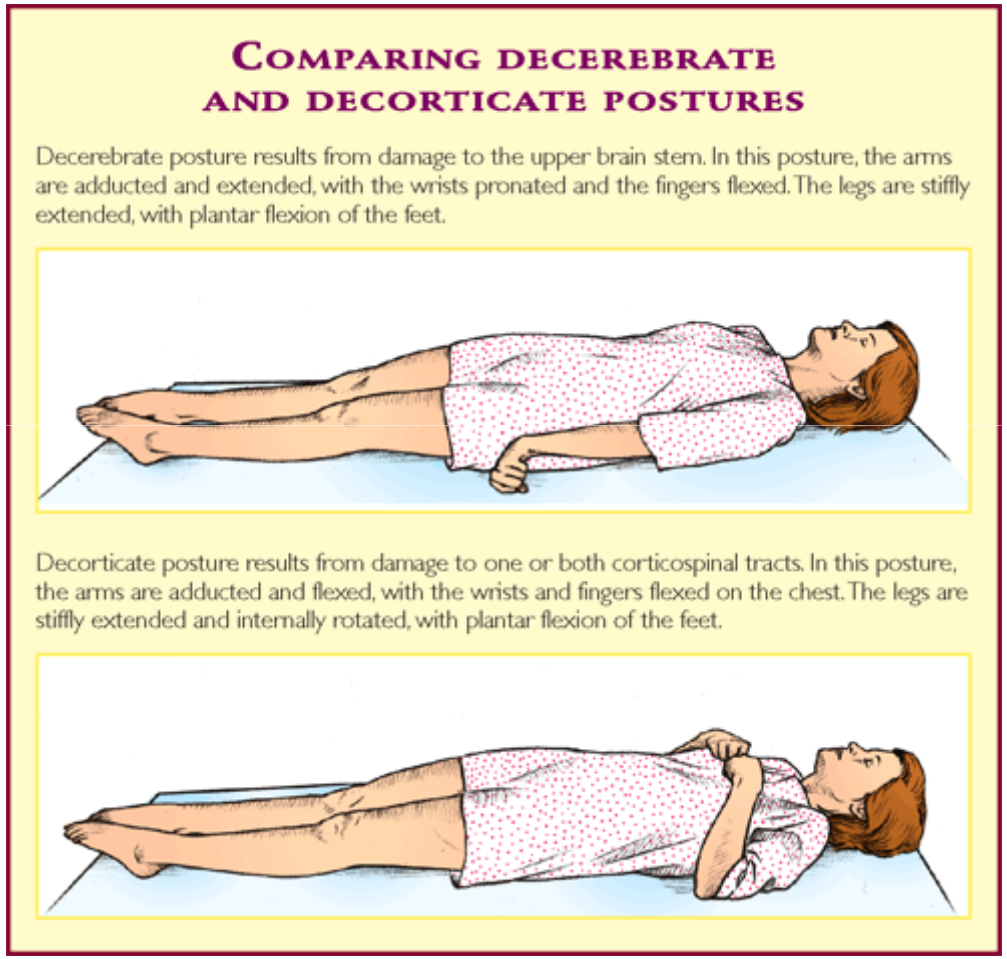
General Neurological Examination

- Tone, power, reflexes
- Identifies lateralising signs: hemispheric lesions
- In general plantars do not help; signifies hemispheric lesions but may be old
- Decerebrate or decorticate

General Neurological Examination

Decerebrate

Decorticate



Upper brain stem

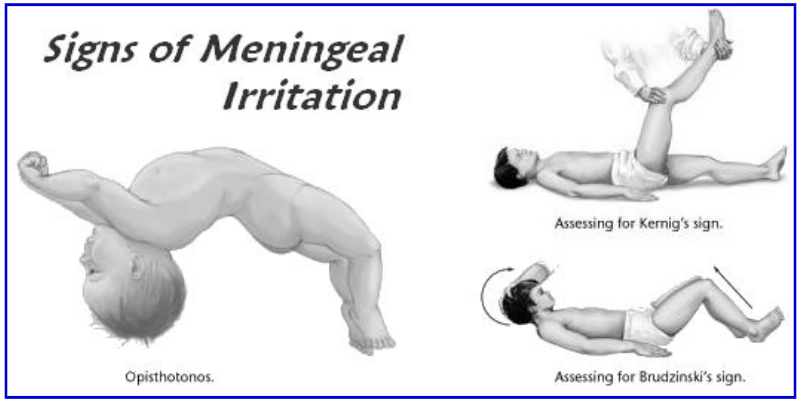
Corticospinal tract

If a patient moves from abnormal flexion (Decorticate) to abnormal extension (Decerebrate) it may be sign that damage has progressed from a supra-tentorial position to infra-tentorial position (e.g. herniation of brainstem through foramen magnum).

Meningism

Meningism is present in 3 conditions:

- Sub Arachnoidal Haemorrhage (SAH)
- Meningitis
- Meningoencephalitis



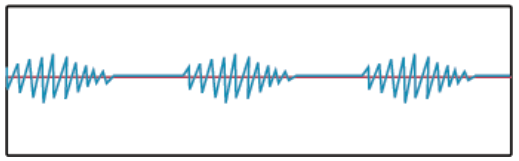
Respiratory pattern

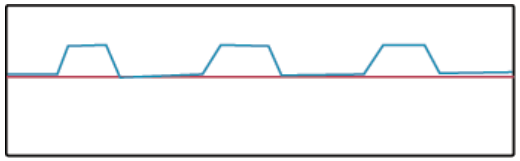
- **Hyperventilation:** pons or midbrain injury (shock, fever, acidosis, psychiatric disease)
- **Cheyne-Stokes:** bilateral diencephalic or hemispheric injury (CHF, COPD, drugs depressing response to CO2, ventilatory over- support)
- **Apneustic breathing:** pons
- **Ataxic:** medulla, usually preterminal

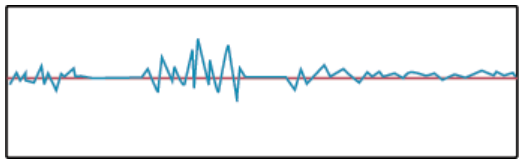
Respiratory pattern

Respiratory patterns in coma

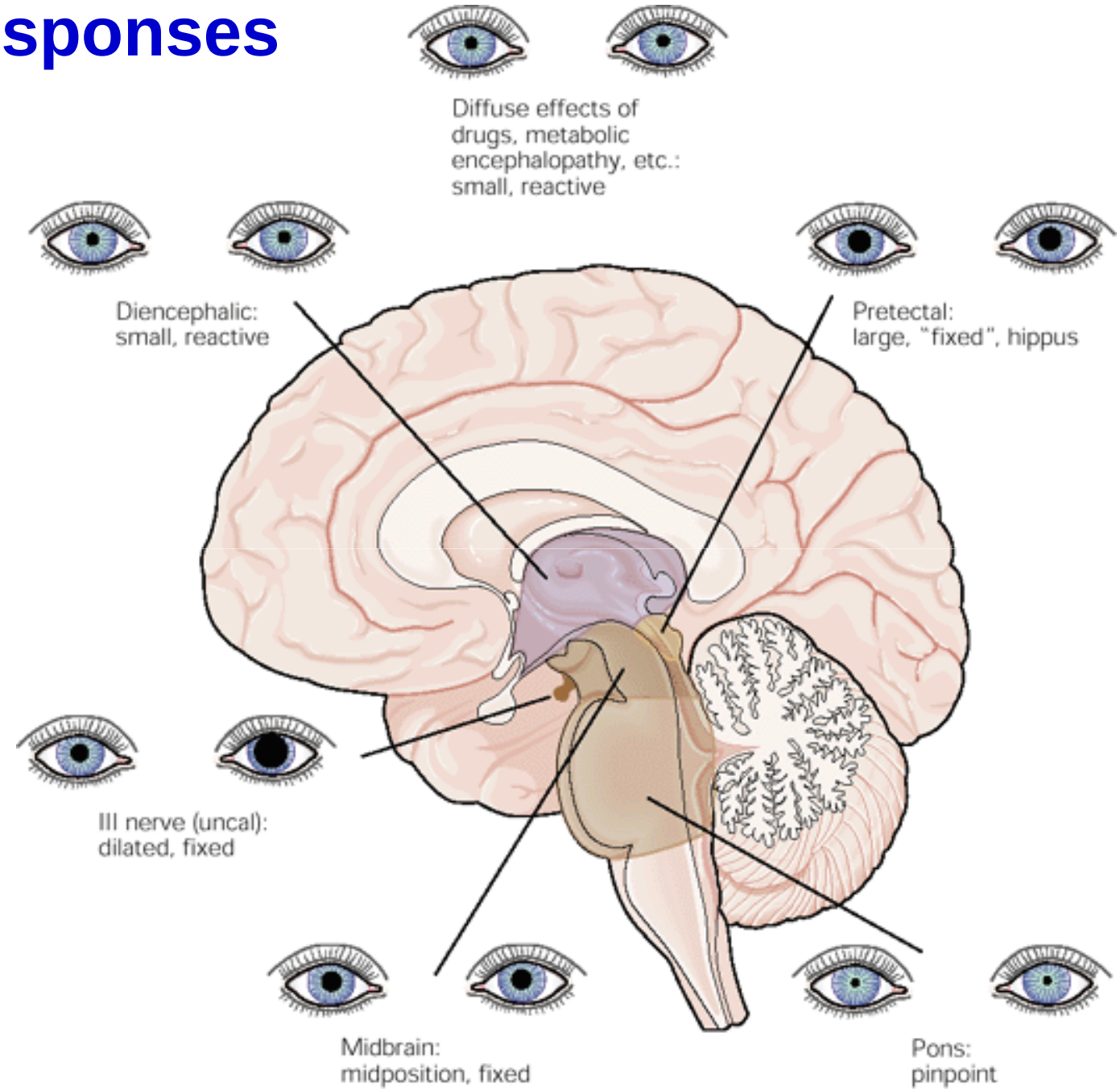
Cheyne stroke	-Bihemispheric damage, metabolic
Kussmaul breathing	-Metabolic acidosis, post mesencephalic lesions
Agonal gasps	-Bilateral lower brainstem lesions
Central neurogenic hyprventilation	-Bihemispheric,midbrain,pons
Apneusis	-Lateral tegmentum of lower pons
Cluster	-Bihemispheric or pons
ataxic	-Dorsomedial medulla RAS







Pupillary responses



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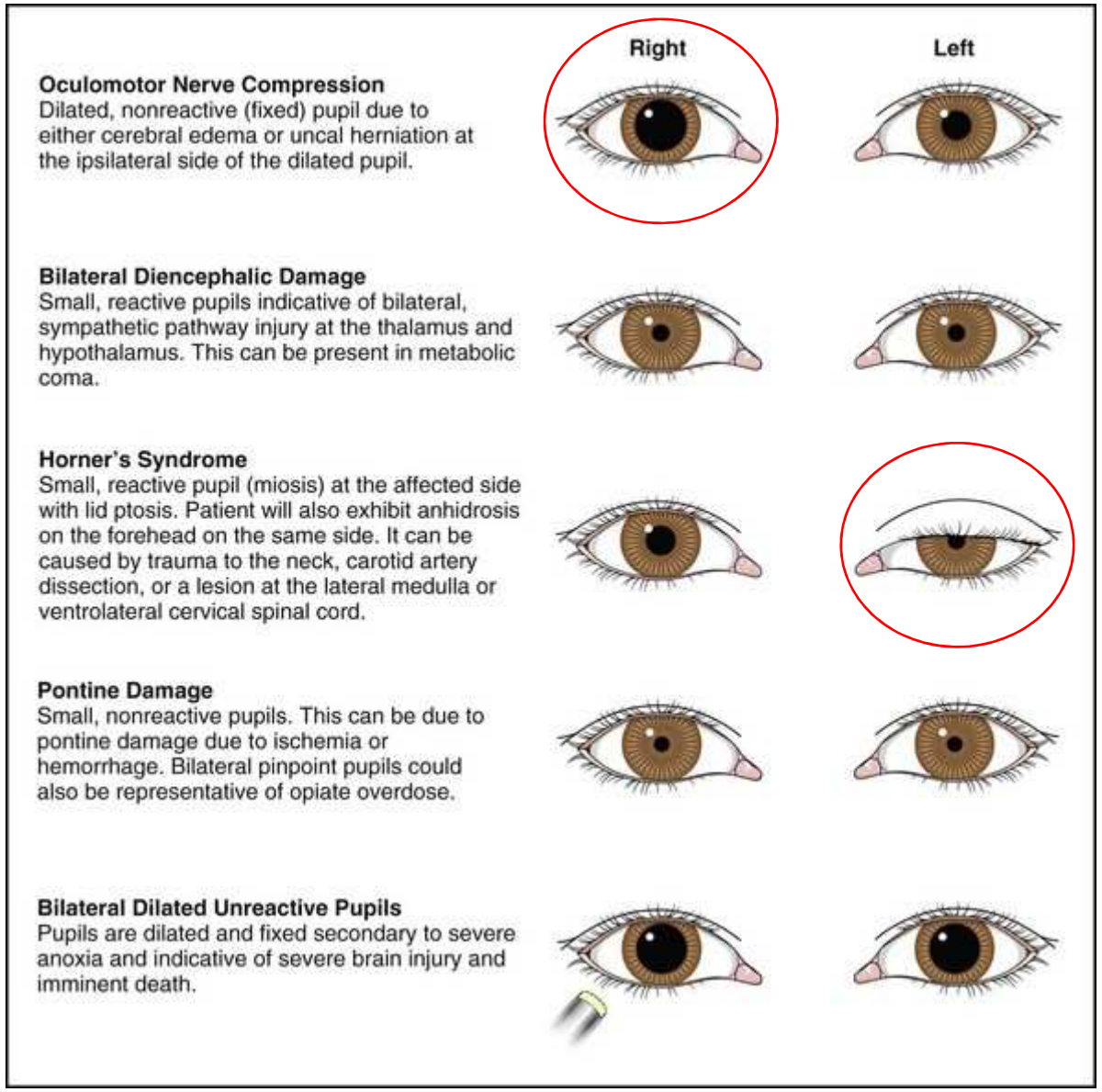
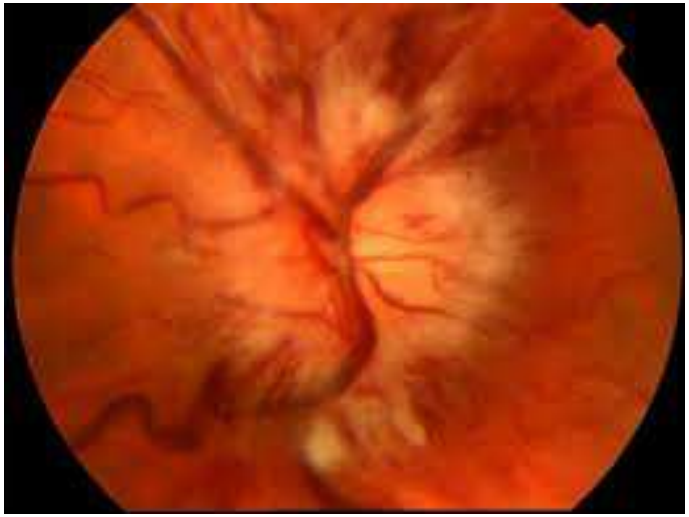
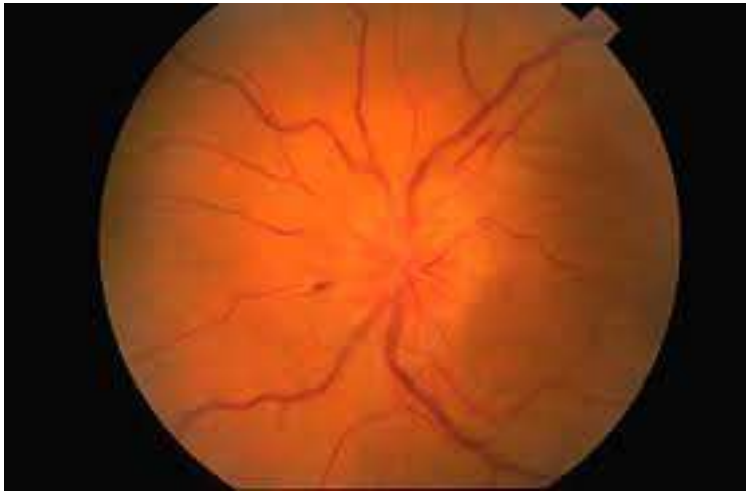


Fundus oculi





Edema della papilla

Wernicke-Korsakoff syndrome

Wernicke-Korsakoff syndrome (also called Korsakoff's psychosis, alcoholic encephalopathy, Wernicke's disease, and alcoholic encephalopathy) is a manifestation of thiamine (vitamin B1) deficiency.

This is usually secondary to alcohol abuse.
It is generally agreed that:
- Wernicke's encephalopathy results from severe ***acute*** deficiency of thiamine, while
- Korsakoff's psychosis is a ***chronic*** neurologic sequela after Wernicke's encephalopathy.

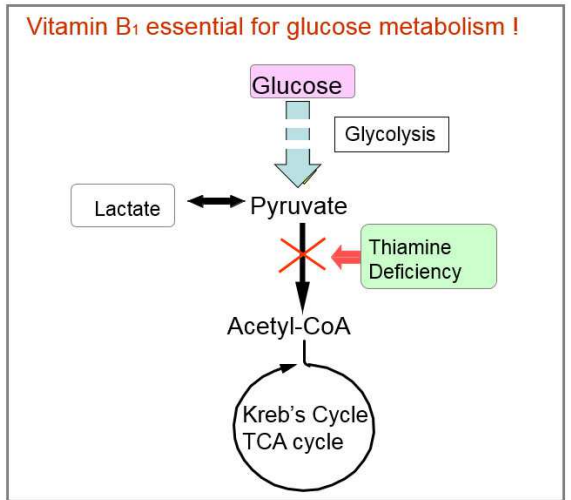
Wernicke-Korsakoff syndrome

Wernicke's encephalopathy is characterized by:
- confusion
- nystagmus
- ophthalmoplegia (impaired eye movement)
- anisocoria
- ataxia
- sluggish pupillary reflexes
- coma and death if untreated

Korsakoff's psychosis is characterized by:
- anterograde amnesia (inability to form new memories)
- retrograde amnesia (loss of existing memories)
- confabulation (false perceptions or memories)
- hallucinations

Wernicke-Korsakoff syndrome

- Thiamine diphosphate plays a major role as a cofactor or coenzyme in ***glucose metabolism***. The enzymes dependent on thiamine diphosphate are associated with the **Kreb's Cycle**. Thus, anything that encourages glucose metabolism will exacerbate an existing clinical or sub-clinical thiamine deficiency.
- Treatment consists of reversing the thiamine deficiency by ***giving supplemental thiamine*** (IV or IM). Some authors think it is important to start thiamine BEFORE giving glucose, as encephalopathy would be worsened by glucose (glucose administration promotes decarboxylation of pyruvate, a biochemical reaction which requires thiamine). However, this is based on case reports.
- By the time amnesia and psychosis have occurred, complete recovery is extremely unlikely.



Adapted from Japanese Society for Parenteral and Enteral Nutrition ed., Guideline on Parenteral and Enteral Comedicals, p61, Nankodo, 2000