

Neurology

Chapter 10 : coma

Learning object 1 : Pathophysiology and Aetiology of coma

objectives



- At the end of this learning object student will be able to:
- Define coma.
 - Explain pathophysiology of coma.
 - Compare between cranial causes and extra cranial causes of coma.
 - Enumerate cranial causes of coma.
 - Enumerate extra cranial causes of coma.
 - Differentiate between different metabolic causes of coma.
 - Compare between respiratory causes and cardiovascular causes of coma.
 - Compare between endocrinal causes, toxins, and drugs causes of coma.

Slide 1 : Pathophysiology of Coma

Pathophysiology: Consciousness consists of two components-

(1)-Awareness (content of consciousness)

: Depend on the activities of the cerebral cortex & thalamus, permitting higher level integration of sensory inputs reaching the cortex leading to reasonable understanding of self & environment.

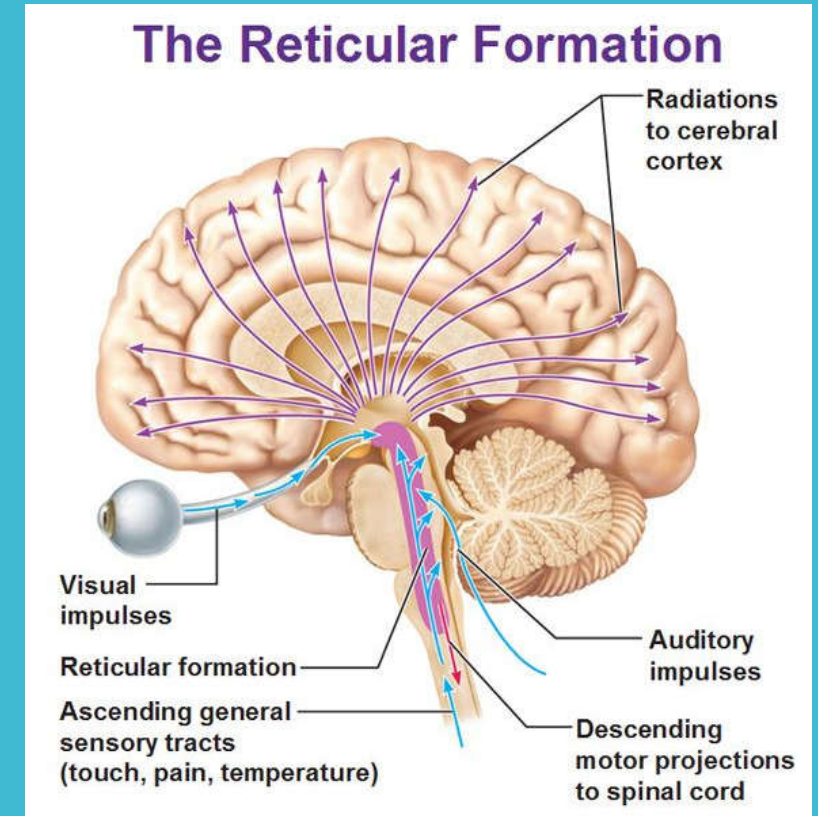
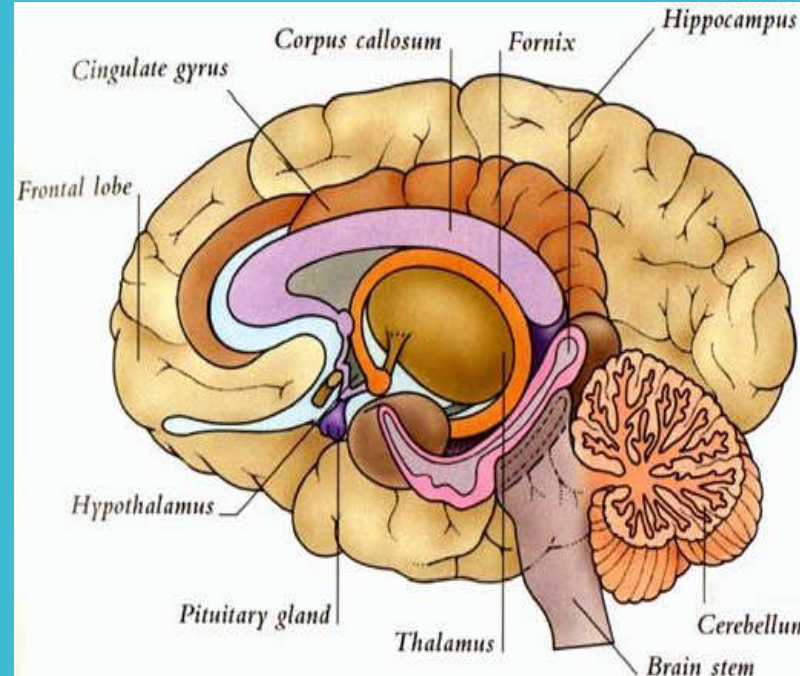
(2)-Arousal (state of consciousness) :

Which is a primitive one depending on the activities of the central reticular formation of the brain stem.

- Thus lesion of the cortex alone affect the content of consciousness (awareness) e.g {emotions, sensations, memories, ideas, experience}, without changing the state of consciousness.

- Therefore coma can be produced by :-

- 1-bilateral or diffuse brain damage.
- 2-brain stem failure or damage
- 3-combined cortical or brain stem failure.



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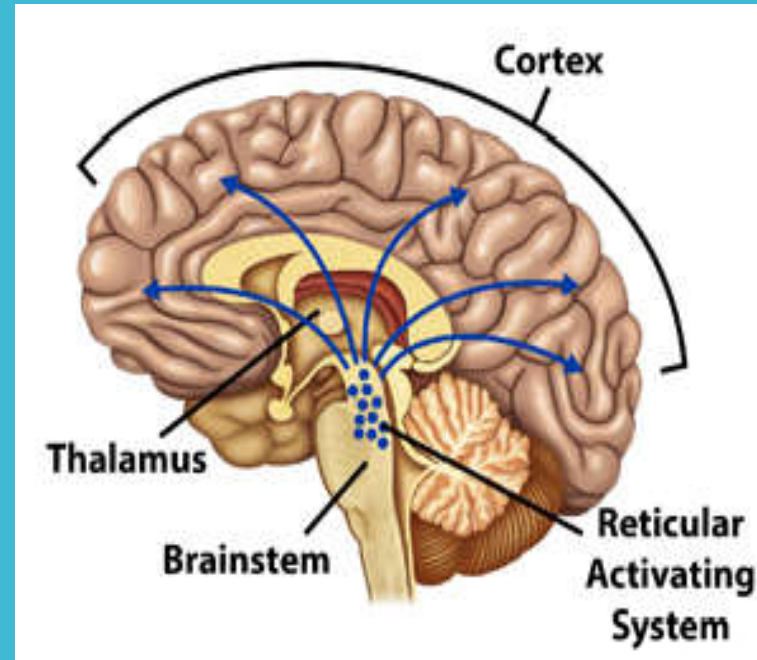
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A side-view of the brain, showing movement of the reticular activating substance (RAS) essential to consciousness. Labels include 'Cortex', 'Thalamus', and 'Mid-brain reticular formation'.

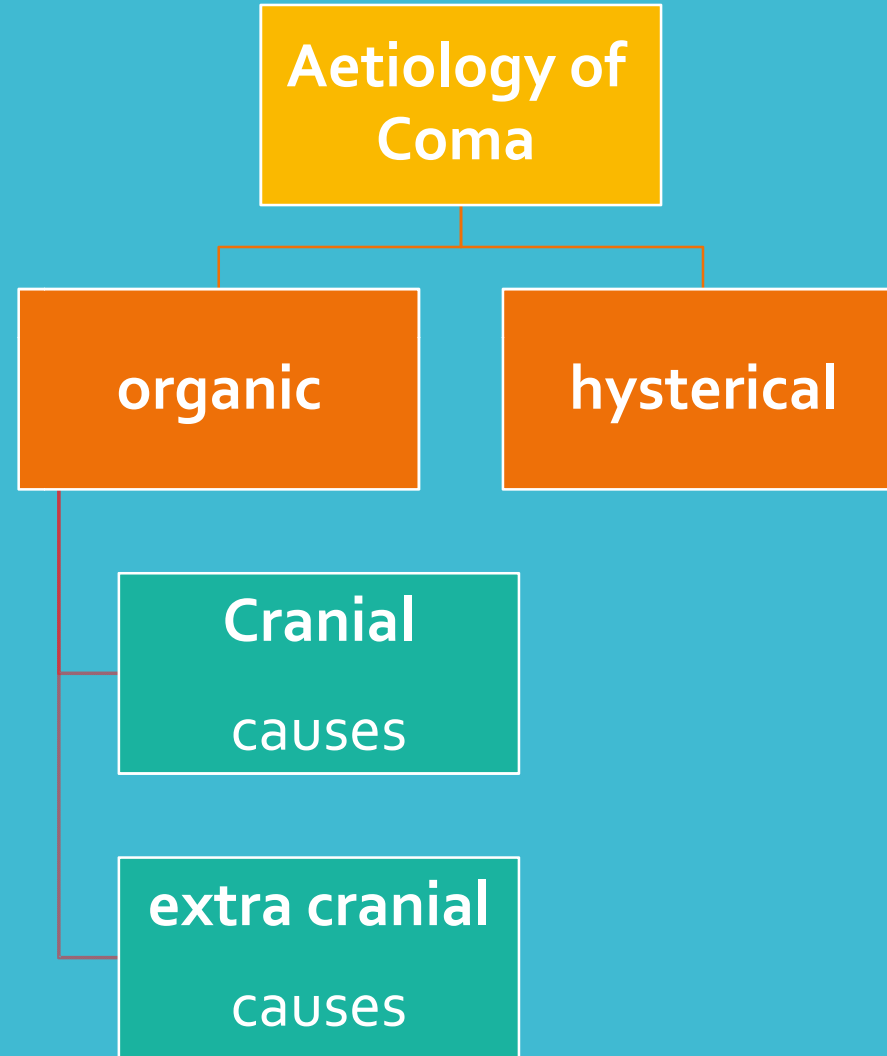
<p>Diffuse and bilateral damage to the cerebral cortex (relative preservation of brain-stem reflexes)</p> <p>Possible causes</p> <ul style="list-style-type: none">• Damage due to lack of oxygen or restricted blood flow, perhaps resulting from cardiac arrest, an anaesthetic accident, or shock• Damage incurred from metabolic processes associated with kidney or liver failure, or with hypoglycemia• Trauma damage• Damage due to a bout with meningitis, encephalomyelitis, or a severe systemic infection	<p>Mass lesions in this region resulting in compression of the brain-stem and damage to the reticular activating substance (RAS)</p> <p>Brain-stem compression</p>
<p>Structural lesions within this region also resulting in compression of the brain-stem and damage to the reticular activating substance (RAS)</p> <p>Local brain-stem pressure Asymmetrical brain-stem signs</p> <p>Possible causes • Cerebellar tumors, abscesses, or hemorrhages</p>	<p>Lesions within the brain-stem directly suppressing the reticular activating substance (RAS)</p> <p>Symmetrical depression of brain-stem reflexes</p> <p>Possible causes • Drug overdose</p>

Slide 2 : Aetiology

Aetiology:

Either → **organic** or → **hysterical**.

Organic causes include → **cranial**
and → **extra cranial** causes.



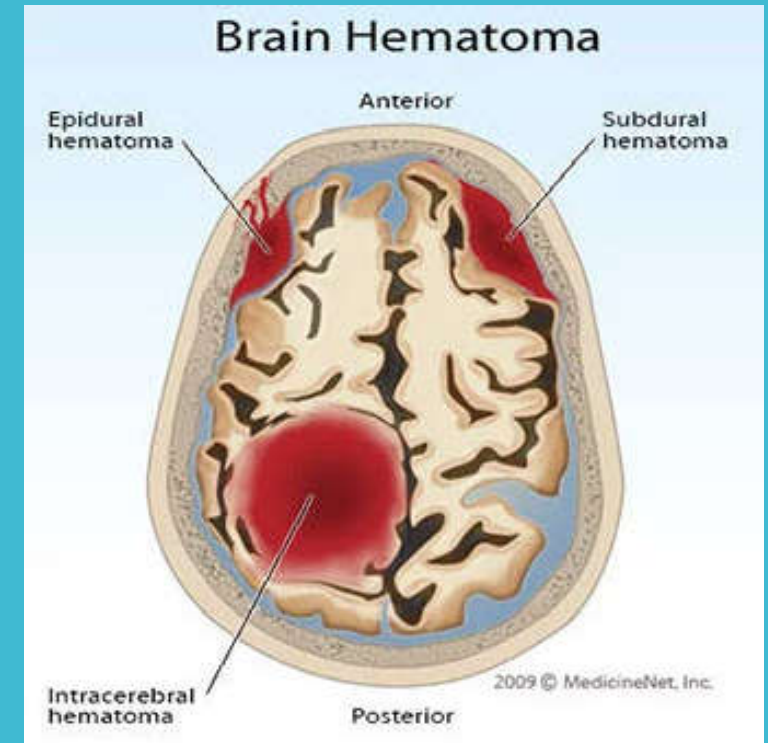
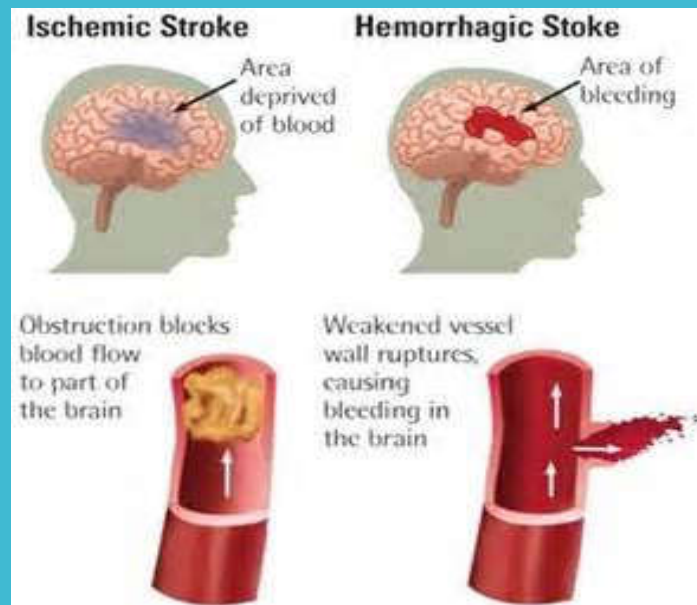
Slide 3 : Cranial causes

(1)-Cranial causes:

- * Trauma: leading to intracranial hge .
- *Cerebrovascular disease: can produce coma directly or indirectly by interfering with the functions of the reticular activating system.

either through:

- Increase ICP.
- Direct pressure of haematoma or edema.
- Impairment of blood supply by e.g :
 - ☐ Massive subarachnoid hge.
 - ☐ Large intracerebral or intracerebellar hge
 - ☐ Large cerebral infarction
 - ☐ Brain stem hge or infarction
 - ☐ Hypertensive encephalopathy
- *Space occupying lesions: as tumors & abscess



Slide 3 : Cranial causes

(1)-Cranial causes:

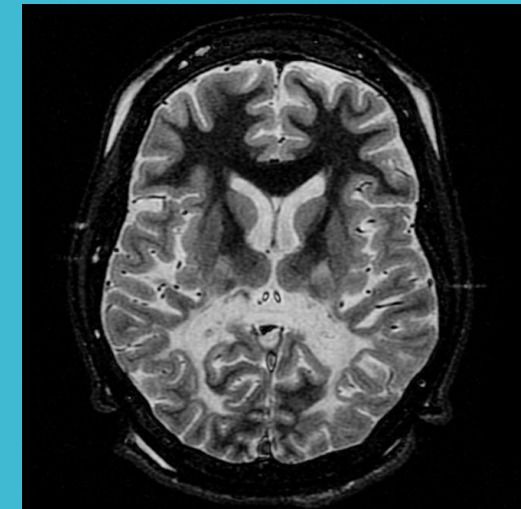
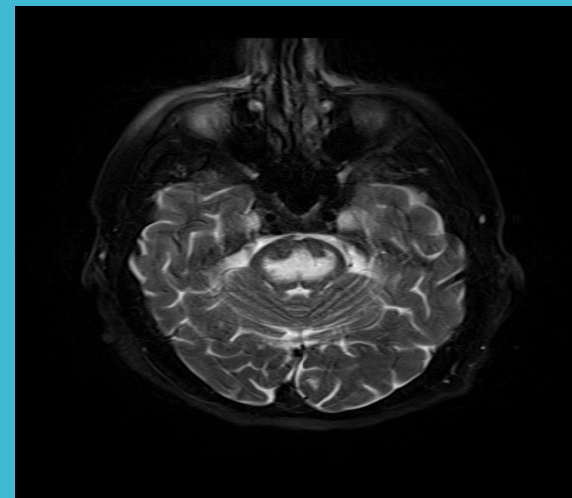
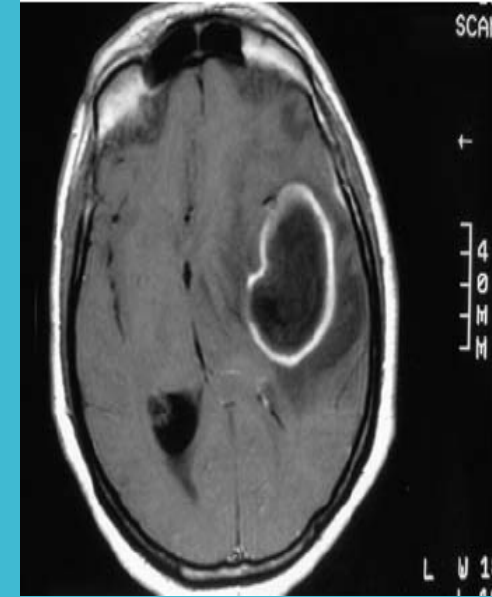
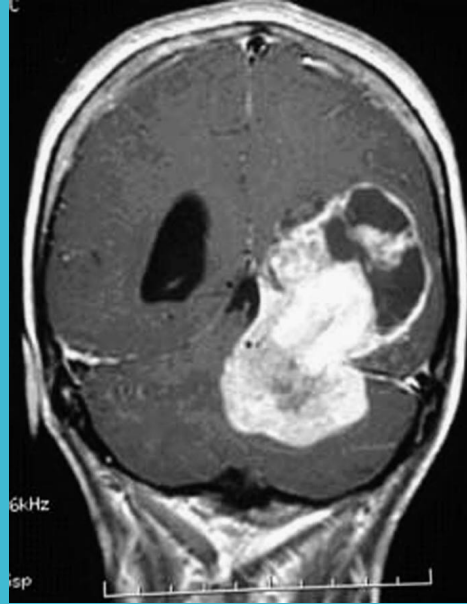
NB. Haemorrhage into the tumor ,
sudden development of edema or
cerebral herniation →coma can occur

*Inflammatory causes:

Meningitis or encephalitis

*Other causes:

- Post ictal Todd's paralysis.
- Leucodystrophy M.S
- Central pontine myelinolysis



Slide 4 : Extra cranial causes

(2)-Extra cranial causes:

A-Metabolic causes:

***Uraemic coma: In acute or Ch.R.F**

due to: –Metabolic acidosis

-Raised blood urea

-Electrolyte disturbance

-water intoxication

-Raised blood pyruvate : →

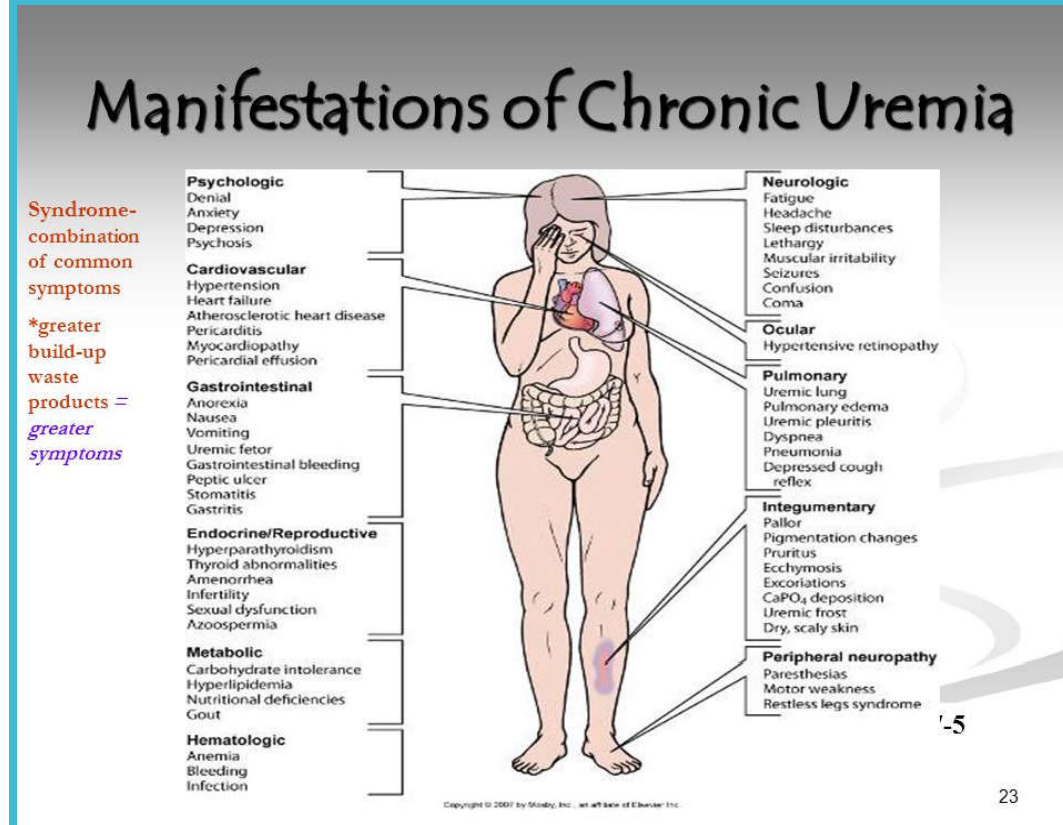
decrease cerebral oxygen

consumption

Cerebral disequilibrium syndrome:

Commoner in children & young adults, characterized by temporary impairment of consc.

Due to rapid change of electrolyte.



Slide 4 : Extra cranial causes

*Hepatic coma:

- Either spontaneously in : Acute viral hepatitis, Eclampsia, Acute chemical poisoning.

- Chronic hepatic Patient with : cirrhosis, Hemochromatosis, Wilson's disease.

-Hepatic coma occur usually due to:

-Increase in blood ammonia → toxic to the brain

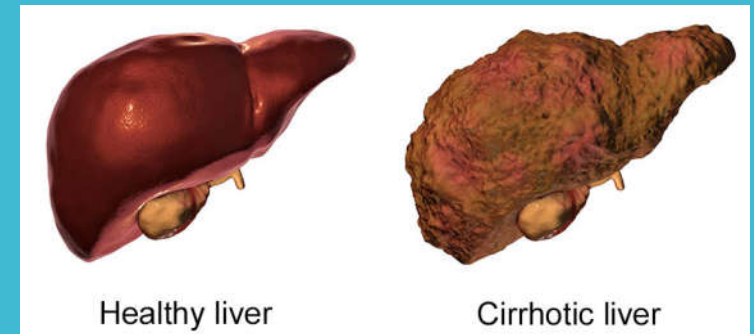
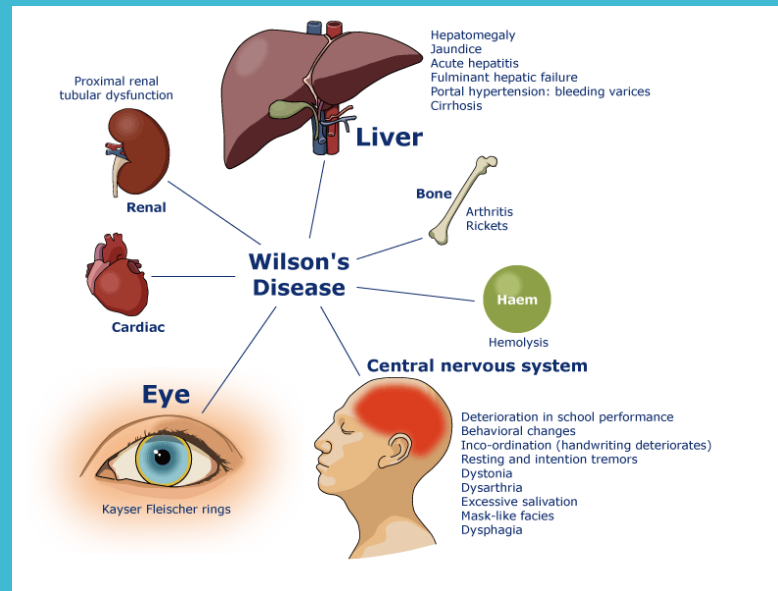
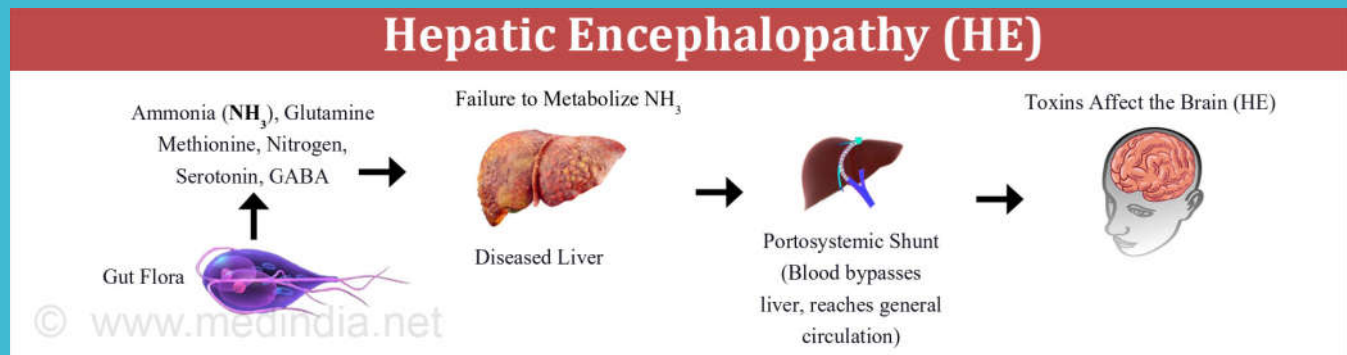
-Electrolyte disturbances

-Increase of circulatory

Amino.Acids

- Cerebral depletion of dopamine

-Increase of false neurotransmitters, as octopamine



Slide 5 : Diabetic comas, Hypoglycemic coma, and Hyperthermia

- *Diabetic comas:** Either
 - Diabetic ketotic coma
 - Lactic acidotic coma
 - Hyperosmolar non ketotic hyperglycemic coma
- *Hypoglycemic coma:**
 - Diabetic pts
 - Hypopituitarism
 - Alcoholism
 - Hepatic patients
 - Addison's disease
- *Hyperthermia (Heat stroke):**
 - After prolonged exertion in hot surroundings
 - Tetanus
 - Pontine he
 - Intraventricular hge
 - Malignant hyperpyrexia as a complication of general anaesthesia.
 - Malignant neuroleptic syndrome as a complication of anti psychotics.



HEAT STROKE

- * Anxiety - Confusion
- * Skin Hot & Dry
- * Impaired Sweating
- * Listlessness
- * Na⁺ and K⁺ Depletion
- * ↑ Body Temperature ↑ 40.6° C or 105° F
- * ↑ Pulse & Resp Rate
- * Hypotension

Cerebral Edema:

- * Seizures
- * Delirium
- * Coma

(Management - Cooling, Rest, Fluid & Electrolyte Support.)

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HYPOGLYCEMIC COMA

The diagram illustrates the management and symptoms of hypoglycemic coma. It features three main elements: 1) A hand holding a small white cube of sugar, labeled "first aid sugar in the mouth". 2) A purple glucometer showing a reading of "1.9", labeled "the reduction of blood sugar levels". 3) A hand with pale, clammy skin, labeled "pale skin cold clammy sweat loss of consciousness".

Slide 6 : Rest of metabolic causes

* Hypothermia:

- Prolonged exposure in cold surroundings
- Hypopituitarism.
- Older patients with arthritis and parkinsonism.
- Drugs as chlorpromazine.
- Shapiro's syndrome.(Recurrent hypothermia with polyuria, polydipsia, hyponatremia, and agenesis of corpus callosum).

*Hypo and hypernatremia.

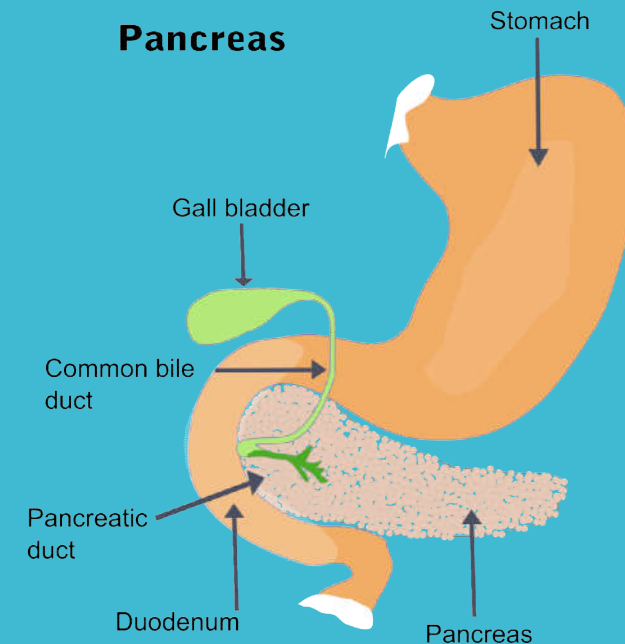
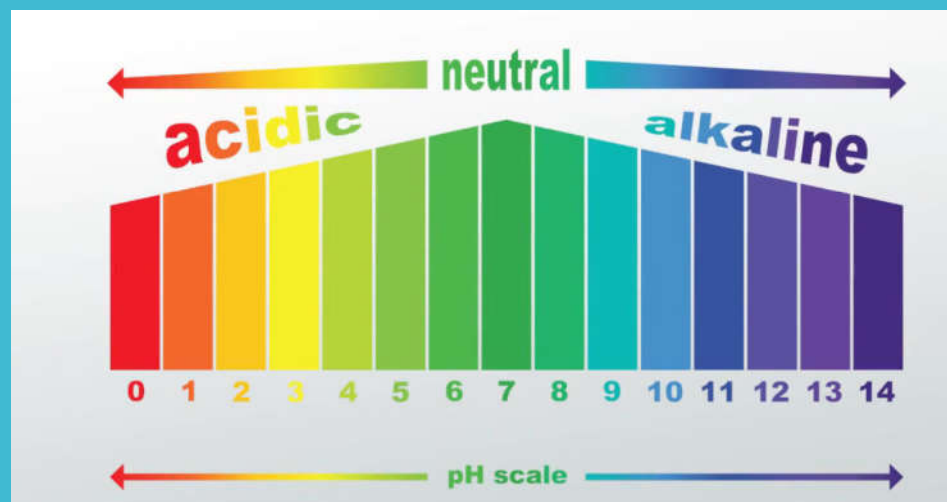
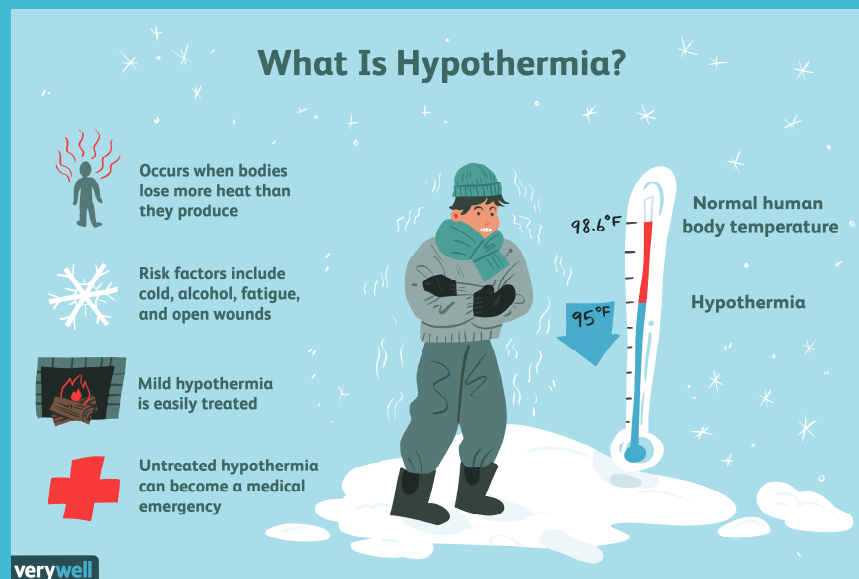
*Hypo and hyperkalemia.

*Hypo and hypercalcemia.

*Acidosis or alkalosis.

*Porphyrias.

*Pancreatic encephalopathy → as in chronic relapsing or acute pancreatitis.



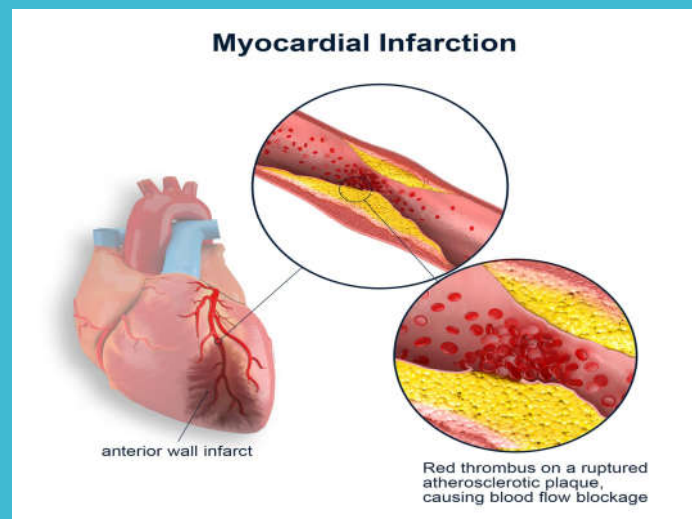
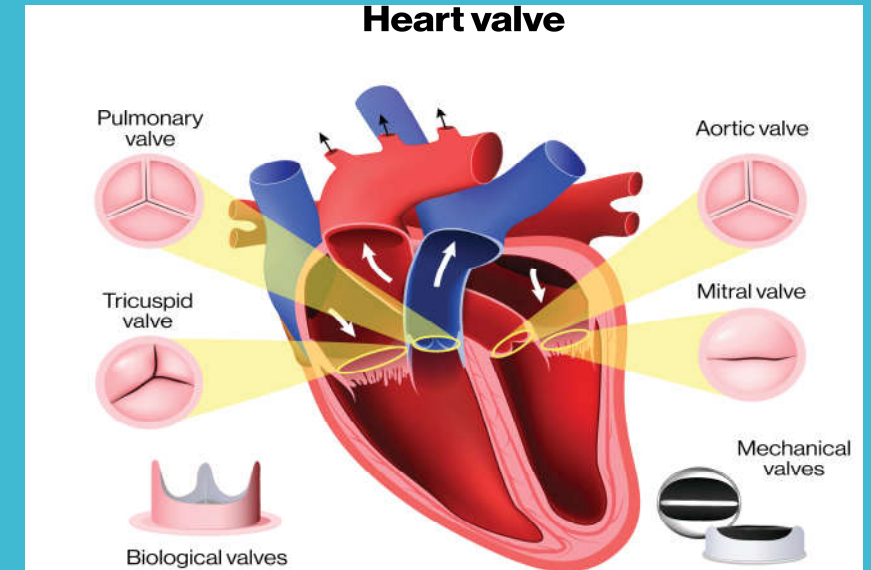
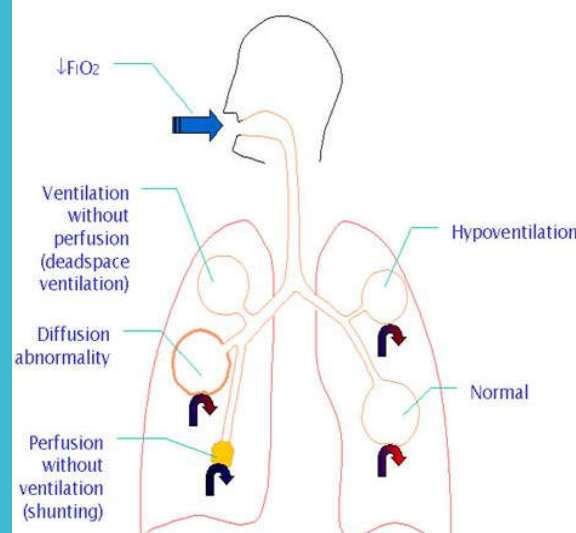
Slide 7 : Respiratory causes and CVS causes leading to cerebral ischemia

B- Respiratory causes:

- *Hypoventilation in COPD leading to anoxia.
- *Diffusion or perfusion defects.

C- CVS causes leading to cerebral ischemia:

- * Cardiac arrest.
- * Severe anaemia.
- * Severe hypotension
- * Myocardial infarction.
- * Valvular heart diseases.
- * Complications of open heart surgery.



Slide 8 : Endocrinal causes, Toxins, and Drugs

D- Endocrinal causes:

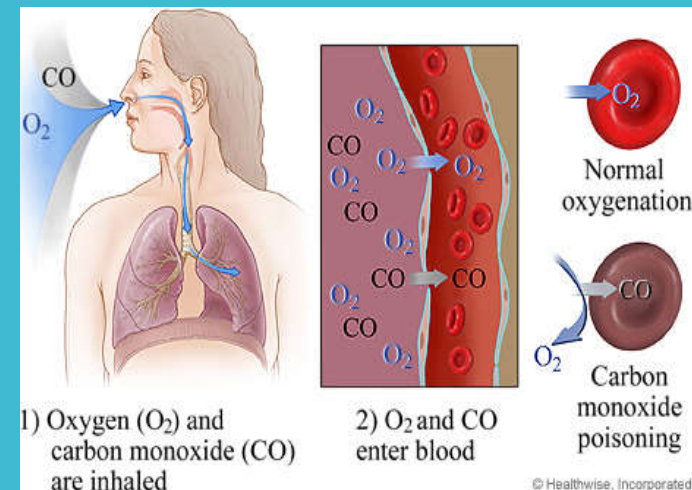
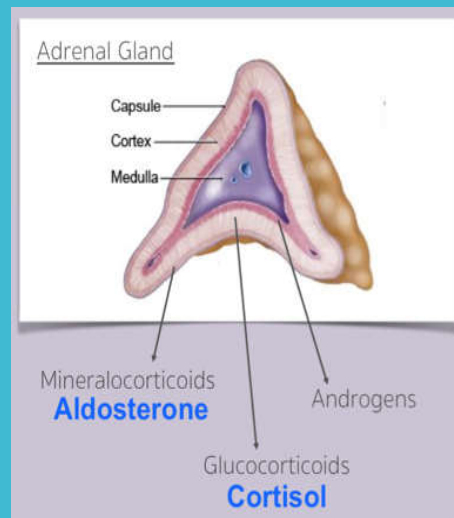
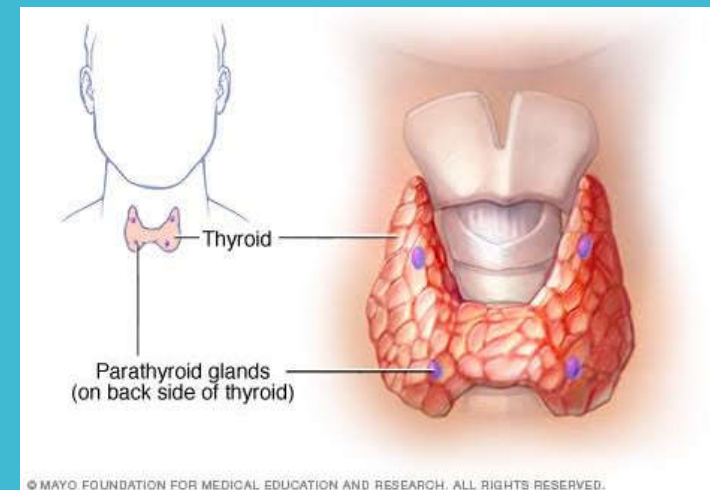
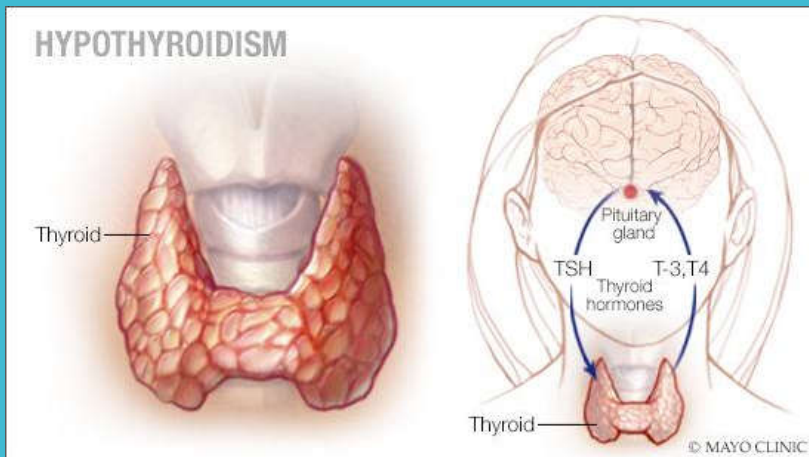
- * Hypopituitarism.
- * Adrenal crisis.
- * Hypo and hyper parathyroidism.
- * Hypothyroidism.

E- Toxins:

- * CO₂ poisoning.
- * CO poisoning.
- * Heavy metals.
- * Alcohol.

F- Drugs:

- * Sedatives and hypnotics.
- * Anticonvulsants.
- * Antidepressants.
- * Anaesthetic agents.



Slide 9 : Grades of impairment of consciousness

Grades of impairment of consciousness:-

Coma: previously defined.

Semi coma: complete loss of consciousness with response only at reflex level.

Stupor: state of impairment of consciousness from which the patient can be aroused by vigorous stimuli.

Lethargy: state of drowsiness and indifference in which increased stimulation may be needed to arouse the patient.

Confusion: impairment of consciousness with poverty of mental process.

Delerium: confusion with excitement.



Slide 10 : Glasgow Coma Scale

Now the **Glasgow Coma Scale** replaced the old classification and has great immediate prognostic value and assessed as following :

Eye-opening:

Spontaneously (with blinking) score (4), To voice score (3), To pain score (2), Nil score (1).

Verbal response:

Orientated score (5), Disorientated score (4), Words score (3), Groans score (2), Nil score (1).

Motor response :

Voluntary score (6), Localizing score (5), Withdrawal score (4), Flexion score (3), Extension score (2), Nil score (1).

Eye-opening	score	Verbal response	score	Motor response	score
Spontaneously (with blinking)	4	Orientated	5	Voluntary	6
To voice	3	Disorientated	4	Localizing	5
To pain	2	Words	3	Withdrawal	4
Nil	1	Groans	2	Flexion	3
		Nil	1	Extension	2
				Nil	1

summary

In this learning object, we presented Coma, we discussed the pathophysiology of coma, then we presented the Aetiology of coma that contained cranial causes like cerebrovascular disease, and Extra cranial causes that regrouped metabolic causes like uremic coma, hepatic coma, and hyperthermia, also regrouped respiratory causes, cardiovascular causes, endocrinal causes, toxins, and drugs. Finally, we presented the different grades of impairment of consciousness like coma, semi coma, stupor, and delirium.