Toxic Coma

Presented

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Introduction

Conscious state is awareness and arousal.

Awareness

- Receive and process all the information communicated by the five senses.
- It consists of **psychological** and **physiological** components.
- The **psychological** component is controlled by the mind and mentality .
- The **physiological** component is function of brain (physical and chemical).
- Awareness is regulated by cortical areas within the cerebral hemispheres,

Arousal

- It is regulated by physiological function .
- It consists of involuntary responses to stimuli.
- It is maintained by the reticular activating system (RAS).

Reticular activating system (RAS)

- It is not an anatomical area of the brain.
- It is a network of structures (brainstem and thalamus) and nerve pathways, which function together to produce and maintain arousal.

Definition

Coma

- It is a state of profound loss of conscious .
- It is characterized by :-

- No spontaneous eye openings (Loss of voluntary movement)
- No response to painful stimuli and speech.
- No arousal (Loss of normal reflexes)

- It is a result of any agent that interferes with the function of cerebral cortex or function of RAS (brainstem and thalamus)

Causes &Types

1- Anatomic al (structural)

- Damage of brain structures (cerebral cortex ,brainstem)
- Lateralizing signs (Unequal pupil size , Asymmetry of tone and deep reflexes)

Etiology :

Trauma (head injury) - **Space Occupying Lesion** (tumor, cerebral edema, hematoma) - **Vascular** disease (thrombosis, hemorrhage, embolism)

2-Toxic - Metabolic encephalopathy

- Change in the function and chemical of brain .

Etiology :

Hypoxia (Co, cyanide) **Hypo-hyperglycaemia**, **metabolic acidosis** (uncontrolled diabetes), **Electrolyte abnormalities** (hypo-hypernatremia)

Endocrine (hypothyroidism), **Hypo–hyperthermia** (heat stroke)

Toxins and drugs overdose

Toxins (internal & external) affect the function of neurons.

- **External** (Drugs or Alcohol)
- **Internal** (Ammonia, Urea)

3- Infection with encephalitis (Septic Coma)

4- Seizures - Electrical disturbance leads to changes in chemical levels of brain

5- Alpha coma - Dominant alpha-wave activity in EEG.

6-Irreversible coma

Brain death (irreversible arrest of all brain function).

7- Coma vigil Locked in syndrome

It is a rare neurological condition: Awake and alert associated with a normal mind and total paralysis except the eye muscles.

8- Persistent Vegetative State

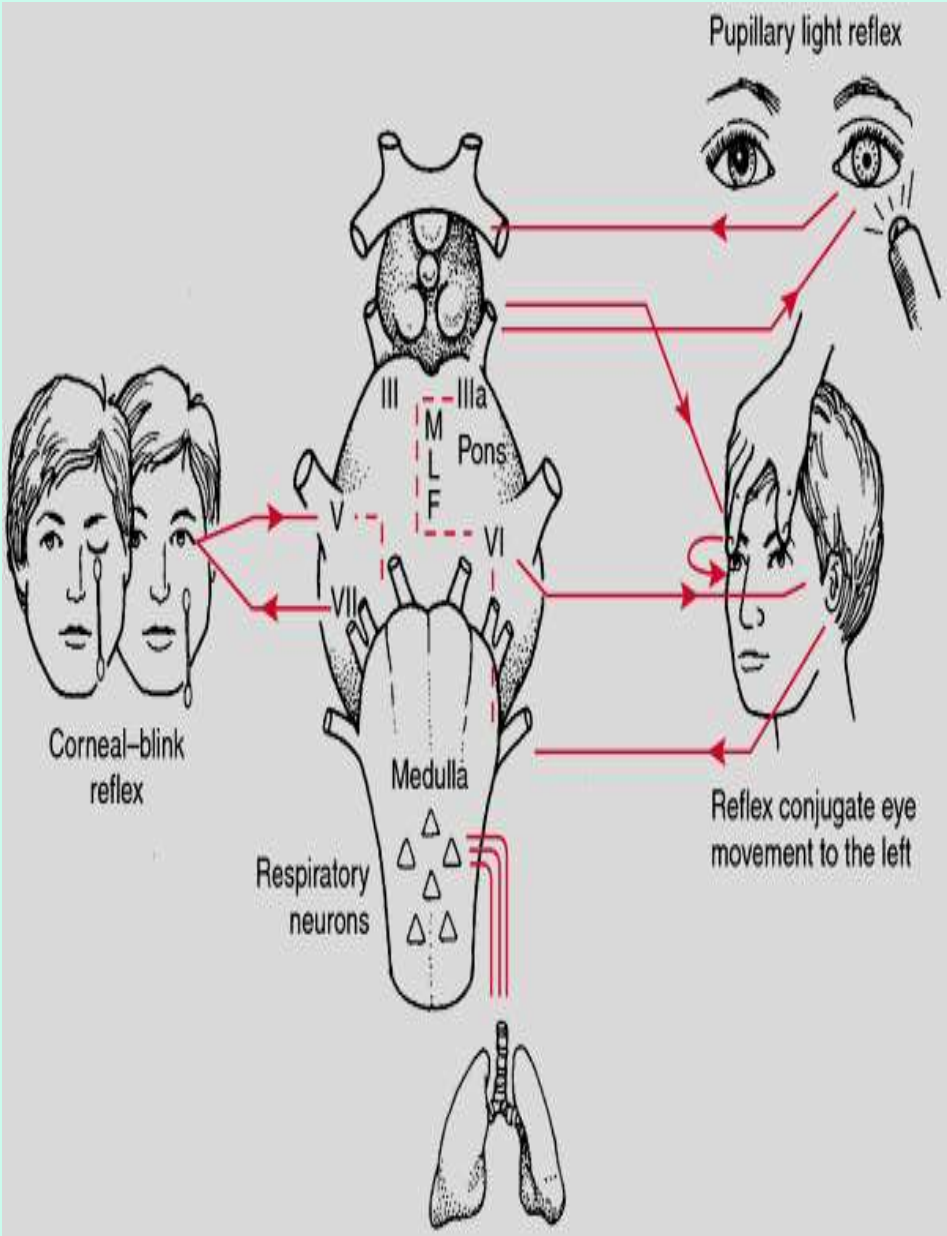
Intact functions of the brain stem (Respiration and circulation)

9- Anoxic Brain Injury.

It results from cardiac arrest, head injury or trauma, drowning, drug overdose, or poisoning.

Common Toxic Agents

1. Opiate
2. Barbiturates
3. Benzodiazepines
4. Neuromuscular Blockers
5. Tricyclic Antidepressants
6. Hallucinogens
7. Anti cholinergic Drugs
8. Alcohol
9. Co - Cyanide
10. Organophosphorous



Management of Comatose Patient

1- Life - Saving Measures:

A- Airway

B- Breathing

C- Circulation

2-Assessment

- Level of conscious (Glasgow Coma Scale).
- Diagnosis (History, Neurological Exam, Investigations).
- Intervention according to the cause.

Glasgow Coma Scale

It measures the depth of coma .

- **Eye** Opening
- **Vocal** Response
- **Motor** Response

Ratings range from **3** to **15** .

1- Total rating is **3 - 5** = **Very Severe Brain Injury**

2-Total rating is **6 - 8** = **Severe Brain Injury** (still in coma)

3- Total rating is **9 - 15** = **Brain Injury Out of Coma**

Total rating of **9 - 12** = **Moderate TBI**

Total rating of **13 - 15** = **Mild TBI**

Glasgow Coma Scale

Eye Opening

Spontaneous	4
To loud voice	3
To pain	2
None	1

Verbal Response

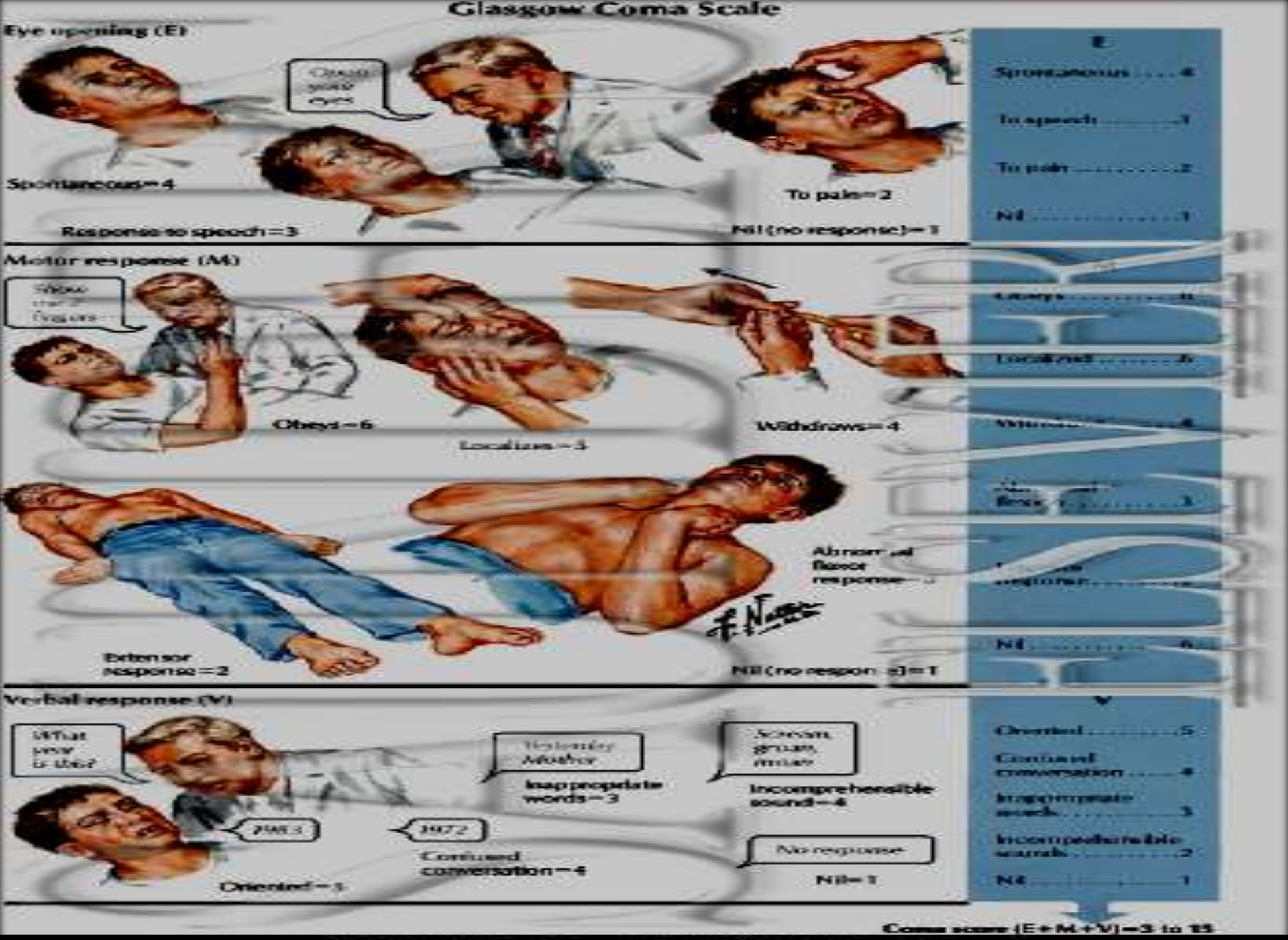
Oriented	5
Confused, Disoriented	4
Inappropriate words	3
Incomprehensible words	2
None	1

Motor Response

Obeys commands	6
Localizes pain	5
Withdraws from pain	4
Abnormal flexion posturing	3
Extensor posturing	2
None	1

- **A fully awake patient has a Glasgow Coma Score of 15 .**
- **A dead person who has a Glasgow Coma Scale of 3 (there is no lower score).**

Glasgow Coma Scale



Barbiturates Poisoning

- It is a Sedative – Hypnotic drugs
- **Classification** according to duration of action.
- **Duration of action** depends on:-

1. **Rate of metabolism**
2. **Rate of excretion**
3. **Distribution properties (Lipid soluble , Protein Binding, Ionization)**

- **More lipid soluble, More binding , Non ionization= More distribution**
  - **Long acting** (6-12 hr)
    - **Phenobarbitone – blood and urine**
  - **Intermediate acting** (4-6 hr)
    - **Amy barbital- urine**
  - **Short acting** (3 hr)
    - **Secobarbital- urine**
  - **Ultra short acting** (15-30 M)
    - **Thiopental – urine**

Acute Barbiturate Poisoning

Significant Toxicity

Long Acting= **4** mg/dl **Short** Acting = **2** mg/dl .

Acute Barbiturate Poisoning

It may cause acute brain death because of a prolonged hypoxia .

Clinical Picture :-

1. Deep prolonged coma
2. Loss of reflexes (deep tendon reflex)
3. Dilated pupil
4. Slow respiration or rapid shallow(cheyne stoke), Cyanosis
5. Hypotension-Weak rapid pulse
6. Hypothermia
7. Nephritis (hamaturia, albuminuria)
8. Skin rash

Chronic Barbiturate Poisoning

1. Amnesia
2. Tremor
3. Ataxia

Cerebellar Affection (Incoordination - Slurring Speech)

1. Rash
2. Renal affection

Haematuria- Albuminuria

Management of Barbiturate Poisoning

1- Life Saving Measures

2- Symptomatic Treatment

3- Assessment

4- Investigations

Plasma Phenobarbitone Level

Renal Function Tests

E.C.G - Arterial Blood Gases

4- Git Decontamination (Lavage , Charcoal)

5- Elimination

- Forced alkaline diuresis
- Peritoneal dialysis
- Haemodialysis
- Haemoperfusion

Questions

- *What is the action of forced alkaline diuresis in the drug elimination?*

- Increase the glomerular filtration rate and then increase the drug excretion
- Decrease the renal tubular reabsorption by the drug ionization

- *Why can we use forced alkaline diuresis in long-acting barbiturate poisoning?*

- Because long acting barbiturate is less lipid soluble, less protein binding and then less distributed

- *When can we use Git decontamination in treatment of barbiturate poisoning?*

- Within 6- 12 hrs. because of the delayed gastric empty

- *What are the indications for haemodialysis use in barbiturate poisoning ?*

- Plasma Phenobarbitone Level exceeds 10mg/dl , and 5mg/dl for short acting+ Late stage of coma
- No response to the intensive care
- Suspected brain death
- According to the Clinical condition of the patient

1- Good patient condition + high Plasma drug Level (tolerance) = No Dialysis

2- Bad patient condition (multiple agents) + low Plasma drug Level = Dialysis

Thank you