Coma and Consciousness

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The concepts of consciousness, coma and wakefulness have fascinated man from the ages [1-3]. The explanation and thereby, understanding of different states of altered consciousness vary from mystic, spiritual to anatomic and scientific. The advent of experimental neurology and anatomy gradually expanded the scientific horizons of man allowing observations and measurements to be made and interpreted.

An epoch breaking understanding occurred with the discovery of the Ascending Reticular Activating System (ARAS), by an Austrian Nobleman Baron Constantin von Economo who demonstrated the seminal influence of the ARAS in wakefulness in animal models. Further research discovered the presence of Orexin as well [4-6] as Seratonin influencing the neurotransmitter release in the ARAS as well as adjoining sections of the Hypothalamus Figure 1.

Consciousness is thus defined as a state of full awareness of the self and one’s relationship to the environment. It was considered to consist of 2 components:

1. Arousal or the level of behaviour and innate responsiveness
2. Content or the sum of functions mediated by the cerebral cortex (cognitive and effective)

Consciousness was hence the ability to stay awake (arousal) as well as the sum total of all the thoughts and activities planned while awake (content).

Hence altered states of consciousness can be defined as

1. Disorders of Arousal such as lethargy, stupor, coma and
2. Disorders of Content such as Confusion, Hallucinations, emotional changes etc.

An important differentiation is to be made between organic causes of altered consciousness and Psychogenic unresponsiveness. Here patients although apparently unconscious show some response to external stimuli. For example, an attempt to elicit the corneal reflex may cause a vigorous contraction of the orbicularis oculi. There will also be marked resistance to passive movement of the limbs without signs of any disease/ organ damage to justify the state of consciousness.

Altered states of Consciousness include

PLUM and POSNER Figure 2.
Confusion
Here the major defect is a lack of attention with disorientation to time place and person. The patient thinks less clearly and more slowly with memory difficulties as well. There is also a Misinterpretation of external stimuli [6-10]. Drowsiness may alternate with hyper -excitability and irritability as well.

Torpidity
Obtundd sensorium or mental blunting

Stupor
A condition of deep sleep or behavioral unresponsiveness from which arousal is only possible through violent stimulus.

Clouding of consciousness
Minimally impaired awareness or wakefulness, which may alternate with hyper excitability.

Delirium
A disturbance of consciousness with impaired ability to focus, sustain or shift attention. It also involves a disturbance of cognition or Perception marked by disorientation, fear, irritability, and misperception of sensory stimuli. The patient is out of true contact with environment and other people.

Common causes: Toxins, metabolic disorders, partial complex seizures, head trauma, acute febrile systemic illnesses

Coma
A state of severely impaired consciousness from which the patient cannot be aroused even with vigorous stimuli.

Locked in Syndrome
A situation where the brain is differentiated, by which response is impossible, but perception is intact.

Dementia
An enduring and progressive decline in mental prowess owing to an organic pathological process. Arousal is usually spared. There is a marked impairment of executive function localized to the cortex.

Akinetic Mutism
State of silent, yet alert appearing immobility (anterior Basal forebrain and hypothalamus lesions)

Abulia
This implies a “lack of will” or slow response (if at all) to verbal stimuli, and inability to initiate activity. Cognition is unaffected. (localization is to bilateral frontal lobe involvement)

Hypersomnia
A state of excessive sleep from which the subject is readily arousable, but into which he relapses into readily. (hypothalamus is generally involved)

Minimally concious state
A condition of severely impaired consciousness in which minimal but definite behavioral evidence of self or environmental evidence is demonstrated. It is generally considered a transitional state during recovery or worsening.

Persistent Vegetative State
This denotes the recovery of the crude arousal cycle. Visceral regulation persists but content is absent. The term is used if the state persists for more than 30 days. Also known as Coma Vigil, and Apallic state Figure 3.

Breathing Patterns In Patients of Altered Consciousness

Cheyne-Stokes Pattern
This indicates diffuse hemispherical damage and localizes the lesion to the diencephalon.

Persistant Neurogenic Hyperventilation
This denotes a persistant hyperventilation without other more common causes such as hyperthermia, acidosis, or hypoxia. It denotes a dysfunction at the level of the rostral midbrain.

Apneusis
This implies a mid to caudal pons localization with periodic peaks and troughs in the breathing rhythm without regularity or predictability of amplitude.

Apneusis
Ataxic or Bitot’s Breathing: This indicates a lower medullary damage and is a chaotic mix of breathing patterns without gainful oxygenation. It is often considered a pre-terminal event.

Signs of Lateralization
Probably the most important signs required to picked up by any resident of nurse in the neurosurgical ICU or emergency are signs of lateralization which imply imminent herniation of either a part or the entire brain in the direction of the force vector it is subjected to due to a variety of pathologies present. These include:

1. Unequal Pupils: These are by far the most significant and are a sign of impending Uncal Herniation
2. Gaze preference
3. Unilateral hypertonia
4. Asymmetrical deep tendon reflexes exaggerated
5. Unilateral Babinski reflex
6. Partial seizures

Causes of altered Consciousness Structural causes
1. Hematomas
2. Infarcts
3. Tumors
4. Abscess
5. Contusions
6. Gliomatosis
7. Hydrocephalus
8. ADEM
9. Leukoencephalopathy

Metabolic causes
1. Dyselectrolytemias
2. DM
3. Liver disease
4. Renal disease
5. Endocrinopathies
6. Poisoning
7. Drug abuse/overdose
8. Seizures
9. Psychiatric unresponsiveness

Common Causes Encountered In Neurosurgical and Neurocritical

CARE include
- METABOLIC COMA: These include:
  - Diabetic coma: which maybe hyperosmolar non--ketotic or hypoglycemic in nature
  - Renal coma: which includes hyperuricemia as well as azotemic states. Of special mention is Dialysis Dysequilibrium syndrome where a complete loss of electrolytes and solutes leads to neuron swelling and encephalopathy.
  - Hepatic coma: well known as detectable in liver cell failure where bilirubin deposition in the basal nuclei leads to extrapyramidal states.
  - Thyroid coma: due to a complete fall of the BMR secondary to thyroid burnout or suppression (Myxedemic Coma)
  - Adrenal coma: secondary to insufficiency of cortisol and mineralocorticoid production in the face of rising demand.
  - Dyselectrolytemia: these include HypoNatrexia, Hypernatrexia as well as associated conditions such as:

SIADH

- ADH release is related to the threshold of the thirst response and there is a lower threshold for thirst in patients with SIADH.
- There is also loss of control of ADH release and plasma.
- SIADH is often a self-limiting disease after brain injury and treatment should only be initiated if the patient is symptomatic, the serum sodium is significantly low or falling rapidly.
- Seizures

Cerebral salt wasting

- CSWS is characterized by renal loss of sodium resulting in polyuria, natriuresis, hyponatraemia, and hypovolaemia occurring as a result of a centrally mediated process.
- It is predominantly associated with SAH and TBI but has also been described after brain tumour surgery, ischaemic stroke, and TB meningitis.
- It usually occurs in the first week after brain injury and resolves spontaneously within 2–4 weeks.
- The primary treatment of CSWS is volume and sodium resuscitation.
- CSWS may be refractory to standard therapy and fludrocortisone (0.1–0.4 mg daily) may limit the sodium loss by increasing sodium reabsorption.

Diabetes Insipidus

- DI results from a failure of ADH release from the hypothalamic–pituitary axis.
- The ability to concentrate urine is impaired resulting in the production of large volumes of dilute urine.
- This inappropriate loss of water leads to an increase in serum sodium and osmolality and a state of clinical dehydration.
- Management: fluid intake as per thirst in conscious patients and like for like replacement in comatose patients.
- Refractory cases require desmopressin.
- Toxin intoxication (the most common being alcohol)
- Neurological diseases

Bibliography


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