## Coma and other Stuporous States

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## Conflicts

• No conflicts of interest



## Learning objectives

- Definitions
- Coma examination
- ABCs
- Evaluate cause of coma and treat presumptive cause(s) simultaneously
- Case presentation



## What is coma

- Arousal wakefulness, vigilance (ie. Eye opening)
- Awareness ability to interact with internal/external stimuli (ie. follow commands)
- State characterized by absence of arousal and awareness (unresponsive to external stimuli) for > 1 hour ie. Not awake or aware





## **Other Definitions**

- Vegetative state/ Unresponsive Wakefulness Syndrome
  - Awake but not aware Return of wakefulness but no awareness to external environment ie. No cognition
  - Sleep wake-cycles exist
  - "Lights are on but no one is home"
- Persistent vegetative state
  - Subjects who have remained in a vegetative state for > 3 months (durations may vary in different parts of the world)
- Minimally conscious state
  - Awake with some awareness with behavioral evidence of consciousness (localize to sounds, eye fixation, smiling or crying, verbilization)
  - Usually inconsistent exam



# Examination of a comatose patient – The coma exam

- 4 big components "FOUR score":
- ➤Level of arousal
- Brainstem assessment
- ➢Motor response
- Breathing patterns



FOUR Score

#### Eye response

- 4 = eyelids open or opened, tracking, or blinking to command
- 3 = eyelids open but not tracking
- 2 = eyelids closed but open to loud voice
- 1 = eyelids closed but open to pain
- 0 = eyelids remain closed with pain

#### Motor response

- 4 = thumbs-up, fist, or peace sign
- 3 = localizing to pain
- 2 = flexion response to pain
- 1 = extension response to pain
- 0 = no response to pain or generalized myoclonus status

#### Brainstem reflexes

- 4 = papillary and corneal reflexes present
- 3 = one pupil wide and fixed
- 2 = papillary or corneal reflexes absent
- 1 = papillary and corneal reflexes absent
- 0 = absent papillary, corneal, and cough reflex

#### Respiration

- 4 = not intubated, regular breathing pattern
- 3 = not intubated, Cheyne-Stokes breathing pattern
- 2 = not intubated, irregular breathing
- 1 = breathes above ventilator rate
- 0 = breathes at ventilator rate or apnea

## FOUR Score for Unresponsiveness



# Neurologic exam: Level of arousal

- Spontaneous opening of the eyes, visual fixation or pursuit (tracking), spontaneous and purposeful movements of the extremities
- Glasgow Coma Scale (GCS) Eye opening, motor response, verbal response

#### OR

 Full Outline of UnResponsiveness (FOUR) score – Eye opening, motor response, brainstem function, respiration

Glasgow Coma Scale for Head Injury	
<b>Glasgow Coma Scale, Eye opening</b> Spontaneous To loud voice To pain None	4 3 2 1
<b>Verbal response</b> Oriented Confused, disoriented Inappropriate words Incomprehensible sounds None	5 4 2 1
<b>Best motor response</b> Obeys Localizes Withdraws (flexion) Abnormal flexion posturing Extension posturing None	6 5 4 3 2



# Neurologic exam: Brainstem assessment

• Pupillary response (CN II, III)





# Neurologic exam: Brainstem assessment

- Corneal Reflex
- Blink to threat
- Cough Reflex
- Gag Reflex
- Eye movements
- ≻Spontaneous
- Oculocephalics / Vestibulo-ocular reflex





## Neurologic exam: Motor Exam

- Movement spontaneous/ purposeful vs. to stimulus (quantify what degree of stimulus required) vs. reflexive
- Posturing decerebrate vs. decorticate
- Tone
- Deep tendon reflexes





## Neurologic exam: Motor





Figure 13. Motor responses to noxious stimulation in patients with acute cerebral dysfunction. Noxious stimuli can be delivered with minimal trauma to the supraorbital ridge, the nail bed, or the sternum as illustrated at ton. Levels of associated brain dysfunction are roughly indicated at left. The text provides

## Neurologic exam: Breathing Patterns

- Can be helpful in localization but often times difficult to see when mechanically ventilated
- Common patterns:

A. Cheyne-Stokes – cortex
B. Neurogenic hyperventilation – midbrain/upper pons
C. Apneusis – mid-pons
D. Cluster (Biot's) breathing – lower pons
E. Ataxic breathing- lower pons/ medulla





## Neurologic exam: Key Points

- Either have to knock out bilateral cerebral hemispheres or reticular activating system (RAS) to result in neurologic causes of coma
- Neurologic deterioration is rostro-caudal





## Initial evaluation and management

Table 1 Coma checklist for the first hour

ABCs and C-spine Blood sugar Consider narcotic overdose Chemistries, ABG, urine toxicology screen Head CT if presumed structural etiology or uncertain

Reverse reversible causes early:

- Hypoglycemia Administer thiamine/ glucose
- Overdoses Antidotes ie. opiods-Narcan
- Consider empiric CNS antibiotics







## Additional management principles

- If diagnosis uncertain or there are focal signs on exam, obtain HCT +/- CTA (if basilar artery thrombosis is on differential)
- Treat underlying cause (seizures, infections, metabolic disease)
- Adjust body temperature
- If there is a concern for raised intracranial pressure (ICP) – raise HOB>30, administer hyperosmolar therapy, contact Neurology/Neurosurgery immediately



## **Differential diagnosis**

#### Table 26.1 Common causes of coma

Structural causes	Functional and other non- structural causes	
Compressive Bilateral epidural or subdural empyema, haematomas Cerebellum abscess, haemorrhage, tumour Pituitary tumour Pontine haemorrhage Subarachnoid haemorrhage Thalamus haemorrhage Uncal herniation	Toxins and drugs Alcohol Amphetamines Anticholinergics Barbiturates Benzodiazepines Ethylene glycol Lithium LSD Monoamine oxidase inhibitors Opiates Tricyclic antidepressants	
Destructive Acute anoxic injury Basilar occlusion Bilateral carotid occlusion Brainstem haemorrhage Central pontine myelinolysis Concussion and traumatic brain injuries Midbrain infarct Thalamus infarct Venous sinus thrombosis	Metabolic Acidosis Addison's disease Alkalosis Cushing's disease Hepatic encephalopathy Hypercapnia Hypertensive encephalopathy and posterior reversible leucoencephalopathy syndrome Hypoglycaemia Hypothermia Hypothermia Hypothyroidism Ionic disturbances (Na <sup>+</sup> , Ca <sup>2+</sup> , Mg <sup>2+</sup> , hyper- and hypo-, PO <sub>4</sub> <sup>3-</sup> , hypo-) Sepsis Severe hyperglycaemia, with or	

without ketoacidosis Severe hyperthermia Uraemic encephalopathy Wernicke's encephalopathy (thiamine deficiency)

- Establish likely diagnosis and treat presumptively -Diagnosis and treatment should be occurring simultaneously
- Use neurologic exam to localize lesion

Infections Acute disseminated encephalomyelitis Bacterial meningitis Viral encephalitis Oxygen-related Carbon monoxide intoxication Cyanide intoxication Diffuse ischaemia Disseminated intravascular coagulation Hyperviscosity Hypoxia

#### Other

Adrenoleucodystrophy Creutzfeldt-Jakob disease Non convulsive status epilepticus Postictal seizure Vasculitis

## Causes of coma

Is the cause

- Supratentorial structural disease
- Infratentorial structural disease
- **Diffuse brain disease** (metabolic)?
- Psychogenic? (Lids close actively, motor tone inconsistent/normal, normal reflexes, normal EEG)

### Metabolic causes

- Non-convulsive seizures
- Poisoning
- Hypoxia
- Hypoglycemia
- Diffuse Ischemia
- Acid-Base, Electrolyte disorders
- Primary Organ Failure
  - Liver, kidney, lung



## **Clinical Scenario #1**

- 52 year old woman brought to ED by EMS after being found down on a quiet street. Her last known well based on a 7-Eleven receipt in her purse was 35 minutes ago
- Vitals: Afebrile, HR-145 bpm, BP 100/60 mmHg, RR 12/min, SpO2 98% on RA.
- No visible signs of trauma
- Neurologic assessment GCS 7 (E1V1M4), Eyes-PERRL 7mm, corneal+ oculocephalics+. Motornormal tone, withdraws all extremities symmetrically. Respiration-Cheyne-stokes breathing pattern



## Initial steps in management?

- ABCs Patient is intubated for airway protection, IV access obtained peripherally successfully, C-collar placed as a precaution
- Basic labs including serum chemistry, LFTs, NH<sub>3</sub>, toxicology, ABG sent
- Fingerstick glucose 120, thiamine and naloxone administered without any change in mentation
- Husband contacted for brief history stated her only medical history is depression but he does not know his medications off the top of his head



# Other management considerations

- Focal neurologic exam neuroimaging
- Concern for seizure EEG
- Concern for CNS infection LP after neuroimaging



### What do we think could be a likely cause?



 Given a non-focal examination (motor withdrawal, normal cranial nerves), the suspicion for a supratentorial/infratentorial primary neurologic cause is low but because the cause remains unclear, a HCT/CTA are done



## Neuroimaging was normal. What are other possible causes?

- Non-structural neurologic (seizures)?
- Metabolic?
- Psychogenic?





# Since diagnosis is still unclear...

- EEG is done, LP is performed and CSF sent to lab
- EEG shows diffuse slowing but no epileptiform discharges





## **Case Conclusions**

- BMP/ABG show severe metabolic acidosis
- EKG shows a widened QRS and prolonged QTc
- You managed to reach the patient's pharmacy, who confirms patient has been taking TCA...
- You suspect TCA toxicity



 Within 48 hours, the patient wakes up and gets extubated





## Clinical Scenario #2

- 78 year old man brought to ED by EMS after being found down at home by her son. He last spoke to her 5 hours prior over the phone, when she seemed normal.
- Vitals: Afebrile, HR-98 bpm, BP 188/52 mmHg, RR 12/min, SpO2 98% on RA.
- No visible signs of trauma
- Neurologic assessment GCS 7 (E1V1M4). Eyes closed, does not arouse to pain. PERRL 1.5mm, R corneal+ but left absent, L gaze preference, does not cross midline on oculocephalics testing. Motor-Decreased tone on right, RUE and RLE 1/5 weakness, withdraws LUE And LLE to pain. Respiration-Apneustic breathing pattern



## First steps in management

- ABCs Patient is intubated for airway protection, IV access obtained peripherally successfully, C-collar placed as a precaution
- Basic labs including serum chemistry, LFTs, NH<sub>3</sub>, toxicology, ABG sent
- FSG 145
- Son not reachable over the phone for additional history
- Where do you think the problem localizes to?



### Is the cause

- Supratentorial structural disease
- Infratentorial structural disease
- Diffuse brain disease (metabolic)?
- Psychogenic?



- Focal findings on exam suggest this is perhaps structural disease, and presence of unresponsiveness, cranial nerve and motor findings suggest this perhaps localizes to the brainstem
- What do you do next?





- You decide to obtain a HCT/CTA given focal findings and neurologic examination and suspicion for brainstem structural disease such as infarct or hemorrhage
- Non-contrast HCT is unremarkable
- CTA head shows a mid-basilar occlusion
- Neurology (Stroke) and Neurointerventional teams are consulted and patient undergoes emergent mechanical thrombectomy







- He undergoes a successful mechanical thrombectomy
- He undergoes a 1 week ICU stay and a prolonged course at a rehabilitation facility, and comes to the Neurology Stroke clinic for a follow up 6 months later
- He walks without assistance and his only deficits are decreased abduction of L eye and subtle R hemiparesis (4+/5)







## **Clinical Scenario#3**

- 21 year old woman brought to ED by EMS after being found down in her bathtub by her mother, with a used needle next to her. She was last known well 30 minutes prior when she came downstairs for breakfast. Her mother commented patient seemed withdrawn.
- Vitals: Afebrile, HR-68 bpm, BP 108/52 mmHg, RR 8/min, SpO2 98% on RA.
- No visible signs of trauma
- Neurologic assessment GCS 3 (E1V1M1). Eyes closed, does not arouse to pain. PERRL 1mm, sluggishly reactive. Oculocephalics, corneals, cough, gag reflexes present. Motor-Decreased tone, no motor responses to pain.
- Respiration-Cheyne-Stokes breathing pattern
- How would you define her clinical condition? What do you think her most likely cause of coma is? What would be first steps in management?



### How would you define her clinical condition?

• She appears to be in a coma, as she is not awake and not aware of her environment.

### What do you think her most likely cause of coma is?

- Use clinical history and examination as a guide when considering differential diagnoses.
- Her most likely diagnosis is opioid overdose
- Supported by: clinical history, constricted pupils, depressed respiratory drive and otherwise non-focal neurologic examination
- Opioid overdose is a national epidemic

### What would be first steps in management?

- First steps would be ABC she likely needs to be intubated for airway protection
- Administer antidote Naloxone (Narcan)



## **Clinical Scenario #4**

- 18 year old college student is brought to ED by his friend, who noticed that patient has been getting more and more somnolent in the past 2 hours. He also noticed he was disoriented. His last known well was the previous night when they had dinner together, approximately 8:30pm. They both live in the same dormitory.
- Vitals: Temperature-103F, HR-114 bpm, BP 82/52 mmHg, RR 12/min, SpO2 97% on RA.
- No visible signs of trauma. Diffuse petechial skin rash most prominent in abdomen and trunk
- Neurologic assessment GCS 12 (E3V4M5). Drowsy, arouses to voice, but unable to follow simple commands. Oriented to self only but not place or time and at times speaks inappropriate words. PERRL 5mm, EOMI, no facial asymmetry or dysarthria. Motor-normal tone, localizes all extremities symmetrically. Respiration pattern – normal.
- How would you define his clinical condition? What do you think his most likely cause of coma is? What would be first steps in management?



### How would you define his clinical condition?

• He appears to be lethargic but clearly demonstrates wakefulness and responsiveness to external stimuli

### What is the most likely cause of coma?

- Use clinical history and examination as a guide when considering differential diagnoses
- His most likely diagnosis is meningitis, supported by: clinical history, fever, non-focal neurologic exam

### What would be first steps in management?

 First steps would be ABC – he may need volume resuscitation and/or vasopressor support; ensure IV access



**3L of 0.9% normal saline has been administered and his repeat VS are as follows:** Temperature-102.8F, HR-122 bpm, BP 78/51 mmHg, RR 12/min, SpO2 97% on RA. The decision has been made to proceed with vasopressor therapy. While the nurse is hanging the bag, she notices worsening of the patient's neurologic examination.

**His examination is as follows:** GCS 8 (E2V2M4). Drowsy, arouses to pain only, does not follow simple commands. Makes only incomprehensible sounds. PERRL 5mm, EOMI, no facial asymmetry, corneals, cough, gag reflexes all present. Motor-decreased tone, withdraws all extremities symmetrically. Respiration pattern – Cheyne-Strokes.

Would you characterize the patient as being in a coma? What are immediate next steps in diagnosis and management?



## Would you characterize the patient as being in a coma?

 Patient has decreased wakefulness (stuporous) but is not in a coma as he still demonstrates reactivity to external stimuli (opens eyes to pain)

## What are immediate next steps in diagnosis and management?

- With his neurologic deterioration, it would be prudent to revisit his ABCs
- Decision made to intubate the patient for airway protection, he is continued on vasopressor therapy

### His repeat VS are: Temperature-103.2F, HR-102 bpm, BP 100/78 mmHg, RR 12/min, SpO2 97% on RA. Now what is the best next step?



- Start empiric CNS antibiotics
- Obtain a HCT as his etiology is still uncertain and he is worsening

HCT was unremarkable. Now what?

- Perform LP
- Consider EEG



His opening pressure was 32mmHg. Preliminary gram stain grows gramnegative diplococci concerning for Neisseria Meningitis. He is admitted to the Medical ICU for further management





A 42 year old man was involved in a MVA 2 months ago where he sustained severe TBI. On examination now, his eyes are open spontaneously. He does not track or blink to threat. He does not follow commands and remains mute. His motor exam is remarkable for left hemiparesis with contractures of his arm and leg. How you best characterize his level of consciousness?

- A. Coma
- B. Vegetative State
- C. Obtundation
- D. Persistent Vegetative State
- E. Minimally conscious state



- A. Coma
- B. Vegetative State
- C. Obtundation
- D. Persistent Vegetative State
- E. Minimally conscious state

The patient appears to have wakefulness evidenced by his spontaneously open eyes. However, he does not show any awareness to external stimuli. This is consistent with vegetative state. He has not been in this state for longer than 3 months and it is uncertain if he may still improve, thus he is not in a persistent vegetative state at this time. His wakefulness makes coma and obtundation incorrect choices. His lack of awareness makes minimally conscious state an incorrect choice.



A 68 year old man is brought to the ED after being found down at home in feces and urine, foaming at his mouth. His vital signs were remarkable for BP 200/132, HR 112bpm, RR 22/min, SaO2-88% on RA. On exam, his eyes are closed and he does not arouse to painful stimuli. His pupils are 3mm and ractive, he has a left gaze preference. He is flaccid and has a strength of 0/5 in the LUE and LLE. On the right, he appears to have a weak withdrawal to painful stimulus. What is the next best step?

- A. Check stat labs including ABG, LFTs, serum chemistry
- B. Obtain an emergent head CT
- C. Lower his blood pressure
- D. Consult Neurology/ Neurosurgery
- E. Intubate for airway protection



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The first steps in early management is ensuring airway, breathing and circulation. This patient likely has impending respiratory failure given his altered mentation. The first best step is intubating him for airway protection. While laboratory tests and neuro-imaging can be very helpful and aid in diagnosis, ABCs take precedence over any further diagnostic evaluation. Consultations should be deferred until after the patient is stabilized.



## THE END



Lastly, the best kind of coma to be in – CARB coma

## **QUESTIONS????**

