



Chapter 13 – Depressed Consciousness and Coma

NOTE: CONTENT CONTAINED IN THIS DOCUMENT IS TAKEN FROM ROSEN'S EMERGENCY MEDICINE 9th Ed.

Italicized text is quoted directly from Rosen's.

Key Concepts:

1. *Consciousness consists of arousal (subcortical) and awareness (cortical).*
2. *Damage to the dorsal brainstem, thalamus, or axonal projections to the cortex, or extensive injury to bilateral cortices, may result in depressed consciousness or coma.*
3. *Toxic, metabolic, and infectious causes of coma make up 65% of cases; of these, toxins are the most common. Structural brain diseases make up most of the remaining 35% of cases.*
4. *An abrupt onset of coma suggests stroke, seizure, cardiac event, or poisoning.*
5. *A patient with depressed consciousness is unlikely to provide reliable history. Historical information should be elicited from other available sources, such as EMS and family.*
6. *The neurologic examination includes evaluation of the level of consciousness, cranial nerves, brainstem reflexes, and motor responses.*
7. *Pinpoint pupils may represent a pontine infarct or intoxication from opioids, clonidine, or cholinergic medications.*
8. *Hypoglycemia and hypoxia are two easily identifiable and reversible causes of coma.*
9. *An empirical trial of naloxone will lead to rapid reversal of opioid toxicity and other medication overdoses.*
10. *Non-convulsive status epilepticus should be suspected in cases of coma of undetermined causes and is diagnosed by EEG.*
11. *Most patients with coma will require intensive care. Transfer patients if the cause of coma is not treatable in the current facility (e.g., structural lesion requiring neurosurgery).*

Core Questions:

1. Define coma and differentiate coma from lethargy and stupor.
2. Name five neuroanatomic structures involved in maintaining arousal.
3. List five critical and five emergent causes of depressed consciousness. (see Table 13.1)
4. Describe your approach to the history and physical examination for the patient with depressed consciousness.
5. Outline your exam to accurately assess the Glasgow Coma Scale (GCS). (see Table 13.2)
6. What is the FOUR score, and how is it calculated? (see Table 13.3)
7. What ancillary tests should be ordered in the patient with depressed consciousness?



8. Outline your plan of management for the patient with depressed consciousness. (see Figure 13.2)

Wisecracks:

1. What is the best noxious stimulus to apply to evaluate GCS?
2. What are the oculocephalic and oculovestibular reflexes, and what information do they provide?
3. Describe decorticate and decerebrate posturing.
4. What is the utility of serum ammonia testing?

Rosen's in Perspective

We are back at it again and you all know what that means: we are jumping into the episode with a case:

You are working the afternoon shift at a community hospital when you get paged by the charge nurse to come to the triage desk. There is an 84-year-old female with a PMHx of mild cognitive impairment, hypertension, and hypercholesterolemia that is being brought into the ED by EMS. She was found by her daughter and was noted to be more somnolent than usual. En route, BP 95/85, HR 110, Temp 35.9 degrees Celsius, RR 25, SpO2 93% RA. Her GCS is 13-14. Yesterday, she was her normal self with no specific complaints.

You look down at your shoes, take a breath, and get ready. You think about all of the things that could cause this presentation. You reach a differential list of 4000 diagnoses and your mind breaks a bit. You begin to get a little confused yourself and start to feel a bit panicked. What are you going to do?

Answer: Don't sweat it. As always we got your back.

This post will have everything you need to quell your anxiety. We will give you the tools to be able to organize that mammoth list into a concise table in your mind. Additionally, we will give you a good run down of the relevant elements on history and physical exam to elicit in your next altered patient. These are patients you will be seeing A LOT of in the ED, so having a solid approach will help you immeasurably. Most of the people will have a toxic ingestion or metabolic derangement as the cause of their depressed LOC, but structural brain lesions, infections, and other things can cause your patients to be a little stuporous.

This podcast will be a keeper, but as we always say, read the chapter. Use our resources to supplement your learning, because as good as we think we are, we know that repeated exposure to core content in Emergency Medicine is key.

With that said, grab your coffee, settle in, and take a listen.



Core Questions:

[1] Define coma and differentiate coma from lethargy and stupor.

Coma is defined by Rosen's Emergency Medicine (9th Edition) to be the following:

- "A state of profoundly decreased arousal, resembling sleep"
 - These patients, by definition, will not be able to be aroused by external stimuli
 - It is important to note that these patients can have variable reflex behaviours present and still be comatose
- When you consider lethargy and stupor, these people will have diminished level of consciousness, but they are aroused by external stimuli

[2] Name five neuroanatomic structures involved in maintaining arousal.

Tight list for you guys. Here we go:

1. Brainstem nuclei
2. Hypothalamus
3. Thalamus
4. Ascending Reticular Activating System (ARAS)
5. Basal forebrain

The ARAS is the most important structure to consider. The neurons are located in the pons and midbrain. Their projections extend through the thalamus and into the cortices

Aside from equipping you for your next Neuro pimp session, knowing this content will allow you to help localize potential lesions causing depressed LOC. Any insult that affects the aforementioned structures or the cortices bilaterally will produce coma, so look for other signs on physical exam to either confirm or refute your clinical suspicions of lesion in this area.



[3] List five critical and five emergent causes of depressed consciousness. (see Table 13.1)

Diagnosis	Cause	Findings	Treatment	Comments
Metabolic				
Critical Diagnoses	Hypoglycemia	Diaphoresis, insulin pump	D50W, 50 ml	
	Hyperglycemia (DKA/HHNKS)	Tachypnea, N+V, abdo pain, dehydration	Isotonic fluids, insulin	
	Beriberi	Hypothermia, hypotension	Thiamine 100 mg IV	
	Adrenal Crisis	Weakness, weight loss, hypotension, hyperpigmentation	D5NS, correct low gluc, hydrocortisone 100 mg IV	Look for hyperK+
	Pituitary Apoplexy	HA, visual impairment, multiple hormone dysfunction	Treat electrolyte abnormalities, IV hydrocort 100 mg IV	May have pit adenoma; consult NeuroSx
	Sepsis	SIRS criteria, poor end-organ perfusion, delirium	ABX + isotonic fluids	
Emergent Diagnoses	Wernicke's Encephalopathy	CN III or VI palsies, sluggish pupils, anisocoria, gait instability, peripheral neuropathy	Thiamine replacement	Alcoholics or malnourish; rare in Hyperemesis Gravidarum
	Hyponatremia	Progressive confusion, HA, anorexia, seizure	Fluid restriction, hypertonic saline if seizing	Think med ADE
	Hyperammonemia	Lethargy, irritability, vomiting, seizures, poor feeding	Monitor protein intake, dialysis	Seen in liver disease, IEM's, ADE valproic acid, complication of bariatric Sx



	Hypercalcemia	Lethargy, polyuria, AKI, constipation	Isotonic fluids	Causes nephrogenic DI, suspect malignancy
	Uremia	N+V, anorexia, fatigue, ammonia breath	Treat hyperkalemia, dialysis	Check EKG for hyperK changes
	Hepatic Encephalopathy	Fetor hepaticus, asterix, ascites, stigmata of cirrhosis	Lactulose	Rule out sepsis, GIB, SBP
	Thyrotoxicosis	Fever, tachycardia, sweating, diarrhea	Isotonic fluids, propranolol 1 mg IV, PTU 600 mg PO	May need to treat adrenal insuff
	Myxedema Coma	Sluggish, weight gain, edema, depression, hair loss, constipation	Thyroxine 500 ug IV; hydrocortisone 100 mg IV	Search for acute precipitant
	Heat Stroke	Hyperpyrexia (<41.1 Celsius), flushing, exertion in heat, dehydration	Isotonic fluid, evaporative cooling	Look at elderly unable to seek cool environment
	HACE	Rapid ascent, HA, confusion, psychosis	Rapid descent, hyperbaric O2, dexamethasone 10 mg IV	More common above 3500 m
Toxic				
Critical Diagnoses	Hypoglycemic Agents	Older adult with poor renal function, intentional OD	D50W; octreotide 50-100 ug IV q 8h if refractory	Frequent OPCK in peds; admit all
	Opioids	Stupor, apnea, miosis, needle tracks	Naloxone 0.4 mg IV, up to 10 mg IV	Check skin for fent patches
	Simple Asphyxiants	Sudden lightheadedness, collapse, syncope	100% O2	Leaking CO2 tank in enclosed space; also



				helium, nitrogen, argon gas
	CO	Combustion of fuel in enclosed space, HA, confusion, malaise, nausea	100% O ₂ , hyperbaric O ₂ per toxicology	Look for others involved; think of pregnant patients carefully
	Histotoxic Hypoxia	Confusion, seizure, H ₂ S with rotten egg smell, cyanide with bitter almond scent; may result from products of combustion	100% oxygen; hydroxocobalam 70 mg/kg (or 5 g) for CN	Consider CN in any house or car fire
	MethHgB	Use of meds, cyanosis, pulse ox 85%	100% O ₂ , methylene blue 1-2 mg/kg IV	Can be caused by diarrhea in children
Emergent Diagnoses	Sedatives	Alcohol, benzos, may others	Supportive	Avoid flumazenil
	Toxic OH's	N+V, early osmolar gap then AGMA, renal failure	Fomepizole; 15 mg/kg IV load; correct electrolytes, isotonic fluid at 500 cc/hr	Consult Nephro and Tox for hemodialysis
	Inhalants	Young, paint on hands/face, diplopia, slurred speech, arrhythmias	Check EKG; definitive airway if lip or tongue edema	Consider frostbite causing airway edema
	Psychiatric Meds	Hypotension, prolonged QRS	NaHCO ₃ for TCA's	
	Anticonvulsants	Confusion, slurred speech, elevated levels	Supportive measures	Hyperammo. with Valproate
	Anticholinergics	Hyperpyrexia, pupil dilation,	Pyridostigmine rarely used;	



		urinary retention, visual hallucinations	benzos for severe agitation	
	Clonidine	Bradycardia, hypotension, somnolence	Naloxone up to 10 mg IV, then infusion 2-4 mg/h	
	Beta Blockers	Bradycardia, hypotension, hypoglycemia, seizure	Glucagon 5 mg IV, epi 1-4 ug per min; atropine 0.5 mg IV; pacing	Discuss IV lipid infusion with Tox
	Salicylates	N+V, tinnitus, delirium, hyperpnea, AGMA + mixed resp alk	D5W with 150 mEq/L NaHCO ₃ ; correct hypoK+, consider hemodialysis	Consider oil of wintergreen
	NMS	Hyperpyrexia, rigidity, delirium, autonomic instability	Cooling, isotonic fluids, benzos	May need to paralyze with ND agent
	Serotonin Syndrome	Multiple agents, hypertension, tachy, hyperreflexia, rigidity, tremor, nausea, diarrhea	Isotonic fluids, check CK, cyproheptadine 12 mg PO, benzos	May need to paralyze with ND agent
Structural				
Critical Diagnoses	Hemorrhage	HA, HTN, sudden onset, neuro deficits	CT w/o contrast, reversal of anticoagulation	Consult NeuroSx early
	Cortical Infarct	Sudden neuro deficits (unilateral)	CT/CTA, Neuro consult	TPA, if no CI's: 0.8 mg/kg IV, max 90 mg dose; administer 10% over 1 min, then rest over 60 min



	Cerebellar Infarct	Sudden vertigo, nausea, ataxia, dysarthria	Consider TPA	NeuroSx
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[4] Describe your approach to the history and physical examination for the patient with depressed consciousness.

History:

Point One: As you most you would likely assume, getting a history from an obtunded patient is not necessarily easy. Additionally, salient points on history from these patients may also be unreliable. Thus, it is important to stress that the VAST MAJORITY of the history in patients with altered LOC will come from collateral sources. Valuable sources for health information include the following:

- Family, friends, bystanders
- EMS (these people are trained to gather information at the scene; ask them what is what like during the extrication)
- First responders (e.g., police services)
- Electronic medical records

Point Two: It is important to contextualize the events leading up to the diminished LOC, the time period when the person became altered, and the patient's course after being discovered. Make sure to establish points of chronology:

- When were they last seen normal?
- How was their health prior to being altered?
- Had they any specific cardiovascular, respiratory, urinary, or neurologic complaints of note?
- How did they become altered? Was it a sudden event (more indicative of an acute cardiac, neurologic events like CVA or seizure, or toxic ingestion) or was it gradual (more indicative of a metabolic or infectious cause of their symptoms).

Point Three: Do a thorough medication review. These can often cause or contribute to the patient's altered state. Also, do not forget about OTC and supplements.

- You can consider contacting the patient's pharmacy to get more information about their medication history and compliance

Point Four: Look for any information that the patient may be carrying that will help you discern the cause of their cookiness. For example, many elderly persons carry cue cards with relevant PMHx and medication information on them. Medical alert bracelets and tattoos may also help.



Physical Exam:

These guys get a full head-to-toe examination. A wise practitioner of Emergency Medicine once said to undress all of your patients greater than 60 years old. Their AARP membership or Humpty's Emerald Club Card buys them a strip search.

Relevant structures to assess on physical exam include the following:

Vital Signs - don't forget a blood glucose

HEENT - look for signs of trauma, hemorrhage, skull fracture; also, look inside the mouth for evidence of tongue laceration to help discern if a seizure took place. Look for goiters and other stigmata of thyrotoxicosis/myxedema coma.

C-Spine - if there is any suspicion that this patient has a C-spine injury, immobilize and treat appropriately

CVS - full exam; listen for murmurs or extra heart sounds

RESP - listen for signs of focal consolidation or diffuse fluid volume overload

ABDO - look for stigmata of chronic liver disease, acute urinary retention, or peritonitis

DERM - look for signs of trauma, infection, and transdermal patches. Additionally, look for purpura or petechiae to support a diagnosis of meningococemia

NEURO - The breakdown of the NEURO exam is as follows:

1. GCS
2. Cranial Nerves
 - a. Pay attention to those pupils AND positioning of the eyes; dysconjugate gaze or pupillary abnormalities may indicate pathology intracranially
3. Motor Exam
 - a. Look at gross motor movements
 - b. Look for rigidity, spasticity, clonus
 - c. Evaluate reflexes
 - d. Evaluate posturing
4. Brainstem Reflexes
 - a. Oculocephalic Reflex
 - b. Oculovestibular Reflex
 - c. Corneal Reflex
 - d. Gag Reflex



[5] Outline your exam to accurately assess the Glasgow Coma Scale (GCS). (see Table 13.2)

This is an oldy but a goody. You have to know this stuff well, as knowledge of the GCS will most certainly influence management decisions. While not a perfect test, having a reliable exam is important. It will give you valuable information about disease progression and patient status going forward, and may give you a little more help with your differential and disposition plan.

Parameter	Rating	Score
Eye Opening		
Open before stimulus	Spontaneous	4
Open on spoken or shouted request	To voice	3
Open to fingertip stimulus	To pain	2
No opening at any time, no interfering factor	No response	1
Closed by local factor	Not testable (NT)	NT
Best Verbal Response		
Correctly gives name, place, and date	Oriented	5
Not oriented but communicates coherently	Confused	4
Intelligible single words	Inappropriate words	3
Only moans or groans	Incomprehensible sounds	2
No audible response, no interfering factor	No response	1
Factor interfering with communication	NT	NT
Best Motor Response		
Obeys two-step request	Obeys command	6
Moves hand across the body or above clavicle to stimulus	Localizes pain	5



Bends arm at elbow rapidly, features not predominantly abnormal	Withdraws from pain	4
Bends arm at elbow, features clearly predominantly abnormal	Flexion to pain	3
Extends arm at elbow	Extension to pain	2
No movement in arms or legs, no interfering factor	No response	1
Paralyzed or other limiting factor	NT	NT

[6] What is the FOUR score, and how is it calculated? (see Table 13.3)

The FOUR score, or the Full Outline of Unresponsiveness score is another tool we can use to assess the level of consciousness in a patient. It has been shown to have a higher interrater reliability than the GCS and has been validated in multiple clinical settings. This score integrates a more detailed assessment of brainstem reflexes. It is, albeit, not commonly used in many centers, but it is still a good tool - so remember it.

It is easy to remember: 4, 4, 4, 4

- Eye Response
 - 0 - closed to pain
 - 1 - open with pain
 - 2 - open to loud voice
 - 3 - open but not tracking
 - 4 - open or opened, tracking, or blinking to command
- Motor Response
 - 0 - no response to pain or generalized myoclonus status
 - 1 - extension to pain
 - 2 - flexion to pain
 - 3 - localizing pain
 - 4 - thumbs-up or peace sign
- Brainstem Reflexes
 - 0 - absent pupillary, corneal, and cough reflex
 - 1 - pupil AND corneal response absent
 - 2 - pupil OR corneal response absent
 - 3 - one pupil wide and fixed
 - 4 - pupil and corneal reflexes present



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

[7] What ancillary tests should be ordered in the patient with depressed consciousness?

This is a difficult question to answer, as you will select ancillary tests that are appropriate for your patient's presenting issues. With that being said, however, given that the differential is so broad for altered LOC, consider casting a wide net. Fixation is one of the leading causes of cognitive error in these scenarios, so do not focus on diagnosis too quickly.

According to Rosen's 9th Edition, consider the following diagnoses:

- Bedside glucose
- CBC
- Electrolytes, Extended Electrolytes
- VBG with Lactate
- Co-oximetry
- INR/aPTT
- Urinalysis
- Acetaminophen, ASA
- Antiepileptic drug serum levels (as appropriate)
- TSH
- Blood and urine cultures (if infection is suspected)
- CSF sampling (if appropriate)
- CT Scan (contrast versus non-contrast depending on clinical picture)
- CXR
- EKG

Other things you can consider are the following:

- Troponin/CK
- Serum Osm
- Serum EtOH (Rosen's states that this is rarely useful, however, we disagree)



[8] Outline your plan of management for the patient with depressed consciousness. (see Figure 13.2)

Rosen's 9th Edition actually has a decent algorithm that can help guide you through the initial management and work up for the patient with depressed consciousness. We have attempted to summarize it below, but check out the textbook for more details.

- Step One: Assess ABC's
- Step Two: measure glucose at bedside, obtain IV access, maintain SpO2 at approx 97%, initiate cardiac monitoring, obtain portable CXR, EKG, and labs
- Step Three: administer glucose if hypoglycemic, naloxone if opioid toxicity is suspected, and thiamine if malnourished
- Step Four: complete physical and neurologic examinations
 - If they improve with initial investigations and the clinical picture is clear, treat and observe
 - If initial management does not improve the patient and the work up at this point does not identify a cause, investigate for other causes
- Step Five: Determine if brainstem signs are present, secure airway, and administer ABx if infectious cause is suspected
 - If present - CT/CTA
 - If absent, plain CT
- Step Six: Determine if structural cause is present
 - If present, consult specialist, treat cause, and provide neuroprotective interventions. Also consider ICU admission.
 - If no structural cause is present, run through the DDX again
 - Toxins - labs, antidote/dialysis?
 - Seizure - EEG, anticonvulsant?
 - PRES - MRI, antihypertensive?
 - Infection - LP, antibiotics?
 - Nutrition - thiamine, glucose?
 - Endocrine - thyroid studies, steroids?

Wisecracks:

[1] What is the best noxious stimulus to apply to evaluate GCS?

Trick question, there are many. Another thing you should know is this: you should be applying two different types of painful stimuli depending on what you are trying to assess. While there is not a lot of high quality evidence surrounding this topic, Critical Care Services Ontario (an organization of hospital administrators and clinicians) recommends the following painful stimuli be used to adequately evaluate GCS:



When attempting to evaluate the patient's eye opening score on GCS, use a peripheral painful stimulus. Specifically, use interphalangeal pressure to determine this score. To do so, you should:

- Apply pressure with a pen/pencil to the lateral outer aspect of the proximal or distal interphalangeal joint (lateral aspect of the patient's finger or toe) for 10 to 15 seconds to elicit a response.
- Note: just because the patient flexes their arm does not mean the person has intact brain functioning; this is a spinal reflex

While attempting to elicit a motor response to assess the Motor score of the GCS, you should use a central stimulus. Specifically, you should use either the trapezius twist (CN XI), supraorbital pressure method (CN V), or jaw margin pressure method (CN V). To do this:

- Trap Twist - grab two inches of the muscle at the angle where the shoulder meets the neck with your thumb and first two fingers and twist for 10-20 seconds
 - Note: high SCI's may falsely obscure this finding
- Supraorbital Pressure Method - place the flat aspect of the thumb over the supraorbital ridge for 10-20 seconds, applying gradually increasing pressure
 - Note: do not use with suspected maxillofacial or head trauma
- Jaw Margin Pressure Method - apply the flat of your thumb at the maxillomandibular joint and apply gradually increasing pressure for 10-20 seconds
 - Note: use caution in patients with suspected elevated ICP, as this may compress the jugular vein and worsen pressures

This organization does not recommend using sternal rub as a noxious stimulus as the patient is often left with residual pain and injury afterwards. So, be kind to your next sleepy man when assessing GCS.

[2] What are the oculocephalic and oculovestibular reflexes, and what information do they provide?

We are going back to the days of medical school neurology lectures with this one. Strap in tight and enjoy the ride.

Oculocephalic Reflex

- Otherwise known as the Doll's Eye Reflex
- A normal oculocephalic reflex is seen when the patient's eyes move in the direction opposite the head when rotating the patient's head laterally. Normally, the eyes will appear to maintain visual fixation on an object
- Indicative of normal brainstem functioning
- Testing for the Doll's Eye reflex is not always possible given potential concern regarding cervical spine injury in altered trauma patients



Oculovestibular Reflex

- Otherwise known as the Cold Caloric Test
- More sensitive test for brainstem dysfunction
- With the patient's head of the bed elevated at 30 degrees, infusion of ice water into the external auditory canal produced sustained conjugate deviation toward the irrigated ear
- If the patient is conscious, the patient will also demonstrate nystagmus with the fast phase away from the irrigated ear
 - They will also likely have intense vertigo, nausea, and vomiting
- Again, an absent conjugate gaze likely indicates brainstem dysfunction; these people will keep their eyes in mid position with respect to the orbits and their eyes will move in unison with their head

[3] Describe decorticate and decerebrate posturing.

Pretty quick one here:

- Decorticate Posturing
 - Reflexive shoulder adduction, flexion of the elbows, wrists, and fingers
- Decerebrate Posturing
 - Reflexive shoulder adduction, elbow extension, and forearm pronation

Remember, this reflexive posturing is usually seen in patients with focal brain lesions; however, patients with systemic conditions (including toxic ingestions and metabolic derangements) can demonstrate these findings.

[4] What is the utility of serum ammonia testing?

This topic is controversial to say the least. While serum ammonia levels correlate well with the degree of hepatic encephalopathy (up to 2 x the ULN), there are some issues with them

1. You do not need elevated ammonia levels to make the diagnosis of HE - some patients can have normal ammonia levels and still have HE
2. Levels can change depending on a multitude of factors, including whether or not a tourniquet was used during the blood draw, whether or not the sample was placed on ice directly after being drawn, and whether or not the patient suffers from another condition that would increase their ammonia levels (e.g., Reye's Syndrome, cigarette smoking, shock, certain inborn errors of metabolism, GI bleeding)

UptoDate notes that they can be useful in monitoring the effects of ammonia-lowering agents, but does not find them useful when attempting to make the diagnosis of HE or when monitoring patients with chronic cirrhosis. Measuring serum ammonia levels may also be useful in states where acute hyperammonemia is caused by drug ingestion (e.g., valproic acid).



Thus the short answer is this: Their utility is in question, but serum ammonia levels may be useful in some patient