Psychiatric Presentations of Decreased Level of Consciousness

James Young, MD
Overview

- Introduction
- Approach to the patient
- Psychotic disorders
- Catatonia
- Mood disorders
- Somatoform Disorders
- Adverse Drug Reactions
Introduction
Introduction

• Decreased LOC not typical of a primary psychiatric disorder

• Consideration of medical causes is the first priority - especially with new onset symptoms in children and over 40
Levels of Consciousness

<table>
<thead>
<tr>
<th>Levels of consciousness</th>
<th>Definition</th>
</tr>
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<tbody>
<tr>
<td>Alert</td>
<td>An alert patient is fully conscious and aware of their environment</td>
</tr>
<tr>
<td>Drowsy or lethargic</td>
<td>A patient who is drowsy is awake, but is fatigued and may fall asleep if not stimulated</td>
</tr>
<tr>
<td>Obtunded</td>
<td>An obtunded patient has an even further reduced alertness along with decreased responsiveness and decreased interest in their environment</td>
</tr>
<tr>
<td>Stuporous</td>
<td>When a patient is stuporous, they are generally not responsive, except to vigorous stimulation</td>
</tr>
<tr>
<td>Comatose</td>
<td>A comatose patient is not arousable</td>
</tr>
</tbody>
</table>

*Data from Sadock BJ, Sadock VA. Kaplan & Sadock’s comprehensive textbook of psychiatry. 7th edition. Philadelphia: Lippincott Williams & Wilkins; 2000.*
Approach to the Patient
Interview

• Key elements
  • Introductions
  • Open ended questions
  • Specific questions about the chief complaint
  • Substance abuse
  • Trauma history
  • Social history
• Should be non-threatening and non-judgmental
• Interview should serve to establish positive patient-physician relationship, elicit information and allow observation of the patients behavior
Mental Status Exam

- Study by Reeves et al.: Chart review of 64 patients who were initially admitted to a psychiatric unit, then later identified as medical emergencies and transferred to a general medical service.

- Most important error identified was the failure to perform an adequate mental status exam.

- Most often missed diagnosis was delirium, which always requires a search for an underlying medical cause and is most readily detected by a mental status exam.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Elements of the mental status examination</th>
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<tbody>
<tr>
<td>Appearance</td>
<td>The overall appearance of the patient, including the nature and appropriateness of their attire and if they seem either sick versus healthy or calm versus distressed, and so forth</td>
</tr>
<tr>
<td>Alertness</td>
<td>The ability of the patient to maintain interest and attention to their environment (Table 2)</td>
</tr>
<tr>
<td>Orientation</td>
<td>A patient's awareness of self, place, and time</td>
</tr>
<tr>
<td>Attitude</td>
<td>The patient's general attitude toward answering questions, cooperation and seeking help</td>
</tr>
<tr>
<td>Mood</td>
<td>The patient's current pervasive and sustained emotional state</td>
</tr>
<tr>
<td>Affect</td>
<td>The present level of emotional responsiveness including the amount and the range of expressive behavior, it should be noted how congruent the affect is to the mood</td>
</tr>
<tr>
<td>Thought process</td>
<td>The patient's reasoning ability and soundness of their logic</td>
</tr>
<tr>
<td>Thought content</td>
<td>What the patient is thinking about, including suicidal, homicidal, or delusional ideation</td>
</tr>
<tr>
<td>Insight</td>
<td>A patient's ability to identify and understand their situation</td>
</tr>
<tr>
<td>Judgment</td>
<td>The ability to make good decisions that will maintain safety</td>
</tr>
<tr>
<td>Impulse control</td>
<td>The ability to delay gratification, follow a plan, and to think of consequences before acting</td>
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</table>

Physical Exam

• Head and neck for evidence of trauma

• Comprehensive neurological exam to assess for CNS pathology

• Abnormal vital signs

Laboratory Testing

• Policy guideline of the American College of Emergency Physicians: "in adult ED patients with primary psychiatric complaints, diagnostic evaluation should be directed by the history and physical examination."

• Patients with decreased LOC do not fit in this category
Psychotic Disorders
Schizophrenia

- Patients with psychotic disorders are usually alert and oriented
- Three realms of symptomatology:
  - **Positive**: Predominantly psychotic symptoms including hallucinations and delusions.
  - **Negative**: Affective flattening, alogia, avolition etc.
  - **Cognitive**: Impaired ability to prioritize, problem solve, organize etc.
- The appearance of diminished level of consciousness in schizophrenia is most likely a misinterpretation
  - The patient may be too psychotic to talk and be therefore functionally mute.
  - Also, those with extremes of negative and cognitive symptoms can appear to have an altered level of consciousness
Catatonia
Catatonia

• First described by Karl Ludwig Kahlbaum in 1874

• Throughout the 20th century there has been a tendency to associate catatonia with schizophrenia

• Artifact of early writings of Kraepelin and Bleuler and their conception of Dementia Precox

Karl Ludwig Kahlbaum
Catatonia

- Catatonia is most often associated with mood disorders - especially the manic and mixed episodes of bipolar disorder.

- Up to 20% of patients with mania exhibit catatonia compared with less than 5% in schizophrenia.

**Box 1: DSM-IV criteria for catatonia**

DSM-IV criteria for catatonia include a clinical picture that is dominated by at least 2 of the following:

1. *Motor immobility* as shown by catalepsy (including waxy flexibility) or stupor
2. *Excessive motor activity* (that is apparently purposeless and not influenced by external stimuli)
3. *Extreme negativism* (apparently motiveless resistance to all instructions or maintenance or present posture against attempts to be moved) or mutism
4. *Peculiarities of voluntary movement* as shown by posturing (voluntary assumption of inappropriate or bizarre postures) stereotyped movements, prominent mannerisms, or prominent grimacing
5. *Echophenomenon*: The automatic mimicking of the actions of another. Echolalia is the mimicry of speech and echopraxia is mimicry of movements.

Medical Causes of Catatonia

- **Endocrinopathies**: Hypoparathyroidism, thyrotoxicosis, pheochromocytoma
- **Neurologic**: frontotemporal lesions, stroke in anterior regions, TBI, epilepsy
- **Toxins**: salicylates, inhalation anesthesia, strychnine, fluoride, PCP
- **Infectious**: HIV, typhoid, tetanus
- **Medications**: antipsychotics, corticosteroids, ketamine, disulfiram
- **Withdrawal**: benzodiazepines, alcohol
Pathophysiology of Catatonia

- **Posterior Parietal Lobe**: Increased glutamate - anosognosia of position (posturing)
- **SMA**: bradykinesia and rigidity
- **Anterior cingulate & orbital frontal**: decreased GABA - diminished arousal, mutism, akinesia
- **Anterior hypothalamus**: malignant catatonia
Clinical entities similar to catatonia

- **Trauma**: may present with elective mutism
- **Parkinson Disease**: May present with mutism and rigidity
  - usually present with cogwheel rigidity and tremor
  - Early onset Parkinson Disease often develops more quickly with a predominance of akinesia, mutism and the absence of cogwheel rigidity and tremor.
- **OCD**: can present with repetitive and stereotypic behavior, especially in patients with mental retardation and autism
- **Locked-In Syndrome**: Bilateral Pontine lesions - consciousness preserved; patient immobile except for blinking and eye movements
Malignant Catatonia

- A severe and potentially life threatening form of catatonia
- Mortality rate estimated between 12% and 20%
- Most common cause is renal failure due to myoglobinuria
- Second most common cause is aspiration pneumonia due to decreased LOC
- Can occur as a complication of antipsychotic and other dopamine blocking medications. **NMS is a drug-induced malignant catatonia.**
Malignant Catatonia

- Risk factors:
  - Dementia
  - Underlying CNS pathology
  - Increased ambient heat
  - Dehydration

- Care should be taken that restrained patients are in well ventilated, climate controlled areas and are kept hydrated.
Malignant Catatonia

- Severe muscle rigidity
- Hyperthermia
- Autonomic Dysfunction
  - Tachycardia
  - Hypertension
  - Tachypnea
- Mental status changes - Can present with altered levels of consciousness ranging from lethargy to coma
- Presentation may include delirium
Malignant Catatonia

- Laboratory Changes
  - **Elevated Creatinine Kinase** - as high as 60,000 IU/L. Non-specific, can be elevated in trauma, IM injections, acute psychosis, exposure to neuroleptics and various other neuromuscular disorders.
  
  - **Leukocytosis** - 10,000 to 40,000 cells/mm³. Also non-specific, can be seen with infections, lithium therapy, stress, excitement and vigorous exercise.
  
  - **Decreased serum Iron levels** - Mechanism is unclear, but degree of decrease correlates with the severity of MC and levels improve as MC improves.
Malignant Catatonia

• MC is a potentially life threatening condition that warrants intensive and timely treatment.

• Treatment:
  • Discontinue antipsychotic and other agents with dopamine antagonism.
  • Benzodiazepines --> ECT (as often as 5 times per week)
  • If the patient is agitated or delirious, steps should be taken to assure the patient’s safety.
  • Hyperthermia should be treated with NSAIDS or acetaminophen. More aggressive measures such as cooling blankets or gastric lavage with ice water may need to be employed.
  • If dehydrated, give appropriate IV fluids
Malignant Catatonia

- Monitor:
  - **Blood pressure**: vasopressors
  - **Heart rate**: Beta blockers
  - **Oxygenation**: Supplemental oxygen
  - **Renal function** (via serum creatine kinase, creatinine and urea nitrogen): Dialysis

- Malignant catatonia presents similarly to an acute infectious process. Rule out with blood cultures, chest x-ray and CSF examination.
Neuroleptic Malignant Syndrome

- NMS is a medication induced MC caused by dopamine antagonist medications.

- Antipsychotic medications:
  - High potency > lower potency
  - Conventional > Atypical
  - More often after initiation or dose increase
  - Higher doses > Lower Doses (Not clearly dose dependent)
Neuroleptic Malignant Syndrome

- Can occur with other dopamine blocking agents such as metoclopramide

- NMS can also occur when dopamine agonists such as L-Dopa have been abruptly withdrawn or decreased.

- There is a likely genetic component to an individual’s susceptibility.
Mood Disorders
Mood Disorders

• Only in severe cases, would the patient’s level of consciousness be altered by the mood disorder itself.

• First rule out medical problems such as drug overdose, severe dehydration or malnutrition.

• Mood Disorder with Psychotic Features

  • Up to 15-20% of patients with major depressive disorder have psychotic features. This percentage can increase to as high as 45% in the elderly population.

  • As discussed the section on psychotic disorders, these symptoms can mimic a decreased level of consciousness. In rare cases, these patients may present as lethargic or obtunded.

  • **Cotard’s Syndrome**: (aka negation delusion or nihilistic delusion): person holds the belief that they do not exist or that they are dead, their organs are rotting away or that they are missing body parts.
Somatoform Disorders
Distinguishing the Groups

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Conscious Action</th>
<th>Conscious Gain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatoform Disorder</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Factitious Disorder</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Malingering</td>
<td>Yes</td>
<td>Yes</td>
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Psychogenic Non-Epileptic Seizures (PNES)
Psychogenic Non-Epileptic Seizures (PNES)

• “a psychogenic non-epileptic seizure is an observable abrupt paroxysmal change in behavior or consciousness that resembles an epileptic seizure, but that is not accompanied by the electrophysiological changes that accompany an epileptic seizure, for which no other evidence is found for other somatic causes for the seizure, whereas there is positive evidence or a strong suspicion for psychogenic factors that may have caused the seizure.”

• Psychogenic non-epileptic seizures (PNES) are a common manifestation of conversion disorder.

• Many would discourage the use of the term pseudoseizure because it implies “faking” or malingering.
Psychogenic Non-Epileptic Seizures (PNES)

- The incidence of PNES in the general population is about 1.5/100,000 persons per year
- This is about 4% of the incidence of epilepsy
- 25-30% of patients referred to tertiary epilepsy centers are eventually diagnosed with PNES
- It is important to make the correct diagnosis of PNES both to:
  - avoid potential iatrogenic hazards of anti-epileptic drugs and
  - decrease the delay of implementing appropriate psychiatric and psychological treatment
- Up to 50% of patients with PNES also have epilepsy and will require anti-epileptic treatment.
Psychogenic Non-Epileptic Seizures (PNES)

- PNES occurs due to a complex array of psychosocial and psychological mechanisms.
- Certain vulnerable individuals given certain circumstances will develop PNES.
  - **Predisposing Factors:**
    - PNES patients have a high incidence of trauma history and co-morbid PTSD
    - Patients with dependent, avoidant or borderline personality traits or disorders are more predisposed to psychosomatic symptoms
    - In addition, those with a combination of high trait anxiety and poor coping mechanisms are at increased risk
  - **Shaping Factor:** If a patient has a relative with epilepsy, or a prior history of epilepsy themselves, this may steer the psychosomatic symptoms in the direction of seizures.
  - **Triggering Factor:** Maladaptive defense and coping mechanisms are brought into play by a psychosocial stressor
  - **Prolongation Factor:** The problem may become chronic given the right prolongation factors, such as secondary gain
Characteristics of PNES

- Begin slowly
- Lack stereotyped progression of seizure activity
- Flailing, struggling type activity with asymmetric or out of phase clonic movements
- Pelvic thrusting
- Often last longer than 2 minutes
- Absence of tonic-phase, tongue biting, or incontinence
- May retain consciousness and attend to their environment while seizing.
- Rarely bite their tongues, fall, sustain injuries or lose bladder or bowel continence.
- No post-ictal symptoms
- Retrograde amnesia absent
Treatment of PNES

- Treatment begins when the patient is informed of the diagnosis.
- The way that a patient is informed can have long term consequences.
- They should not be abruptly told they do not have seizures and then dismissed.
- This condition is the unconscious generation of symptoms and patients view it as a significant problem regardless of its etiology.
- Patients with PNES are usually victims of abuse with maladaptive coping mechanisms.
- The suggestion that there is nothing medically wrong or to suggest they are “faking it” may traumatize them further.
- Patients should be informed in a respectful way that acknowledges the problem and steers them toward future treatment.
Treatment of PNES

- Effective treatment of PNES can be accomplished through a combination of psychotropic medications and psychotherapy

  - Medications
    - Should be considered to address co-morbid psychiatric symptoms such as depression and anxiety
    - Will itself not directly reduce PNES behavior.
    - Important to withdraw anti-epileptic drug treatment if appropriate
  
  - Psychotherapy
    - Focuses on abuse issues, emotional modulation and healthy coping strategies.
    - Some may also benefit from family therapy and case management.

- Outpatient treatment is likely more effective than hospitalization which may serve to reinforce somatoform behaviors
Psychogenic Coma
Psychogenic Coma

- A conversion disorder in which a patient presents in a comatose state with no apparent medical etiology
- Investigation into organic and life threatening etiologies of unresponsiveness need to be performed.
  - This includes a thorough physical exam, mental status exam, and collection of history from collateral sources
  - Emphasis should be placed on evaluating CNS pathology.
    - **Lumbar puncture** to culture and examine the CSF
    - **Neuroimaging** (preferably an MRI for increased resolution and ability visualize the posterior fossa and brainstem),
    - **EEG**: A “normal” EEG may suggest either a rare localized brainstem abnormality or a psychiatric cause of unresponsiveness.
- When a patient is unresponsive and no organic etiology is found after a thorough and prompt investigation of potentially life threatening etiologies, the diagnosis of psychogenic coma may be considered
- Conservative management and observation. Care should be taken to limit invasive testing and monitoring to avoid iatrogenic medical complications.
Psychogenic Coma

• There are some bedside tests to evaluate for psychogenic coma. However, the response to these maneuvers may be inconsistent and clinical judgment needs to be employed.

• **Noxious Stimuli:** Patients may respond to noxious stimuli such as smelling salts, sternal rub or cotton swabs placed in the nares.

• **Inconsistency:** Patients with psychogenic coma may hold their eyes tightly shut and resist attempts to open them.

• **Oculovestibular testing,** also called caloric testing, can also be used. In a physiologically awake person, irrigation of the ear with warm water produces nystagmus toward the irrigated side and irrigation with cold water produces similar nystagmus away from the irrigated side. Sustained nystagmus may indicate that the patient is unresponsive due to psychogenic causes.

• **Protective reflexes.** An example of this would be to hold the patients hand over their face and drop it. Patients with a psychogenic coma may slightly move their hand to the side as it falls so that it does not hit their face. The physician should catch the hand before it strikes the face to avoid facial injury.
The differential diagnosis of psychogenic coma

- Includes the broad differential for all causes of coma.
  - CNS events
  - Toxic ingestions
  - Endocrine dysfunction
  - Infectious disease
  - Respiratory abnormalities
  - Cardiovascular events
  - Hepatic dysfunction,
  - Renal dysfunction
  - Wernike’s encephalopathy
The differential diagnosis of psychogenic coma

- Neurologic and neuropsychiatric considerations in the differential diagnosis:
  - **Catatonia**: Maintaining postures for long periods of time, staring, negativism and mutism are common characteristics of catatonia that may mimic coma.
  - **Depressive Episode**: A severe depressive episode may present with profound inanition and abulia to the point of unresponsiveness.
  - **Locked-in syndrome**: Patients with bilateral pontine lesions that affect the pontine motor tracts presents as mute and paralyzed, but remain alert and has preserved intellectual functioning, upward gaze and eye blinking.
  - **Frontal Lobe Dysfunction**: Damage to the frontal lobes can result in symptoms of apathy and abulia, as well as impaired executive functioning. Frontal lobe dysfunction from congenital causes or head trauma may not be apparent on brain imaging.
The differential diagnosis of psychogenic coma

- **Akinetic mutism**: Caused by either a unilateral or bilateral lesion in the superior mesial region of the frontal lobe leaving patients unable to move or speak, except for eye tracking and movements to perform certain tasks such as eating.

- **Global aphasia**: Typically have an array of neurologic impairments due to a significant ischemic or hemorrhagic event; are rare cases where only language has been disrupted and all other cerebral functions remain intact.

- **Malingering**: The intentional generation of symptoms for a conscious secondary gain.
  - It is not a neuropsychiatric disorder
  - Does not preclude the presence of an actual physical disorder
  - Difficult to detect and clinicians have a tendency to over diagnose malingering
  - Often inconsistencies in presentation, collateral information and behavior
Adverse Drug Reactions
Adverse Drug Reactions

• There are several adverse drug reactions associated with medications that are commonly used to treat psychiatric disorders.

• Such reactions are not unique to psychiatry, and can occur with many different pharmacologic agents.

• It is important to understand what medications the patient is taking and adherence to the prescribed regime, as well as over the counter medications, herbal supplements, and drugs of abuse.

• It is also important to be mindful of how such agents can combine to cause problems.

• Here we will discuss:
  • acute dystonic reactions
  • serotonin syndrome
  • (NMS was discussed previously with Malignant Catatonia)
Acute Dystonic Reaction
Acute Dystonic Reaction

- **Acute Dystonic Reaction:** A sustained, involuntary and sometimes painful muscle contraction affecting either a single muscle or a group of muscles caused by a dopamine antagonist medication

- Cranial, pharyngeal, cervical and axial muscles are most commonly affected

- This can result in the patient suddenly assuming an abnormal posture or facial expression

- Common presentations are oculogyric crisis, grimacing, fixation of the jaw, retrocollis, torticollis, and opisthotonic posturing

- Such reactions are often terrifying to patients and may lead to poor adherence with medications in the future

- Laryngeal involvement may lead to respiratory difficulties, and in rare cases may require airway protection
Acute Dystonic Reaction

- The pathophysiology of ADRs are unknown.
- Since the symptoms can occur days after the initial blockade of dopamine receptors, likely secondary dopamine hypersensitivity
- At higher risk:
  - Young
  - Male
  - High muscle mass
  - African American
  - Hispanic
Acute Dystonic Reaction

- Any agent with dopamine antagonist activity can cause an ADR, but most are associated with antipsychotic medications.

- High potency antipsychotics such as haloperidol tend to cause more reactions than low potency antipsychotics such as chlorpromazine.

- Newer atypical antipsychotics (i.e. risperidone, quetiapine) are lower (but not without) risk

- The risk of an ADR is dose related.

- Up to 90% of ADRs occur within the first 4 days of neuroleptic exposure.

- If untreated, such symptoms can last for hours or days or longer with depot preparations
The cornerstone of treatment is with **anticholinergic medications**

- Prompt intramuscular (IM) or intravenous (IV) administration of anticholinergic medications usually results in rapid resolution of symptoms.

- **Benztropine** 1 to 2 mg is usually given either IM or IV. **Diphenhydramine** 25 mg can also be administered IM or IV.

- The anticholinergic medication given to reverse an ADR may wear off prior to the effects of the antipsychotic that caused the condition. Therefore, additional oral dosing of anticholinergic medications should be given for a few days, or longer.

- **Amantadine** 100 mg po BID may be an alternative in those in which anticholinergic medications are contraindicated.

- In high-risk patients, oral anticholinergic medications should be used prophylactically with the initiation of treatment of high potency antipsychotic medications. The typical regimen is **benztropine** 1 - 2 mg po BID
Serothotonin Syndrome
Serotonin Syndrome

- Serotonin syndrome is a reaction to medication that causes excessive serotonin agonism, both centrally and peripherally.
- The condition can progress to coma and death, and may require intubation, paralysis and sedation.

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Symptoms of serotonin syndrome</th>
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<tbody>
<tr>
<td>Neuromuscular hyperactivity</td>
<td>Tremor, clonus, myoclonus, hyperreflexia and, in advanced stages, pyramidal rigidity</td>
</tr>
<tr>
<td>Autonomic hyperactivity</td>
<td>Diaphoresis, fever, tachycardia, and tachypnea</td>
</tr>
<tr>
<td>Altered mental status</td>
<td>Agitation, excitement and, in advanced stages, confusion</td>
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Serotonin Syndrome
Treatment

• The main treatment for serotonin syndrome is withdrawing the offending agent.

• This will not usually result in an immediate resolution of symptoms, so supportive measures need to be provided.

• Benzodiazepines are helpful in decreasing agitation. Diazepam is the best studied, and has been shown in animal models to increase survival by blunting hyperadrenergic symptoms.

• In mild cases, IV hydration and observation are usually sufficient.

• In moderate cases 5-HT 2A antagonists, such as cyproheptadine, can be used.

• Severe cases with hyperthermia will often require intubation, paralysis and sedation as necessary.