Psychiatric Considerations in Patients with Decreased Levels of Consciousness

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KEYWORDS

- Altered level of consciousness
- Catatonia
- Malignant catatonia
- Neuroleptic malignant syndrome
- Serotonin syndrome
- Pseudo seizures
- Psychogenic nonepileptic seizures
- Psychogenic coma

In most cases, a decreased level of consciousness is not typical of a primary psychiatric illness. In any patient with a decreased level of consciousness, including those with current or a past history of psychiatric illness, consideration of medical causes, such as systemic illness, intoxication, acute drug reactions, and trauma is the first priority. In the absence of an underlying medical problem or intoxication, patients with psychiatric illness are usually fully alert.

This article discusses how patients with psychiatric illness may present with altered levels of consciousness. Ways in which severe cases of psychotic disorders, mood disorders, and somatoform disorders can present in this fashion are discussed. The often misunderstood syndrome of catatonia is also presented. Adverse drug reactions that can occur with medications used to treat psychiatric illness are also considered.

Evaluation of patients with psychiatric issues that present with decreased levels of consciousness are discussed first.

APPROACH TO THE PATIENT

Patient Interview

All patients suspected of having a psychiatric illness who present to the emergency department with altered levels of consciousness need to be thoroughly screened for medical causes, including an initial assessment to determine the acuity of their illness. Extra vigilance is warranted in the evaluation of children and patients older than 40 years.

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with no previous history of psychiatric illness who present with an altered level of consciousness.

Once stability is ensured, a thorough interview can be performed. The interview should be nonthreatening and nonjudgmental, and should contain key elements such as introductions, open-ended and specific questions about the chief complaint, past psychiatric history, substance abuse history, history of trauma, and a social history. The interview should serve to elicit information, establish a positive patient-physician relationship, and allow for observation of the patient’s behavior.\(^1\)

The patient’s past psychiatric history, including past history of suicide attempts, drug or alcohol use, hospitalizations, or other psychiatric treatments is important. It is necessary to note the patient’s past psychiatric evaluations and adherence to prescribed medications, psychiatric or otherwise. As discussed later in this article, reactions to medication, including psychotropic medications, can cause altered levels of consciousness.

In patients with a suspected psychiatric illness, a thorough social history is essential. It is vital to understand the patient’s living environment and how this environment may affect their current condition. Time should be spent reviewing any history of abuse or trauma. Any of these conditions can be exacerbated by recent difficult situations, so it is necessary to gather information about recent stressors and the environment in which patients are living.

In patients with severe psychiatric illness and an altered level of consciousness the interview can be limited. Contacting corroborating sources such as family members and medical records from previous providers or treatment facilities can provide useful diagnostic information.

**Mental Status Examination**

The mental status examination is an essential element in the evaluation of any patient, especially those with altered levels of consciousness. Mental status abnormalities can indicate serious underlying medical pathology. It is a snapshot of the patient’s current level of alertness, emotional state, the content of thoughts, and current cognitive functioning. It is important to be aware that the patient’s mental state can change, and they may require periodic re-evaluation.

The relevance of the mental status examination in making an accurate diagnosis is illustrated in a study done by Reeves and colleagues.\(^2\) In this study, a chart review was performed of 64 patients who were initially admitted to a psychiatric unit, later identified as medical emergencies and then transferred to a general medical service. The single most important error identified was failure to perform an adequate mental status examination. Other reasons cited were failure to perform an adequate physical examination, failure to obtain indicated laboratory and radiologic studies, and failure to obtain an adequate history. The most common missed diagnosis was delirium, which was most readily detected by an adequate mental status examination. Delirium always requires a search for an underlying medical cause, especially in a person with no psychiatric history.

The mental status examination is best done in a systematic fashion, and should be comprehensive. It is a wise investment of time. Information and observations made while conducting the mental status examination further guide the diagnostic hypotheses made through the rest of the evaluation.\(^3\)

Common elements of the mental status examination are listed in Table 1.\(^4\)

**Physical Examination**

A complete physical examination is essential to thoroughly assess for medical causes for altered levels of consciousness. Areas that may require focused attention are
examination of the head and neck for evidence of trauma, and a comprehensive neurologic examination to assess for central nervous system (CNS) pathology. In addition, vital signs are a critical component of the examination. Abnormal vital signs in a patient with diminished consciousness indicate an increased need to search for medical pathology. A diminished level of consciousness should also suggest the need for pulse oximetry and a finger stick glucose test in the vital sign assessment.

### Laboratory Testing

Laboratory testing required in the assessment of patients with psychiatric illness is somewhat controversial but ultimately depends on the clinical assessment. If the

**Table 1**

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<th>Elements of the mental status examination</th>
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<td>Appearance</td>
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<td>Orientation</td>
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**Table 2**

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<td>Alert</td>
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<td>Drowsy or lethargic</td>
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<td>Obtundated</td>
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<td>Stuporous</td>
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<td>Comatose</td>
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patient is admitted to a psychiatric facility, medical evaluation may be limited as psychiatric assessment and treatment become priorities. In a literature review and clinical policy guideline published by the American College of Emergency Physicians in 2006, the authors concluded “in adult ED patients with primary psychiatric complaints, diagnostic evaluation should be directed by the history and physical examination.” Routine preset laboratory testing is not recommended for all patients. Patients with diminished level of consciousness do not belong in this category, as diminished levels of consciousness may well be the presenting feature of a medical illness.

**PSYCHIATRIC PRESENTATIONS OF ALTERED LEVEL OF CONSCIOUSNESS**

Various psychiatric conditions and situations that may be at issue when a patient presents with altered levels of consciousness are now described, including psychotic disorders, mood disorders, somatoform disorders, and adverse drug reactions to psychiatric medications.

**Psychotic Disorders**

The *Diagnostic and Statistical Manual of Mental Disorders* (4th edition) (DSM-IV) lists several types of psychotic disorders that are differentiated by type of symptoms, severity of illness, and time course. Here the focus is on schizophrenia, but many of these concepts can be generalized to the other psychotic disorders.

Schizophrenia is unfortunately a chronic life-long condition. A prodromal phase including social isolation or eccentric behavior typically precedes the first psychotic break for several years, and is usually only recognized in retrospect. The first episode of psychosis usually occurs between late adolescence and the mid-thirties. A new onset psychotic symptom in general, especially when the patient is outside of this age range, warrants further investigation for a neurologic or medical illness.

Patients with schizophrenia have 3 primary types of symptoms: positive symptoms, negative symptoms, and cognitive symptoms. The positive symptoms are predominately psychotic symptoms, which include delusions and hallucinations. Delusions have a variety of themes and persecutory delusions are most common. Hallucinations are typically auditory in schizophrenia, whereas visual symptoms are more suggestive of medical illness. A presentation of psychotic symptoms may have the appearance of a decreased level of consciousness. For example, patients may be too paranoid to talk and thus seem functionally mute. Negative symptoms include affective flattening, alogia, and avolition. Alogia refers to diminished speech (in brief replies to questions, for instance) and apparent diminished thought processes. Avolition refers to diminished initiative or goal-directed activity. Cognitive symptoms manifest as decreased ability to problem solve and organize. Many patients with schizophrenia have difficulty with performing basic activities of daily living, such as budgeting, making and keeping appointments, and shopping for groceries. Patients with schizophrenia are usually alert and oriented. It is possible that those with extremes of the negative and cognitive symptoms can appear with an altered mental state.

**Catatonia**

The syndrome of catatonia was first described by Karl Ludwig Kahlbaum in 1874 and has been a misunderstood clinical entity. Throughout the twentieth century, there has been a tendency to associate catatonia with schizophrenia. This may be an artifact of the early writings of Emil Kraepelin and Eugen Bleuler and their conception of dementia precox. It has been clearly shown that 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\textsuperscript{6} compared with less than 5% in schizophrenia.\textsuperscript{7} DSM-IV criteria for catatonia are listed in Box 1.

Although usually a manifestation of a primary psychiatric condition, there are some medical conditions that can cause the syndrome of catatonia. The medical causes of catatonia are varied and similar to the causes of delirium. Endocrinopathies such as hypoparathyroidism with resulting hypocalcemia, thyrotoxicosis, and pheochromocytoma can present with catatonic features. Neurologic pathology such as frontotemporal lesions, strokes in anterior brain regions, traumatic brain injury, and epilepsy may also be associated with catatonia. Catatonia can also be caused by exposure to salicylates, inhalation anesthesia, strychnine, and fluoride. There are also some infectious causes such as human immunodeficiency virus, typhoid fever, tetanus, and staphylococcus.\textsuperscript{8} Some medications may induce or facilitate catatonia. These include antipsychotics, corticosteroids, ketamine, and disulfiram. Catatonia may also be caused by the withdrawal of benzodiazepines. In addition, patients abusing phencyclidine (PCP) can also become catatonic.\textsuperscript{9}

There have been many recent advances in the understanding of the pathophysiology of catatonia. The syndrome is likely caused by several different neurochemical abnormalities in different areas of the brain, including decreased \( \gamma \)-aminobutyric acid (GABA) and dopamine activity, and increased glutamate activity. Abnormal functioning of these neurochemical circuits in different regions of the brain results in manifestations of the different catatonic symptoms. Increased glutamate activity at \( N \)-methyl-\( D \)-aspartate (NMDA) receptors in the posterior parietal lobe results in anosognosia of position, seen as bizarre and mundane posturing behavior.\textsuperscript{10} Abnormal activity in the posterior parietal lobe may influence GABA and dopamine activity in areas such as the supplemental motor area and the medial orbital gyrus resulting in bradykinesia and rigidity. Involvement of the anterior cingulate-medial orbitofrontal circuit may result in diminished arousal, mutism, and akinesia.\textsuperscript{11} If the anterior hypothalamus is involved, malignant catatonia, a life-threatening type of catatonia can occur.\textsuperscript{9} Malignant catatonia is further explored in the next section.

**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.

There are several different clinical entities that may present with symptoms similar to catatonia. Some patients who have been recently traumatized or who suffer from a personality disorder may present with elective mutism. In this case, the symptom of mutism would occur in isolation, and should not be confused with catatonia. Patients with Parkinson disease may present with mutism and rigidity in the form of assuming peculiar and abnormal postures. This may pose a diagnostic dilemma, but patients with Parkinson disease usually present with cogwheel rigidity and tremor. This is especially difficult to distinguish in early onset Parkinson disease, which tends to develop more quickly with a predominance of akinesia, mutism, and often the absence of tremor and cogwheel rigidity. Relief of symptoms with an anticholinergic agent such as benztrapine may aid in the diagnosis. Obsessive compulsive disorder may present with repetitive and stereotypic behavior, as well as grimacing and tics that may bear a resemblance to catatonia. This is especially true in patients with autism and mental retardation. The locked-in syndrome that results from bilateral pontine lesions renders a patient immobile except for eye movements and blinking. Cortical functioning, including consciousness is preserved, demonstrated by voluntary eye blinking in response to questions.

The definitive treatment of catatonia is to treat the underlying cause. In all patients with catatonia, potential toxic precipitants should be eliminated and any precipitating general medical or neurologic conditions should be treated. Treatment of an underlying psychiatric disorder should also be initiated. As stated earlier, there is a tendency to associate catatonia with schizophrenia when in fact it most commonly occurs with mood disorders. The use of antipsychotic medications in a catatonic patient is generally discouraged because they may worsen the catatonic symptoms, and they also place the patient at higher risk for malignant catatonia or neuroleptic malignant syndrome (NMS). Patients with a retarded catatonia and a body temperature less than 39°C should be given lorazepam parenterally or orally at 3 mg per day, and the dose should be increased rapidly until resolution is achieved. Doses of up to 20 to 30 mg/d may be necessary. Remission of catatonia with lorazepam has been reported in 80% to 100% of patients. Electroconvulsive therapy (ECT) has been shown to be an effective treatment of catatonia. Bilateral ECT is more effective than unilateral ECT in patients who are febrile, delirious, do not respond to lorazepam, or are otherwise at physiologic risk. ECT can be done daily for 2 to 5 days and remission of catatonia has been reported from 82% to 96%.

**Malignant Catatonia and Neuroleptic Malignant Syndrome**

Malignant catatonia (MC) is a severe and potentially life-threatening form of catatonia. MC that develops as a complication of antipsychotic and other dopamine blocking medication administration is called NMS. NMS is considered to be a drug-induced MC. MC can present with altered levels of consciousness ranging from confusion to coma. Although it is difficult to predict who will develop MC, there are certain risk factors for the disease such as underlying CNS pathology and dementia. Increased ambient heat and dehydration may increase the risk. This is important to consider when treating a severely psychotic patient who exhibits poor judgment and self-care. Care should be taken that restrained patients are in well-ventilated climate-controlled areas and are adequately hydrated.

MC is most commonly recognized as severe muscle rigidity and hyperthermia. In addition, patients often present with autonomic dysfunction such as tachycardia, hypertension, and tachypnea, and mental status changes including delirium.
Serum creatine kinase (CK) levels may be as high as 60,000 IU/L and leukocytosis can range from 10,000 to 40,000 cells/mm³ with a left shift. An increased CK level is nonspecific and can also be seen in trauma, intramuscular injection, acute psychosis, exposure to neuroleptics, and various other neuromuscular disorders. The level of CK does not correlate with the degree or duration of muscle rigidity or increase in temperature. Evaluating and monitoring CK levels may help make the diagnosis of MC, monitor for improvement or relapse, and serve as a marker for risk of renal failure. Leukocytosis is also a nonspecific finding and may occur in several physiologic conditions such as infection, lithium therapy, stress, excitement, and vigorous exercise.

It has also been shown that serum iron levels are decreased in MC. The mechanism for this finding is not clear, but the severity of MC has been correlated to the degree of decrease in serum iron. Conversely, it has been shown that iron levels improve as MC symptoms improve.

MC can also present atypically. For example, MC can occur with no muscle rigidity or hyperthermia. This poses a true diagnostic dilemma because of the unpredictability of symptom onset related to starting or withdrawing neuroleptic medicines.

The mortality from MC is estimated to be 12% to 20%. The most common cause of death is renal failure secondary to myoglobinuria. The second most common cause of death is aspiration pneumonia caused by decreased levels of consciousness and dysphagia. Mortality can be caused by cardiovascular events such as myocardial infarction exacerbated by autonomic instability or fatal dysrhythmias exacerbated by electrolyte abnormalities. Deaths in patients with MC have also been attributed to thrombocytopenia and disseminated intravascular coagulation.

The pathophysiology of MC has not been firmly established, however it is likely a complex dysregulation of different systems resulting in a hypermetabolic state resulting from lowered dopaminergic activity.

NMS is a medication-induced MC caused by dopamine antagonist medications. In addition to antipsychotic medications, the syndrome can occur with other dopamine blocking agents such as metoclopramide and amoxapine. NMS has also been seen in patients with extrapyramidal disorders such as Parkinson disease, Wilson disease, Huntington chorea, and striatonigral degeneration. In such conditions dopamine agonists such as L-3,4-dihydroxyphenylalanine (L-Dopa) may have been abruptly withdrawn or decreased. Amantadine may be of some benefit in treating NMS because of its NMDA glutamate receptor antagonist properties.

The incidence of NMS may be as high as 1.0% of patients treated with neuroleptics. High-potency conventional antipsychotics such as haloperidol and thiothixene are believed to have a higher risk of NMS than lower-potency neuroleptics, such as chlorpromazine and mesoridizine. NMS has been reported with the newer atypical antipsychotic medications such as olanzapine; risperidone, and even clozaril. NMS commonly occurs soon after initiation, or a recent dose increase of a neuroleptic, but can occur at any time during the treatment course. The syndrome is not clearly dose dependent but more likely occurs in individuals taking high doses of neuroleptics and those who are neuroleptic naive. There is a likely genetic component to an individual’s susceptibility. Although extremely rare, NMS has been reported after only 1 dose.

Another condition that can be considered as a medication-induced MC is malignant hyperthermia. Malignant hyperthermia is a rare autosomal dominant disorder that renders susceptibility to inhalation anesthetics and succinylcholine. These patients show a hypermetabolic response to these agents resulting in muscle rigidity, increased creatinine kinase level, tremor, and fever. This condition has considerable symptom overlap with NMS and MC.
MC is a potentially life-threatening condition that warrants intensive and timely treatment. The first step is to discontinue all antipsychotic and other agents with dopamine antagonism. If the patient is agitated or delirious, steps should be taken to ensure the patient’s safety. Hyperthermia should be treated with nonsteroidal antiinflammatory drugs or acetaminophen. More aggressive measures such as cooling blankets or gastric lavage with ice water may need to be used. If dehydrated, patients should receive appropriate intravenous (IV) fluids. The patient’s blood pressure and heart rate should be monitored. β-Blockers and vasopressors may be given as necessary. Oxygen saturation must also be monitored with supplemental oxygen given as needed. Patients also require frequent monitoring of their renal function via serum CK, creatinine, and urea nitrogen levels. Dialysis may be required if the symptoms do not resolve quickly. MC often presents similarly to an acute infectious process. Moreover, infection should be ruled out with blood cultures, a chest radiograph, and cerebrospinal fluid (CSF) examination.

Mood Disorders

The mood disorders consist mainly of major depressive disorder and bipolar disorder. Because mania generally presents with a heightened level of consciousness, except when the patient is catatonic, it is not discussed here. A patient experiencing a depressive episode, caused by major depressive disorder or bipolar disorder, may in some extreme circumstances present with decreased levels of consciousness.

When a patient presents with an altered mental state caused by depression or severe psychotic depression, it is extremely important to first rule out medical problems such as drug overdose, severe dehydration, or malnutrition. Psychiatric consultants will typically recommend blood chemistry analysis (including electrolytes, calcium, magnesium, blood urea nitrogen, and creatinine), liver function studies, thyroid function studies, complete blood count, and a comprehensive urine drug screen.

A depressed episode is characterized by a syndrome of symptoms including depressed mood, alterations in sleeping and eating patterns, anhedonia, excessive guilt, poor concentration, psychomotor slowing, and suicidal thoughts. The symptomatology and level of dysfunction caused by a depressed episode can range from mild to severe. Altered levels of consciousness would be atypical and should alert the clinician to possible complications, such as a comorbid medical illness or drug toxicity/overdose. Only in severe cases is the patient’s level of consciousness altered by the mood disorder itself.

A patient experiencing a severe depressive episode can also present with psychotic features such as delusional thinking or auditory hallucinations. As discussed in 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. Mood congruent delusions include guilt, deserved punishment, sickness, or delusions of poverty. An example of a delusion that is congruent with the state of depression is Cotard syndrome. A person with Cotard syndrome (also known as negation delusion or nihilistic delusion) is when the person holds the belief that they do not exist or that they are dead. Variations on this theme are the belief that their organs are rotting away or that they are missing body parts. Auditory hallucinations tend to be transient and are typically insulting or demeaning. It is possible, although less common, that the hallucinations or delusions are not congruent with the patient’s mood. A lack of congruency with the patient’s mood indicates a poor prognosis.

Studies have shown that up to 15% to 20% of patients with major depressive disorder have psychotic features. This percentage can increase to as high as 45%
in the elderly population. Major depressive disorder with psychotic features is associated with poor short-term outcomes, longer recovery times, greater levels of disability, and higher mortality.\textsuperscript{19}

Other variations of a depressive episode include melancholic depression and atypical depression. Melancholic depression (also called endogenous depression) is characterized by severe anhedonia, early-morning waking, weight loss, and profound feelings of guilt over trivial events.\textsuperscript{19,20} Atypical depression is characterized by oversleeping and overeating. These patients typically have a younger age of onset, more severe psychomotor slowing, and higher rates of comorbid anxiety, substance abuse, and somatization disorder. Patients with atypical depression have an increased chance of a longer course of illness, a future diagnosis of bipolar disorder, and a seasonal pattern to their moods.\textsuperscript{19,20}

**Conversion Disorder**

Conversion disorder is a type of somatoform disorder in which a patient experiences medically unexplained neurologic symptoms such as hemianesthesia, blindness, involuntary movements, tics, or nonepileptic seizures. These symptoms are not consciously generated and there is an absence of conscious secondary gain. Conversion disorder typically occurs in a time of psychosocial stress. Van der Kolk and colleagues\textsuperscript{21} explain that posttraumatic stress disorder (PTSD), dissociation, somatization, and affect dysregulation represent a spectrum of adaptations to trauma. It is important to gather information about psychosocial stressors and trauma/abuse history when treating patients with conversion disorder. Patients with conversion disorder often present in the context of a stressful situation. Conversion disorder symptoms usually appear abruptly and disappear abruptly and it is rare, but not unheard of, for someone to have chronic conversion disorder.

Two variations of conversion disorders that may present with altered levels of consciousness are discussed: psychogenic nonepileptic seizures and psychogenic coma.

**Psychogenic Nonepileptic Seizures**

Psychogenic nonepileptic seizures (PNES), often called pseudo seizures, are a common manifestation of conversion disorder. Many would discourage the use of the term pseudo seizure because it implies faking or malingering. A PNES is an episode of altered behavior, motor activity, and perceptions that appear like an epileptic seizure but with an absence of neurologic evidence of seizure activity.\textsuperscript{22} Bodde and colleagues\textsuperscript{23} offer the following definition: “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 electrophysiologic 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.”

The incidence of PNES in the general population is about 1.5/100,000 persons per year which is about 4% of the incidence of epilepsy; 25% to 30% of patients referred to tertiary epilepsy centers are eventually diagnosed with pseudo seizure.\textsuperscript{23} It is important to make the correct diagnosis of PNES to avoid potential iatrogenic hazards of antiepileptic drugs and to decrease the delay in implementing appropriate psychiatric and psychological treatment.\textsuperscript{23}

PNES occurs as a result of a complex array of psychosocial and psychological mechanisms. It is believed that certain vulnerable individuals will develop PNES in certain circumstances. First, there is likely an initial psychological cause, such as
the experience of abuse or trauma. PNES patients have a high incidence of trauma history and comorbid PTSD. Some people are more predisposed to psychosomatic symptoms. 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. Patients with an abuse history and vulnerable personality traits who develop PNES often have a shaping factor, such as a relative with epilepsy, or a previous history of epilepsy themselves, that steer the symptoms in the direction of seizures. There is often a triggering factor in which the maladaptive defense and coping mechanisms are brought into play by a psychosocial stressor. The problem may become chronic given the right prolongation factors, such as secondary gain.

It is important to recognize nonepileptic seizures, as failure to do so may result in iatrogenic injury and death resulting from unnecessary treatments. However, it can be confusing as up to 50% of patients with PNES also have epilepsy and will require antiepileptic treatment.

Patients with PNES often do not show typical seizure activity. They can show motor phenomena uncharacteristic of epileptic seizures such as asynchronous out of phase clonic movements, opisthotonus, side-to-side head movements, atonia, eyes deviating upward, and resistance to eye opening. Patients can also show decreased responsiveness.

One way to understand the presentation of PNES is to compare it with the presentation of a true ictal event, such as a grand mal seizure. A grand mal seizure usually begins with an aura. Following this the patient abruptly loses consciousness and begins a tonic then clonic phase of movement. In a grand mal seizure these are bilaterally symmetric forceful movements. Depending on the position of the patient at the onset of the seizure, injuries can occur. Common injuries occur when the patient falls or bites their tongue. A grand mal seizure typically lasts 30 seconds and rarely more than 2 minutes. Following the seizure there is a 15 to 20 minute postictal period in which the patient is initially nonverbal and disoriented. During this time the patient gradually recovers consciousness and the ability to speak.

Pseudo seizures typically do not follow these expected patterns. A PNES resembling a grand mal seizure may begin gradually instead of abruptly and without a stereotyped progression of seizure activity. Instead of bilaterally symmetric forceful contractions, patients with PNES often have struggling or flailing type movements. Patients with PNES may retain consciousness and attend to their environment during seizure. Patients with PNES rarely bite their tongue, fall, sustain injuries, or lose bladder or bowel continence. It is also uncommon to have postictal symptoms with PNES.

There is a dearth of literature on the efficacy of treatment strategies for patients with PNES, and there are no standardized treatment protocols. However, there are some basic strategies that can be followed.

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 cause. 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.
Effective treatment of PNES is accomplished through a combination of psychotropic medications and psychotherapy. Medications should be considered to address comorbid psychiatric symptoms such as depression and anxiety but do not directly reduce PNES behavior. Patients also need psychotherapy that focuses on abuse issues, emotional modulation, and healthy coping strategies. Some may also benefit from family therapy and case management.

When a diagnosis of PNES is firmly established in the absence of a diagnosis of epilepsy, it is important to withdraw antiepileptic drug treatment. Outpatient treatment is likely more effective. Hospitalization may serve to reinforce somatoform behaviors and should be avoided when it is safe to do so.

**Psychogenic Coma**

A psychogenic coma is a conversion disorder in which a patient presents in a comatose state with no apparent medical cause. When a patient is unresponsive and no organic cause is found after a thorough and prompt investigation of potentially life-threatening causes, the diagnosis of psychogenic coma may be considered.27

First and foremost, investigation into organic and life-threatening causes of unresponsiveness need to be performed. This includes a thorough physical examination, mental status examination, and collection of history from collateral sources. Emphasis should be placed on evaluating CNS pathology. The workup includes a lumbar puncture to culture and examine the CSF, neuroimaging (preferably magnetic resonance imaging for increased resolution and ability to visualize the posterior fossa and brainstem), and an electroencephalogram (EEG). A normal EEG may suggest either a rare localized brainstem abnormality or a psychiatric cause of unresponsiveness.28

There are some bedside tests to evaluate for psychogenic coma. However, the response to these maneuvers may be inconsistent and clinical judgment is necessary. Patients may respond to noxious stimuli such as smelling salts, sternal rub, or cotton swabs placed in the nares. 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.27 The clinician may also try to elicit 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.27 The physician should catch the hand before it strikes the face to avoid facial injury.

Once the diagnosis of psychogenic coma has been established, conservative management and observation should be practiced. Care should be taken to limit invasive testing and monitoring to avoid iatrogenic medical complications.

The differential diagnosis of psychogenic coma includes the broad differential for all causes of coma. These include CNS events, toxic ingestion, endocrine dysfunction, infectious disease, respiratory abnormalities, cardiovascular events, hepatic dysfunction, renal dysfunction, and Wernike encephalopathy.28

There are some neurologic and neuropsychiatric considerations in the differential diagnosis as well. Patients with catatonia may present in an unresponsive state. Maintaining postures for long periods of time, staring, negativism, and mutism are common characteristics of catatonia that may mimic coma. A severe depressive episode may present with profound inanition and abulia to the point of unresponsiveness. Patients in this state may be uninterested in their environment and subsequently may stop
talking and eating. A patient with locked-in syndrome resulting from bilateral pontine lesions that affect the pontine motor tracts presents as mute and paralyzed, but appears alert and has preserved intellectual functioning, upward gaze, and eye blinking. 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. Patients with akinetic mutism are unable to move or speak, except for eye tracking and movements to perform certain tasks such as eating. These patients seem to be unconcerned with their lack of ability to communicate, unlike patients with aphasia. This condition is caused by a unilateral or bilateral lesion in the superior mesial region of the frontal lobe. Patients with global aphasia have often incurred a significant ischemic or hemorrhagic event and typically have an array of neurologic impairments. However, there are rare cases where only language has been disrupted and all other cerebral functions remain intact. Patients with isolated global aphasia are unable to communicate or comprehend spoken language. They are unresponsive, but are alert and able to perform meaningful tasks. They may also be able to communicate nonverbal. Malingering is the intentional generation of symptoms for a conscious secondary gain. It is not a neuropsychiatric disorder, merely the intentional manipulation to achieve a goal. It may, at times, be difficult to detect malingering. The clinician may suspect malingering on the observation of inconsistencies in behavior. Circumstantial evidence of malingering may also be obtained through collateral sources. Clinicians have a tendency to over diagnose malingering; malingering does not preclude the presence of an actual physical disorder.  

**Adverse Drug Reactions to Psychiatric Medications**

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. Information about medications the patient is taking and adherence to the prescribed regime, as well as over-the-counter medications, herbal supplements, and drugs of abuse, is important. How such agents can combine to cause problems must also be considered. Acute dystonic reactions and serotonin syndrome are now discussed (NMS was discussed earlier).

**Acute Dystonic Reaction**

An acute dystonic reaction (ADR) is 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.

Any agent with dopamine antagonist activity can cause an ADR, but most are associated with antipsychotic medications. All antipsychotic medications have the potential to cause an ADR, but high-potency antipsychotics such as haloperidol tend to cause more reactions than low-potency antipsychotics such as chlorpromazine. Newer atypical antipsychotics (ie, risperidone, quetiapine) are lower risk.  

Young men are at highest risk, especially those with a high muscle mass. African American and Hispanic patients are also at high 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.29

The pathophysiology of ADRs are unknown. However, dystonia is related to the dopamine pathway and because the symptoms can occur days after the initial blockade of dopamine receptors, it is postulated that secondary dopamine hypersensitivity plays a role.29

The cornerstone of treatment is with anticholinergic medications. Prompt intramuscular (IM) or 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 before 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 by mouth twice a day may be an alternative in those in whom anticholinergic medications are contraindicated.30

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 to 2 mg by mouth twice a day.

**Serotonin Syndrome**

Serotonin syndrome is a reaction to medication that causes excessive serotonin agonism, centrally and peripherally.31 Serotonin syndrome is characterized by a triad of symptoms (Table 3).32

The condition can progress to coma and death, and may require intubation, paralysis, and sedation.

The main treatment of serotonin syndrome is withdrawing the offending agent. This does 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-hydroxytryptamine 2A antagonists, such as cyproheptadine, can be used. Severe cases with hyperthermia often require intubation, paralysis, and sedation as necessary.31,33

**Other Adverse Drug Reactions**

There are other adverse psychiatric drug reactions that can present to the emergency department with altered levels of consciousness. For example, Kimmel and colleagues34 describe a case of encephalopathy caused by increased ammonia levels associated with valproic acid. In addition, cases of lithium toxicity can present with nausea/vomiting, ataxia, and altered mental status including stupor or coma. Patients with psychiatric illness are at increased risk for suicide often times with home medications or street drugs.

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<td><strong>Symptoms of serotonin syndrome</strong></td>
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<td>Neuromuscular hyperactivity</td>
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SUMMARY

An overview of several different psychiatric issues that should be considered when confronted with a patient with a decreased level of consciousness is provided. Symptoms commonly regarded as psychiatric may be caused by systemic illness and in turn often what seems to be systemic illness is in fact psychiatric. Although severe cases may present in this fashion, it is uncommon for patients with psychiatric issues to present with a decreased level of consciousness. Medical causes such as systemic illness or adverse drug reactions need to be ruled out first.

REFERENCES

Psychiatric Considerations