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Catatonia in adults: Epidemiology, clinical features, assessment, and diagnosis

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INTRODUCTION

Catatonia is a behavioral syndrome marked by an inability to move normally despite full physical capacity to do so. The syndrome occurs in the context of many underlying psychiatric and general medical disorders [1]. Within psychiatric nosology, catatonia is not conceptualized as a separate diagnostic entity [2,3]. Rather, the term catatonia is used to specify a subtype of the underlying disorder, similar to the term “psychotic features.” (However, some experts view catatonia as a syndrome that warrants a stand-alone diagnosis [4].)

Recognizing catatonia is important because it may be caused or exacerbated by treatment of the underlying disorder. As an example, antipsychotic drugs used for bipolar or psychotic disorders may worsen catatonia [5].

This topic reviews the epidemiology, pathogenesis, clinical features, assessment, and diagnosis of catatonia, as well as the disorders that can progress to catatonia. The treatment and prognosis of catatonia are discussed separately. (See "[Catatonia: Treatment and prognosis](#)".)

EPIDEMIOLOGY

The estimated incidence of catatonia has been studied primarily in acutely ill psychiatric inpatients [3]. The incidence is approximately 10 percent, but estimates range from 5 to 20

percent, based upon prospective studies conducted for one to twelve months at individual psychiatric units [6-14]. The range is probably due in part to differences in study methods, including how catatonia was defined. The syndrome may go unrecognized, leading to the false conclusion that it is rare. In a Dutch study of 139 acutely psychotic inpatients, the treatment team diagnosed catatonia nine times less often than the research team (2 versus 18 percent of patients) [8].

It is not clear if there are any sociodemographic risk factors for catatonia. Catatonia seems to occur more commonly in patients with unipolar major depression or bipolar disorder, compared with other disorders [6]. In addition, catatonia may perhaps occur more often in patients with prior episodes. (See '[Underlying disorders](#)' below and '[Recurrence](#)' below.)

PATHOGENESIS

Catatonia occurs in the context of an underlying psychiatric or general medical disorder, and treatments that are used for these underlying disorders may cause or exacerbate catatonia. As an example, antipsychotic drugs used for bipolar or psychotic disorders may worsen catatonia [5]. (See "[Catatonia: Treatment and prognosis](#)", section on '[Avoid dopamine blocking drugs](#)'.)

The pathophysiology of catatonia is unknown. Neurobiologic correlates of catatonia have been identified, but it is not clear if these findings represent etiologic causes or sequelae of the disorder [15].

Neurobiologic studies have led to a hypothesis that catatonia involves pathways that connect the basal ganglia with the cortex and thalamus [16]. Neuropathologic studies of patients with catatonic schizophrenia have found alterations in the basal ganglia (caudate nucleus, nucleus accumbens, and pallidum) and thalamus [17]. Neuroimaging studies suggest that catatonia may possibly involve functional alterations in the prefrontal, orbitofrontal, and parietal cortices [18,19]. In addition, neuropsychologic tests of remitted catatonic patients found a deficit in visual-spatial abilities that may involve right parietal cortex function [17,20]. Neurotransmitter studies suggest that catatonia may be associated with decreased activity at gamma-aminobutyric acid A and dopamine D2 receptors, and increased activity at N-methyl-D-aspartate receptors [19,21].

Some patients may possibly have a genetic predisposition to catatonia. Preliminary evidence suggests that genes on chromosomes 15 and 22 may be linked to the periodic form of catatonia [22-25]. (See '[Other forms](#)' below.)

CLINICAL FEATURES

The core feature of catatonia is a motor disturbance in which patients are unable to move normally despite full physical capacity in the limbs and trunk [1]. The disturbance can range from marked reduction in movements to marked agitation. Starting, stopping, and planning movement can be impaired, and motor behavior may be repetitive, purposeless, impervious to external stimuli, and contrary to intent [11].

Small prospective studies of catatonia and case series have found that catatonia is marked by heterogeneous signs that are observed or elicited ([table 1](#) and [table 2](#)); the most common are [1,11,26]:

- Immobility (hypokinesia or akinesia)
- Mutism
- Stupor (decreased alertness and response to stimuli)
- Negativism (resistance to all instructions or all attempts to be moved)
- Waxy flexibility
- Posturing
- Excessive, purposeless motor activity (excitement)
- Staring
- Echophenomena
 - Echolalia – Senseless repetition of another person's utterances
 - Echopraxia – Senseless repetition of another person's movements

Onset of catatonia can be either acute or insidious [1,9]. Following remission, some patients cannot recall their experience during the episode, whereas others describe an awareness of their activity and the inability to communicate [27].

Catatonia occurs in patients who are severely ill with an underlying psychiatric or general medical disorder [3,28] (see '[Underlying disorders](#)' below). Thus, catatonia is typically seen in hospitalized patients.

Subtypes — There are several subtypes of catatonia, based upon the specific nature of the movement disturbance and other associated features [1,29,30]. Although psychiatric nosology does not formally recognize the subtypes as diagnostic entities, the subtypes are clinically useful constructs that are consistent with the symptoms that constitute a diagnosis of catatonia (See '[Psychiatric nosology](#)' below.)

The behavioral phenotype of catatonia exists on a continuum between retarded and excited subtypes, depending upon the motor disturbance. In addition, the severity of catatonia exists on a continuum between nonmalignant and malignant, depending upon other associated features. The three principal forms of catatonia in order of incidence are retarded, excited, and malignant. While a factor-analytic study of 314 psychotic patients supported separation of retarded and excited catatonia as subtypes [31], patients can exhibit signs of both subtypes concurrently [32]. Subtypes are not necessarily stable during a catatonic episode, and patients may transition from retarded to excited catatonia or vice versa [33,34]. In addition, retarded and excited catatonia can each progress to malignant catatonia; alternatively, malignant catatonia can occur de novo.

Retarded catatonia — The retarded form of catatonia is characterized by mutism, inhibited movement, posturing, negativism, and staring [1,10]. Postures may be mundane (eg, sitting or standing in the same position for hours) or unusual (eg, head raised above the bed as if on a pillow). Response to voice and noxious stimuli is decreased. Although speech and spontaneous movements are reduced, some patients appear to be alert and aware of the environment. In more severe cases, eating and drinking may cease and stupor and incontinence may occur [34].

Excited catatonia — Excited catatonia is characterized by excessive and purposeless motor activity in both the upper and lower limbs (hyperkinesia), restlessness, stereotypy, impulsivity, frenzy, and combativeness [1,10]. Delirium may occur in severe cases, and the excess motor activity may cause self-injury or harm to others.

Malignant catatonia — Malignant catatonia (also called lethal catatonia) is a life-threatening condition that is characterized by fever (less likely in older patients), autonomic instability (labile or elevated blood pressure, tachycardia, tachypnea, and diaphoresis), delirium, and rigidity [1,16]. The syndrome is typically fulminant and progresses rapidly within a few days [35]. Common but nonspecific laboratory findings include leukocytosis, elevated creatine kinase, and low serum iron [16,36]. Signs of malignant catatonia overlap with signs of the neuroleptic malignant syndrome (NMS). (See '[Differential diagnosis](#)' below.)

Other forms — Other, infrequent forms of catatonia may occur [1]. One example is periodic catatonia, which is marked by waxing and waning of catatonic signs [29] or periods of retarded catatonia alternating with excited catatonia [1].

Underlying disorders — Psychiatric nosology does not conceptualize catatonia as a specific disease; rather, catatonia is a syndrome that occurs in the context of an underlying psychiatric or general medical illness [2,3,16]. Multiple underlying conditions may be present in the same catatonic patient [32,37,38]. However, it is not known if comorbidity increases the risk of

catatonia. None of the underlying disorders appear to be associated with a specific catatonic sign or subtype [9,32].

Although catatonia can occur in the context of many mental disorders, it is most often found in [3,39-42]:

- Bipolar I disorder
- Bipolar II disorder
- Unipolar major depression (major depressive disorder)
- Psychotic disorders
 - Schizophrenia
 - Schizoaffective disorder
 - Brief psychotic disorder
 - Schizophreniform disorder
- Autism spectrum disorder
- Delirium

Many general medical disorders can also lead to catatonia. A review of case reports found that catatonia occurred in more than 100 general medical conditions [32]; these included infectious, metabolic, neurologic, and rheumatologic disorders [1,3,43,44]. In addition, a study of 136 patients treated on an intensive care unit with mechanical ventilation and/or vasopressors found that catatonia was present in approximately 35 percent [45].

On acute inpatient psychiatric units, catatonia most frequently occurs in the context of bipolar disorder or unipolar major depression [6,30,39]. A study of 148 patients with catatonia found that the underlying condition was bipolar disorder or unipolar major depression in 46 percent of patients, psychotic disorder in 26 percent, general medical condition in 20 percent, and another psychiatric disorder in 8 percent of patients [46]. Other reviews have found a similar distribution of underlying diagnoses [47].

The presence of catatonia reflects the severity of the patient's underlying illness. As an example, a study of 148 catatonic patients found that nearly 75 percent were concurrently psychotic [46].

Treatments that are used for the underlying disorder may cause or exacerbate catatonia (eg, antipsychotic drugs used for bipolar or psychotic disorders may worsen catatonia) [5]. (See "[Catatonia: Treatment and prognosis](#)", section on 'Avoid dopamine blocking drugs'.)

Recurrence — Patients who recover from catatonic episodes may suffer recurrences. A retrospective study of patients with bipolar disorder and catatonia (n = 26) found that a prior history of catatonia was present in 38 percent [48].

ASSESSMENT

Signs of catatonia can be observed or elicited ([table 1](#) and [table 2](#)) [28]. Clinicians may overlook these signs because of the severity of associated psychopathology (eg, psychosis). In addition, common signs of catatonia such as mutism, posturing, and rigidity may be absent; many catatonic patients speak and move about. Thus, patients exhibiting only one apparent catatonic sign should be examined for others.

For patients with an underlying psychiatric disorder, the motor dysregulation of catatonia can be similar to the underlying psychopathology. As an example, the increased motor activity and impulsivity observed in mania may progress to excited catatonia. Mania uncomplicated by catatonia is marked by goal-directed activity and intact cognition. By contrast, activity in catatonia is often repetitive and not goal-directed. As another example, the psychomotor retardation, anergia, anhedonia, and anorexia observed in major depression may resemble retarded catatonia. The hypokinesia and refusal to eat or drink that characterize severe depression may be catatonic signs, and should prompt an assessment for other catatonic signs. Clinical features that often occur in depression not complicated by catatonia include suicidality, sleep disturbance, and feelings of worthlessness, guilt, helplessness, and hopelessness. In addition, catatonia can be distinguished by additional motor signs, such as posturing, rigidity, and negativism, as well as a positive response to the [lorazepam](#) challenge test. (See '[Underlying disorders](#)' above and '[Lorazepam challenge](#)' below.)

Rating scales have been used for research purposes to systematically examine patients at the initial assessment and quantify progress during treatment, but their utility for routine clinical care is not well-established [26,46,49]. The most widely used measure is the Bush-Francis Catatonia Rating Scale, which is a 23-item rating scale with good inter-rater reliability (0.93) and construct validity; the first 14 items can be used to screen for catatonia [50]. Each item is defined, the procedure for rating each item is described, and the scale appears to be sensitive to clinical change [30]. The Northoff Catatonia Scale and the Kanner Catatonia Scale are reasonable alternatives [51,52].

There is no laboratory test specific for catatonia. However, malignant catatonia is associated with non-specific test results that include leukocytosis, increased creatine kinase, and low serum iron ([table 3](#)) [36,53-55].

The underlying disorder may be unknown in some patients with catatonia, in which case the initial clinical evaluation should include the following [9,32,46]:

- Psychiatric and general medical history.
- Mental status and physical examination.
- Laboratory tests – Tests should be guided by the symptoms, history, and findings on the physical examination. Patients with an established underlying disorder (eg, unipolar major depression with psychotic features) may not need any tests.

Catatonic stupor and mutism may preclude assessing the patient for symptoms of the underlying disorder. However, family and friends can often compensate for the patient's inability to provide a history. After catatonia resolves, the patient should be re-evaluated for psychopathology or a general medical disorder. (See "[Unipolar depression in adults: Assessment and diagnosis](#)" and "[Bipolar disorder in adults: Clinical features](#)" and "[Schizophrenia in adults: Clinical features, assessment, and diagnosis](#)".)

Lorazepam challenge — Nearly every catatonic patient is treated initially with [lorazepam](#) or another benzodiazepine (patients with malignant catatonia may be quickly switched to electroconvulsive therapy); the response to the first one or two doses can help make the diagnosis of catatonia. Case series suggest using an intravenous (IV) bolus of lorazepam 1 to 2 mg [32]. IV administration allows for rapid, complete absorption, although intramuscular (IM) injections and oral administration have also been used [30,56]. Intravenous benzodiazepines should be injected or infused slowly. In addition, clinicians should be cognizant of concomitant drugs that can cause sedation or respiratory depression, eg, opioids. Benzodiazepines cause varying degrees of motor incoordination and increase the risk of falls [12].

Partial, temporary relief of signs 5 to 10 minutes after IV administration of [lorazepam](#) is consistent with a diagnosis of catatonia [1,29]. If there is no change and the patient remains awake with stable vital signs, a second dose is given, and the patient is examined within 5 to 10 minutes, although observation can continue for longer periods. A negative response does not rule out catatonia, and occurs in approximately 20 percent or more of catatonic patients.

Reasonable alternatives to [lorazepam](#) are available. A randomized trial that compared a single dose of oral [oxazepam](#) 60 mg with a single dose of oral lorazepam 2 mg in 17 patients found that the benefit of each drug was comparable [57]. In case reports, oral [zolpidem](#) 10 mg was useful [26,37,58-60].

The treatment of catatonia is discussed separately. (See "[Catatonia: Treatment and prognosis](#)".)

DIAGNOSIS

A minimum of two to four signs for at least several hours is required to make the diagnosis of catatonia ([table 1](#) and [table 2](#)) [1,12,26,50]. However, neither the number of catatonic features nor their duration is established, and no one sign is pathognomonic [47]. A positive [lorazepam](#) challenge test can help verify the diagnosis. (See '[Lorazepam challenge](#)' above.)

Psychiatric nosology — According to psychiatric nosology, catatonia is not a separate, specific disease; rather, catatonia is a behavioral syndrome that is diagnosed in patients who are ill with an underlying psychiatric and/or general medical disorder [2,3]. In the American Psychiatric Association's Diagnostic and Statistical Manual, Fifth Edition, Text Revision (DSM-5-TR), the term catatonia is used to specify a subtype of the underlying disorder, similar to the term “psychotic features” [3]. The underlying psychiatric disorders that can give rise to catatonia include autism spectrum disorder, bipolar disorders, psychotic disorders, and unipolar major depression.

In DSM-5-TR, criteria for catatonia are met when the clinical picture is dominated by at least three of the following [3]:

- Stupor (decreased psychomotor activity or decreased reactivity to the environment)
- Catalepsy (passively allowing the examiner to position the body or a body part)
- Waxy flexibility (slight, even resistance to positioning by the examiner, as in bending a candle)
- Mutism (lack of verbal response; not applicable to patients with an established aphasia)
- Negativism (motiveless resistance to instructions or external stimuli)
- Posturing (voluntarily maintaining a position of the body or a body part against gravity for a long time)
- Mannerisms (odd movements)
- Stereotypy (repetitive movements that are not goal directed and often are awkward or stiff)
- Agitation or excessive motor activity that is purposeless and not influenced by external stimuli
- Grimacing

- Echolalia (mimicking another person's speech)
- Echopraxia (mimicking another person's movements)

DIFFERENTIAL DIAGNOSIS

Signs of catatonia can overlap with signs of other disorders; in particular, malignant catatonia may resemble the neuroleptic malignant syndrome (NMS) [5,61]. Both disorders present with fever, autonomic instability, rigidity, and delirium. Patients who present with these signs should be assessed with neuroimaging and a lumbar puncture to rule out central nervous system and systemic infection.

The differential diagnosis of catatonia includes a number of conditions, some of which can cause catatonia (eg, delirium, stroke, and nonconvulsive status epilepticus) [6]:

- **Neuroleptic malignant syndrome** – The neuroleptic malignant syndrome (NMS) and malignant catatonia are life-threatening disorders with similar clinical features. Both disorders can present with delirium, autonomic instability, fever, and rigidity [61,62]. In addition, an elevated creatinine kinase and white blood count and low serum iron are common to NMS and malignant catatonia. A review of 292 cases of malignant catatonia found that the clinical features were indistinguishable from NMS in more than 20 percent of cases [63]. (See "[Neuroleptic malignant syndrome](#)", section on '[Clinical manifestations](#)'.)

Many authorities consider NMS a drug-induced form of malignant catatonia, based upon their overlapping clinical features as well as similarities in the hypothesized pathophysiology for each disorder [1,5,9,11,16,21,26,62,64]. In addition, discontinuing antipsychotics and starting benzodiazepines can be helpful for both NMS and malignant catatonia, and electroconvulsive therapy can benefit patients with either disorder even after failed pharmacotherapy [1,61].

However, it is not established that NMS is a subtype of catatonia [18,46]. Malignant catatonia can present with signs (eg, impulsivity, posturing, and repetitive stereotypic movements) that are not generally observed in NMS [46,65,66], affective symptoms that can occur in catatonia do not seem to be as prominent in NMS [18], and laboratory abnormalities that occur in NMS may not occur as consistently in patients with malignant catatonia [46]. Some authorities hypothesize that NMS and malignant catatonia have overlapping but distinct pathophysiologic mechanisms [18]. Establishing the diagnosis of NMS can lead to treatment that differs in some respects from that of catatonia [61,63],

although some authorities recommend a common and consistent therapeutic approach [64]. (See "[Neuroleptic malignant syndrome](#)", section on 'Treatment'.)

- **Serotonin syndrome** – Serotonin syndrome and malignant catatonia can both present with delirium, autonomic instability, hyperthermia, and rigidity. Distinguishing features of the serotonin syndrome include ingestion of a serotonergic drug such as an SSRI, and the prominence of hyperreflexia, myoclonus, nausea, vomiting, and diarrhea. Some authorities view the serotonin syndrome as a form of catatonia, due to similarities in presenting signs and response to specific treatments [67]. (See "[Serotonin syndrome \(serotonin toxicity\)](#)".)
- **Malignant hyperthermia** – Malignant hyperthermia and malignant catatonia can both present with autonomic instability, hyperthermia, and rigidity. Malignant hyperthermia is a rare genetic disorder that occurs in susceptible patients exposed to an inhalational anesthetic (eg, halothane) or a depolarizing muscle relaxant (eg, [succinylcholine](#)). The distinguishing feature of malignant hyperthermia is its clinical setting, and various procedures are used to diagnose it (muscle contracture testing, muscle biopsy, and genetic testing). (See "[Malignant hyperthermia: Diagnosis and management of acute crisis](#)".)
- **Akinetic mutism** – Akinetic mutism and catatonia may both manifest with immobility, mutism, and waxy flexibility. However, akinetic mutism does not include posturing, echolalia, or echopraxia; is not responsive to benzodiazepines; and the lesions to the prefrontal or premotor areas that cause akinetic mutism should be detectable with neuroimaging. (See "[Stupor and coma in adults](#)", section on 'Akinetic mutism'.)
- **Nonconvulsive status epilepticus** – Nonconvulsive status epilepticus and catatonia can both present with stupor and respond positively to benzodiazepines. Nonconvulsive status epilepticus may be distinguished by the presence of subtle rhythmic twitching and ocular movement abnormalities, and is diagnosed by epileptic activity on the electroencephalogram (EEG). By contrast, catatonia is associated with either a normal EEG or diffuse, nonspecific changes (eg, slowing). (See "[Nonconvulsive status epilepticus: Classification, clinical features, and diagnosis](#)".)
- **Locked-in syndrome** – Locked-in syndrome is characterized by immobility and mutism. However, no other signs of catatonia are present, neuroimaging typically reveals brainstem lesions, and locked-in patients usually attempt to communicate with eye movements and blinking, whereas catatonic patients are not motivated to communicate. (See "[Locked-in syndrome](#)".)

- **Stiff-person syndrome** – Stiff-person syndrome is marked by rigidity and a positive response to a benzodiazepine, and autonomic instability may occur as well, all of which may occur in catatonia. However, stiff-person syndrome is generally accompanied by a fixed deformity of the spine and anti-glutamic acid decarboxylase antibodies, which are not observed in catatonia. In addition, catatonic signs such as mutism and posturing are not part of stiff-person syndrome. (See "[Stiff-person syndrome](#)".)
- **Parkinson disease** – Retarded catatonia and late-stage untreated Parkinson disease may both present with immobility, rigidity, and decreased speech output. In addition, major depression can occur in Parkinson disease and may underlie catatonia. However, catatonia often includes odd postures and a positive response to benzodiazepines, whereas distinguishing features of Parkinson disease include rest tremor, postural instability, and a positive response to levodopa. (See "[Clinical manifestations of Parkinson disease](#)".)
- **Stroke** – Stroke can manifest with mutism, but focal neurologic signs and positive findings on neuroimaging are often present as well, and other signs of catatonia are absent. (See "[Overview of the evaluation of stroke](#)".)
- **Delirium** – Catatonia may include periods of delirium, and delirium may include some features of catatonia. A study of 136 critically ill patients on mechanical ventilation and/or vasopressors found that catatonia plus delirium were both present in approximately 30 percent [45].

Patients with delirium and catatonia present with both stupor (decreased alertness and response to stimuli), hypokinesia or hyperkinesia, and limited speech output, but catatonia is usually accompanied by other distinguishing features, including posturing, rigidity, and negativism, as well as a positive response to the [lorazepam](#) challenge test. (See "[Diagnosis of delirium and confusional states](#)".)

- **Dementia** – End-stage dementia and catatonia may both manifest with mutism and hypokinesia. However, catatonia is usually accompanied by other distinguishing features, including posturing, rigidity, negativism, and a positive response to the [lorazepam](#) challenge test.
- **Elective mutism** – Elective mutism is the failure to speak in an otherwise vigilant patient, who may selectively refuse to speak with some individuals and speak with others. The disorder is usually accompanied by a personality disorder, preexisting stressor, and no other signs of catatonia.

- **Anti-N-methyl-D-aspartate (NMDA) receptor encephalitis** – Anti-N-methyl-D-aspartate (NMDA) receptor encephalitis is an autoimmune disorder with prominent neuropsychiatric symptoms, including catatonia in approximately 40 percent of patients [68]. This form of encephalitis may rapidly progress to seizures, hypoventilation, and coma.

Anti-NMDA receptor encephalitis and catatonia both can include stupor and motor disturbances; in addition, both anti-NMDA receptor encephalitis and the malignant subtype of catatonia can present with autonomic instability. However, catatonia can be distinguished from this form of encephalitis by the presence of other features, including waxy flexibility, staring, and negativism, as well as a positive response to the [lorazepam challenge test](#). (See ["Autoimmune \(including paraneoplastic\) encephalitis: Clinical features and diagnosis"](#), section on 'Anti-NMDA receptor encephalitis'.)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See ["Society guideline links: Depressive disorders"](#).)

SUMMARY

- **Epidemiology** – The estimated incidence of catatonia in acutely ill psychiatric inpatients is approximately 10 percent. (See ["Epidemiology"](#) above.)
- **Clinical features** – Catatonia is a behavioral syndrome that is marked by an inability to move normally despite full physical capacity to do so. The syndrome occurs in the context of an underlying psychiatric and/or general medical disorder. Relatively common signs of catatonia include immobility, mutism, and excessive, purposeless motor activity. (See ["Clinical features"](#) above.)
 - **Subtypes of catatonia** – The three principal subtypes of catatonia are retarded, excited, and malignant. Patients can exhibit signs of more than one subtype, and subtypes are not necessarily stable during a catatonic episode. (See ["Subtypes"](#) above.)
 - **Underlying psychiatric disorders** – The most common underlying psychopathology is bipolar disorder, unipolar major depression, psychotic disorder, and autism spectrum disorder.

- **Assessment** – Signs of catatonia can be either observed ([table 1](#)) or elicited ([table 2](#)). The initial clinical evaluation for determining the underlying disorder should include a history, mental status and physical examination, and laboratory tests guided by symptoms and findings on examination. (See '[Assessment](#)' above.)
- **Lorazepam challenge** – An intravenous (IV) bolus of [lorazepam](#) 1 to 2 mg can help diagnose catatonia. Partial, temporary relief of signs 5 to 10 minutes after IV administration of lorazepam is consistent with a diagnosis of catatonia. If there is no change, a second dose is given. A negative response does not rule out catatonia, and occurs in approximately 20 percent or more of catatonic patients. (See '[Lorazepam challenge](#)' above.)
- **Diagnosis** – The diagnosis of catatonia requires a minimum of two to four signs ([table 1](#) and [table 2](#)) for at least several hours. However, neither the number of catatonic features nor their duration is established. (See '[Diagnosis](#)' above.)
- **Differential diagnosis** – Signs of catatonia can overlap with signs of other disorders; in particular, malignant catatonia resembles the neuroleptic malignant syndrome (NMS). Both are life-threatening disorders that can present with delirium, autonomic instability, fever, and rigidity.

The differential diagnosis of catatonia also includes the serotonin syndrome, malignant hyperthermia, akinetic mutism, nonconvulsive status epilepticus, locked-in syndrome, stiff-person syndrome, Parkinson disease, stroke, delirium, dementia, elective mutism, and anti-N-methyl-D-aspartate receptor encephalitis. (See '[Differential diagnosis](#)' above.)

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