

Catatonia

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Abstract Catatonia is an important phenomenon in both psychiatry and general medicine. This article provides an overview of the key aspects of catatonia, including clinical features, differential diagnoses, management and prognosis. The different types of catatonia, the position of catatonia in the psychiatric classificatory systems, use of catatonia rating scales and the association between catatonia and neuroleptic malignant syndrome are also covered. Abnormalities that have been hypothesised as being possible underlying mechanisms in catatonia are highlighted. The article aims to provide clinicians with a comprehensive update on the subject, with information derived from an extensive range of relevant references.

The concept of catatonia was first described by Kahlbaum (1874). Catatonic stupor is one of the most dramatic psychiatric presentations, but is becoming increasingly rare in the Western world. However, it has been suggested that catatonia is under-recognised and under-diagnosed (Van der Heijden *et al*, 2005). Although the introduction of antipsychotics has reduced the incidence of catatonia, it is still not uncommon (Stompe *et al*, 2002) and its detection rate can be significantly improved by using a standardised rating scale (Van der Heijden *et al*, 2005).

Mechanism of catatonia

The exact cause of catatonia has not been elucidated, but a number of hypotheses have been offered.

According to Northoff (2002), a 'top-down modulation' of basal ganglia due to deficiency of cortical gamma-aminobutyric acid (GABA), the primary inhibitory neurotransmitter of the brain, may explain the motor symptoms of catatonia. This explanation might account for the dramatic therapeutic effect of benzodiazepines, which cause an increase in GABA activity. Similarly, hyperactivity of glutamate, the primary excitatory neurotransmitter, has also been suggested as an underlying neurochemical dysfunction (Northoff *et al*, 1997).

Osman & Khurasani (1994) have suggested that catatonia is caused by a sudden and massive blockade of dopamine. This may explain why dopamine-blocking antipsychotics are not generally beneficial in catatonia. Indeed, by exacerbating dopamine

deficiency, antipsychotics may actually precipitate a worsening of the condition.

Clozapine-withdrawal catatonia is postulated to be due to cholinergic and serotonergic rebound hyperactivity (Yeh *et al*, 2004).

In chronic catatonia with prominent speech abnormalities, positron emission tomography (PET) has identified abnormalities in metabolism bilaterally in the thalamus and frontal lobes (Lauer *et al*, 2001).

A very interesting hypothesis proposed by Moskowitz (2004) suggests that catatonia may be understood as an evolutionary fear response, originating in ancestral encounters with carnivores whose predatory instincts were triggered by movement. This response, of remaining still, is now expressed in a range of major psychiatric or medical conditions, where catatonic stupor may represent a common 'end-state' response to feelings of imminent doom.

Clinical features of catatonia

Catatonia is a syndrome that encompasses more than two dozen signs, some of which are relatively non-specific. The catatonic signs are listed in alphabetical order in Box 1 and some of the more common ones are briefly described below.

Stupor

Stupor is the classic and most striking catatonic sign. It is a combination of immobility and mutism, although the two can also occur independently.

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Posturing

The patient is able to maintain the same posture for long periods. A classic example is the 'crucifix'. An extreme version of posturing is catalepsy.

Waxy flexibility (*cerea flexibilitas*)

The examiner is able to position the patient in what would be highly uncomfortable postures, which are maintained for a considerable period of time.

Negativism (*Gegenhalten*)

The patient resists the attempts of the examiner to move parts of their body and, according to the original definition, the resistance offered is exactly equal to the strength applied.

Automatic obedience

The patient demonstrates exaggerated cooperation, automatically obeying every instruction of the examiner. *Mitmachen* and *Mitgehen* are forms of automatic obedience. In *Mitmachen* the body of the patient can be put into any posture, even if the patient is given instructions to resist. *Mitgehen* is an extreme form of automatic obedience in which the examiner is able to move the patient's body with the slightest touch, but the body part immediately returns to the original position (unlike in waxy flexibility).

Ambitendency

The patient alternates between resistance to and cooperation with the examiner's instructions; for example, when asked to shake hands, the patient repeatedly extends and withdraws the hand.

Psychological pillow

The patient assumes a reclining posture, with their head a few inches above the bed surface, and is able to maintain this position for prolonged periods.

Forced grasping

The patient forcibly and repeatedly grasps the examiner's hand when offered.

Obstruction

The patient stops suddenly in the course of a movement and is generally unable to give a reason. This appears to be the motor counterpart of thought block.

Box 1 Signs of catatonia

- Ambitendency
- Automatic obedience
- Aversion
- Catalepsy
- Echolalia
- Echopraxia
- Excitement
- Forced grasping
- *Gegenhalten*
- Grimacing
- Immobility
- Logorrhoea
- Mannerisms
- *Mitgehen*
- *Mitmachen*
- Mutism
- Negativism
- Obstruction
- Perseveration
- Posturing
- Psychological pillow
- Rigidity
- Staring
- Stereotypies
- Stupor
- Verbigeration
- Waxy flexibility
- Withdrawal

Echopraxia

The patient imitates the actions of the interviewer.

Aversion

The patient turns away from the examiner when addressed.

Mannerisms

These are repetitive, goal-directed movements (e.g. saluting).

Stereotypies

These are repetitive, regular movements that are not goal-directed (e.g. rocking).

Motor perseveration

The patient persists with a particular movement that has lost its initial relevance.

Excitement

The patient displays excessive, purposeless motor activity that is not influenced by external stimuli.

Speech abnormalities

Echolalia, logorrhoea and verbigeration are the main speech abnormalities in catatonia. Echolalia refers to the repetition of the examiner's words. Logorrhoea is characterised by incessant, incoherent and usually monotonous speech. Verbigeration is a form of verbal perseveration in which the patient repeats certain syllables (logoclonia), words (palilalia), phrases or sentences.

Other catatonic signs

If, in addition to prominent catatonic signs, the patient exhibits hyperpyrexia, clouding of consciousness and autonomic instability, a diagnosis of lethal or malignant catatonia should be considered.

Differential diagnoses of catatonia

Although traditionally linked to schizophrenia, catatonia is more commonly associated with mood disorders (Pommepuy & Januel, 2002). For example, Abrams & Taylor (1976) recorded that, in a sample of 55 people with catatonia, only four had schizophrenia and more than two-thirds had affective disorders, especially mania. Similarly, Barnes *et al* (1986) reported only one person with schizophrenia in their sample of 25, but nine with affective disorders.

Increasing age may be a significant risk factor for catatonia in depression (Starkstein *et al*, 1996). Catatonia may also occur as a feature of post-partum psychiatric disorders (Lai & Huang, 2004).

Temporal lobe epilepsy is a recognised cause of catatonia (Kirubakaran *et al*, 1987).

Catatonia is a potential risk of abrupt discontinuation of clozapine, and is reversible by reinstatement of the drug (Yeh *et al*, 2004).

Immobility seen in advanced dementia might reflect a catatonic state seen in other serious organic disorders, and may respond to lorazepam (Alisky, 2004).

There have been case reports suggesting that patients with thrombotic thrombocytopenic purpura may be at higher risk of developing catatonia (Yacoub *et al*, 2004).

Catatonia induced by cocaine (Gingrich *et al*, 1998) and ecstasy (Masi *et al*, 2002) have been reported. Prescribed medication such as ciprofloxacin (Akhtar & Ahmad, 1993) can also cause catatonia.

Box 2 Differential diagnoses of catatonia

- Schizophrenia
- Depression
- Mania
- Organic disorders: e.g. infections, epilepsy, metabolic disorders
- Drugs: prescribed or recreational
- Hysteria (psychogenic catatonia)
- Idiopathic

Metabolic abnormalities such as hyponatraemia may cause catatonia (Lee & Schwartz, 1997), and people with rare metabolic disorders such as Wilson's disease (Davis & Borde, 1993) and Tay-Sachs disease (Rosebush *et al*, 1995) may also present with the condition.

Prior brain injury and physical illness at onset of psychosis are more common in patients who subsequently develop catatonia than in those who do not (Wilcox & Nasrallah, 1986). A history of severe infectious disease in childhood, including rheumatic fever, is associated with an increased risk of catatonia in adult life (Wilcox, 1986).

Hysteria has also been traditionally mentioned as a cause of catatonia.

In a significant minority, no cause is identified (Barnes *et al*, 1986). Benegal *et al* (1993) reported a high prevalence of idiopathic catatonia, and found it to be more common in females.

The differential diagnoses of catatonia are summarised in Box 2.

Catatonia in ICD-10 and DSM-IV

As highlighted above, it is becoming increasingly clear that catatonia is more commonly a consequence of mood disorders than of schizophrenia. However, historically catatonia has been regarded as being much more strongly associated with schizophrenia. After Kahlbaum first described catatonia, Kraepelin included it as a type of dementia praecox, and when Bleuler introduced the concept of schizophrenia, he recognised catatonia as one of the schizophrenic subtypes. This bias, giving schizophrenia an exaggerated place in the discussion of catatonia, continues to be reflected in ICD-10 (World Health Organization, 1992) and DSM-IV (American Psychiatric Association, 1994).

ICD-10

The ICD-10 diagnosis of catatonic schizophrenia (category F20.2) requires that the patient prominently

exhibits at least one of the following catatonic features, for at least 2 weeks: stupor, excitement, posturing, negativism, rigidity, waxy flexibility and command automatism (automatic obedience).

If a patient with severe depression is in a stupor, a diagnosis of 'severe depressive episode with psychotic symptoms' (F32.3) is made, even if there are no delusions or hallucinations.

Similarly, a patient with manic stupor will be diagnosed as having 'mania with psychotic symptoms' (F30.2).

Thus, for depression or mania, only stupor, which is the most extreme of catatonic signs, seems to have diagnostic implications, whereas for schizophrenia a broader range of signs are considered relevant.

Catatonia due to physical causes is diagnosed as 'organic catatonic disorder' (F06.1).

DSM-IV

In DSM-IV a diagnosis of 'schizophrenia, catatonic type' (code 295.20) is made if the clinical picture is dominated by at least two of the following: motor immobility, excessive motor activity, extreme negativism, peculiarities of voluntary movements, and echolalia/echopraxia.

If a physical cause is identified the diagnosis is 'catatonic disorder due to a medical condition' (code 293.89).

As in ICD-10, there is no separate diagnostic category for catatonia due to either depression or mania, but catatonia can be added as a specifier in mood disorders.

Types of catatonia

Taylor & Fink (2003) believe that catatonia should be classified as an independent syndrome with the following subtypes: non-malignant, delirious and malignant. The non-malignant type refers to the classic features first described by Kahlbaum, the delirious type includes delirious mania, and the malignant type includes lethal catatonia, neuroleptic malignant syndrome and serotonin syndrome.

Van Den Eede & Sabbe (2004) have proposed an alternative classificatory system. They divide catatonia broadly into non-malignant and malignant types, with each further divided into retarded and excited subtypes. In their system, classic catatonia (Kahlbaum syndrome), delirious mania, neuroleptic malignant syndrome and lethal catatonia would respectively be examples of the non-malignant retarded, non-malignant excited, malignant retarded and malignant excited subtypes.

A further classification, used by the Wernicke-Kleist-Leonhard school of psychiatry, which has

proponents especially in Germany, identifies two main types of catatonia – systematic and periodic. These appear to have significant differences in symptomatology, treatment and prognosis (Pfulmann & Stober, 2001). The systematic type is less genetically determined, has a higher prevalence and earlier age at onset in males (Stober *et al*, 1998), and is associated with mid-gestational infections (Stober, 2001). Periodic catatonia has no differences in either age at onset or prevalence between males and females (Stober *et al*, 1998). Periodic catatonia, according to Stober *et al* (2002), is the first subtype of schizophrenia with confirmed genetic linkage, the susceptibility site being 15q15.

Leonhard (1979) differentiated chronic catatonia, on the basis of the speech abnormalities present, into speech-prompt and speech-sluggish (speech-inactive) types.

A specific category of autistic catatonia has been suggested for catatonia occurring in people with developmental disorders (Hare & Malone, 2004). Similarities between autism and catatonia include abnormal GABA function, small cerebellar structures and susceptibility genes on the long arm of chromosome 15 (Dhossche, 2004).

Ictal catatonia, in which the seizure manifests itself as catatonia, is postulated to be due to involvement of the limbic system (Lim *et al*, 1986). Ictal catatonia is considered a manifestation of non-convulsive status epilepticus.

Rating scales for catatonia

Using a rating scale helps to identify people who have catatonia that might otherwise not have been diagnosed (Van der Heijden *et al*, 2005).

The Bush–Francis Catatonia Rating Scale (BFCRS) appears to be the most widely used instrument for catatonia. The BFCRS has 23 items, and there is also a shorter, 14-item screening version. The reliability and validity of the BFCRS has been established (Bush *et al*, 1996). Ungvari *et al* (2005) reported that using the BFCRS, 32% of 225 patients with chronic schizophrenia met the criteria for catatonia. Their study adds strength to the view that catatonia is still not uncommon and that its incidence is grossly underestimated.

Another catatonia rating scale, the Modified Rogers Scale (MRS), has also been validated (Starkstein *et al*, 1996). The MRS rates abnormalities in movement, volition, speech and overall behaviour, and also aids in the distinction of catatonic signs from seemingly similar extrapyramidal side-effects (Lund *et al*, 1991).

Peralta & Cuesta (2001) have postulated that the presence of three or more of the following

11 signs constitutes a diagnosis of catatonic syndrome: immobility/stupor, mutism, negativism, oppositionism, posturing, catalepsy, automatic obedience, echophenomena, rigidity, verbigeration and withdrawal.

Catatonia and neuroleptic malignant syndrome

Fink (1996) has suggested that, on the basis of the similarity of signs, symptoms and response to treatment, malignant (lethal) catatonia and neuroleptic malignant syndrome should be considered to be the same disorder. Neuroleptic malignant syndrome may be conceptualised as an antipsychotic-induced form of lethal catatonia (Mann *et al*, 2001).

However, others stress the distinction between the two disorders. Castillo *et al* (1989) have highlighted an important clinical difference between lethal catatonia and neuroleptic malignant syndrome, in that the former typically begins with extreme psychotic excitement whereas the latter characteristically starts with severe extrapyramidal muscular rigidity.

Nevertheless, there is general agreement that catatonia represents a highly significant risk factor for subsequent neuroleptic malignant syndrome. White & Robins (1991) reported that in a series of five consecutive cases of neuroleptic malignant syndrome, all were preceded by a catatonic state. Similarly, Raja *et al* (1994) reported that, in their sample of three patients with neuroleptic malignant syndrome, all had shown catatonia just before the onset of the syndrome and had been treated with only low doses of antipsychotics.

Just as the presentation of catatonia appears to have both decreased in rate and become more subtle, it has been suggested that neuroleptic malignant syndrome due to atypical antipsychotic drugs may have less obvious clinical features than the classic syndrome (Reeves *et al*, 2002).

Investigations in a patient presenting with catatonia

The patient will need a comprehensive physical examination, with specific emphasis on neurological signs, and a thorough mental state examination, with special emphasis on identifying catatonic signs. This, coupled with the history, usually provided by an informant, may give a clue as to whether the catatonia is functional or organic, and will also determine whether the patient needs admission to a medical or a psychiatric ward.

All patients should have the first-line investigations listed in Box 3. A diagnosis of neuroleptic malignant

Box 3 Investigations for a patient presenting with catatonia

First-line

- Full blood count
- Renal function tests
- Liver function tests
- Thyroid function tests
- Blood glucose measurement
- Creatine phosphokinase measurement
- Drug screen of urine

Further investigations (depending on findings on physical examination)

- Electrocardiography
- Computed tomography
- Magnetic resonance imaging
- Electroencephalography
- Urine culture
- Blood culture
- Test for syphilis
- Test for HIV
- Heavy-metal screen
- Auto-antibody screen
- Lumbar puncture

syndrome is suggested if there is significant elevation of creatine phosphokinase and total white cell count. Depending on the history and examination, any of the further investigations listed may be required. Electroencephalography may be useful in identifying temporal lobe epilepsy, and is also helpful in distinguishing catatonia from non-convulsive status epilepticus (Louis & Pflaster, 1995).

Management of catatonia

Benzodiazepines are the drugs of choice for catatonia. Patients who are unresponsive or insufficiently responsive to benzodiazepines need electroconvulsive therapy (ECT).

In a prospective, open study (Ungvari *et al*, 1994a), 18 patients with catatonia were treated with either oral lorazepam or intramuscular diazepam; 16 showed significant clinical improvement within 48 h, with two showing complete remission after just one dose. However, nine patients needed subsequent ECT to achieve further improvement. Rosebush *et al* (1990) reported an even more dramatic response to lorazepam, with 12 out of 15 in-patients with catatonia responding completely within 2 h. Small doses of benzodiazepines are effective in both catatonic stupor and catatonic excitement (Ungvari *et al*, 1994b). Organic catatonia also responds well to benzodiazepines (Rosebush *et al*, 1990, 1995).

Like benzodiazepines, ECT is effective in catatonia due to either functional psychiatric disorders (including schizophrenia) or organic causes (Rohland *et al*, 1993); it is even effective for hysterical catatonia (Dabholkar, 1988). Benegal *et al* (1993) reported good response to ECT in their sample of 65 patients with catatonia, which included 30 with idiopathic presentation, 19 with schizophrenia and 16 with depression. The duration of illness was shorter in the idiopathic group. In addition, the number of ECT sessions needed for improvement was not related to the underlying diagnosis.

Emergency ECT is the treatment of choice for malignant catatonia (Pommepuy & Januel, 2002). The Royal College of Psychiatrists' guidelines on ECT (Scott, 2005) specify that ECT may be indicated for catatonia if treatment with lorazepam has been ineffective.

Antipsychotics are generally not recommended during a catatonic phase even if there is an underlying psychotic illness such as schizophrenia, as the risk of precipitating neuroleptic malignant syndrome is considerably increased. However, they may be effective in treatment-resistant catatonia: Hesslinger *et al* (2001) reported that a patient with catatonia unresponsive to benzodiazepines showed dramatic and persistent improvement on risperidone. In a literature review, Van Den Eede *et al* (2005) concluded that atypical antipsychotics may have a role in the treatment of non-malignant catatonia.

Kritzinger & Jordaan (2001) have suggested that carbamazepine is effective in both the acute and maintenance phases of catatonia; in their sample of nine patients, four responded completely to carbamazepine, one showed partial response and the remaining four showed no significant improvement.

Combination of lithium and an antipsychotic may be an option in treatment-resistant catatonic stupor (Climo, 1985).

Mastain *et al* (1995) reported that zolpidem was effective in a patient with catatonia that was resistant to benzodiazepines and ECT.

There are case reports of amantadine (Northoff *et al*, 1999) and memantine (Thomas *et al*, 2005) being effective in catatonia. These are antagonists at the *N*-methyl-D-aspartate (NMDA) receptor. Glutamate acts on the NMDA receptor, and if this receptor is blocked, the neurochemical balance is tilted in favour of GABA. Thus, both pro-GABA and anti-glutamate drugs seem to be beneficial in catatonia.

Catatonia will almost certainly necessitate inpatient treatment. The patient will need intensive nursing and regular monitoring of vital signs, and may need transfer to a psychiatric intensive care unit if there is catatonic excitement. The physical condition of the patient, especially in prolonged

Box 4 Treatment options for catatonia

Treatments that have a strong evidence base

- Benzodiazepines
- Electroconvulsive therapy

Other options (usually reserved for catatonia resistant to benzodiazepines and ECT)

- Mood stabilisers: especially carbamazepine
- Antipsychotics
- NMDA antagonists: amantadine and memantine
- Dopamine agonists (e.g. bromocriptine) and skeletal muscle relaxants (e.g. dantrolene), especially if neuroleptic malignant syndrome is suspected

catatonia, may warrant intravenous fluids and parenteral nutrition. If a diagnosis of neuroleptic malignant syndrome is made, it is preferable to continue treatment on a medical ward. Treatment strategies for neuroleptic malignant syndrome, in addition to benzodiazepines and ECT, include skeletal muscle relaxants (e.g. dantrolene sodium) and dopamine agonists (e.g. bromocriptine).

The important treatment options for catatonia are summarised in Box 4.

Prognosis

Abrams & Taylor (1976) reported a favourable overall response to treatment for their sample of 55 patients admitted with catatonia, with two-thirds showing marked improvement or remission. Although the overall prognosis was excellent, a high incidence of recurrent catatonic episodes was reported for idiopathic catatonia and catatonia due to affective disorders (Barnes *et al*, 1986). Cognitive impairment and deficits in activities of daily living were reported to be more severe in catatonic depression than in non-catatonic depression (Starkstein *et al*, 1996).

Suzuki *et al* (2005) have noted that, although ECT is extremely effective in the acute catatonic phase, there is a high relapse rate within a year. They have suggested that continuation ECT is an efficacious treatment for maintaining response for those who relapse after initially responding to ECT.

The presence of catatonic features in chronic schizophrenia is an additional poor prognostic factor in an already severely disabling illness (Ungvari *et al*, 2005).

In general, the prognosis for the acute catatonic phase seems to be good, but the long-term prognosis probably depends on the underlying cause of the catatonia.

Complications of catatonia

Obviously, if a patient with catatonia does not eat or drink for prolonged periods this will lead to dehydration and its attendant complications. The immobility of catatonia may increase the risk of deep-vein thrombosis (Morioka *et al*, 1997). McCall *et al* (1995) have highlighted the increased risk of death due to pulmonary embolism in patients with persistent catatonia; such deaths occurred only after the second week of catatonia, often without warning. During the phase of catatonic excitement, the patient may pose a significant risk of harm to self and others.

Conclusions

Catatonia is still not uncommon in Western countries. It is more commonly associated with mood disorders than with schizophrenia, but its underlying mechanism has still not been elucidated. Catatonic stupor occurs only rarely, and the majority of patients with catatonia present with subtle signs that can be easily missed, unless specifically looked for.

Although catatonia occurs in both functional and organic disorders, the treatment of the catatonic phase is essentially the same, and most patients respond well to benzodiazepines or ECT. In some cases, treatment of the underlying disorder may have to be suspended (e.g. not using antipsychotics in acute catatonic schizophrenia) until the catatonic phase is resolved. This suggests that catatonia is a unique syndrome that requires treatment in its own right, independent of any underlying disorder. Catatonia includes several individual signs and symptoms. However, within the syndrome, certain features, such as stupor, posturing, waxy flexibility and negativism, seem more specific to the catatonia than to the underlying disorder, whereas less acute signs, such as mannerisms, stereotypies and speech abnormalities, appear to be more specific to the underlying disorder than to the catatonia. Hence, the more specific features are given greater significance when making a diagnosis of catatonia, and it is these specific features that generally dictate whether separate treatment, in addition to or, in some cases, instead of, the standard treatment for the underlying disorder is needed.

Declaration of interest

None.

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MCQs

1 The following statements are correct:

- a catatonia was a concept introduced by Kraepelin
- b catatonic excitement is usually influenced by external stimuli
- c *Mitgehen* is a form of negativism
- d *Gegenhalten* is also known as catalepsy
- e abruptly withdrawing clozapine carries with it the risk of precipitating catatonia.

2 The following have been associated with catatonia:

- a GABA deficiency
- b glutamate deficiency
- c unilateral abnormalities in metabolism in the thalamus
- d dopamine overactivity
- e serotonin deficiency.

3 In the differential diagnosis of catatonia:

- a catatonia associated with mood disorders is less common than catatonia associated with schizophrenia
- b a functional or organic cause is almost always identified
- c increasing age is a risk factor for catatonic depression
- d deep-vein thrombosis is a recognised cause of catatonia
- e idiopathic catatonia is more common in males.

4 In the classification of catatonia:

- a catatonic schizophrenia is an ICD-10 diagnostic category
- b catatonic depression is a DSM-IV diagnostic category
- c there is a gender difference in age at onset in periodic catatonia
- d the susceptibility gene for periodic catatonia is located on chromosome 16
- e autistic catatonia is associated with increased cerebellar size.

5 Regarding the treatment of catatonia:

- a clinically significant improvement typically begins to occur about 24h after starting benzodiazepines
- b ECT is indicated in both functional and organic catatonia
- c carbamazepine is contraindicated in the acute catatonic phase
- d NMDA agonists may have a role
- e antipsychotics are a first-line treatment.

MCQ answers

1	2	3	4	5
a F	a T	a F	a T	a F
b F	b F	b F	b F	b T
c F	c F	c T	c F	c F
d F	d F	d F	d F	d F
e T	e F	e F	e F	e F