Catatonia in the emergency department

Walter Jaimes-Albornoz,[1,2] Jordi Serra-Mestres[2]

[1]Department of Psychiatry, Complejo Hospitalario de Navarra, Pamplona, Spain
[2]Central and North West London NHS Foundation Trust, London, UK

Correspondence to
Dr Jordi Serra-Mestres, Woodland Centre, Hillingdon Hospital, Central & North West London NHS Foundation Trust, Uxbridge, Middlesex UB8 3NN, UK;
jordi.serra-mestres@nhs.net

Accepted 10 January 2012
Published Online First 2 March 2012

ABSTRACT

Disturbances of the level of awareness are a frequent motive of attendance to emergency departments where the initial assessment and management will determine the direction of their outcome. The syndrome of catatonia must be taken into consideration and although it is normally associated with psychiatric diagnoses, it is also very often found in a great variety of neurological and medical conditions. Due to the clinical complexity of catatonia, there are still difficulties in its correct identification and initial management, something that leads to diagnostic delays and increased morbidity and mortality. In this article, a review of the literature on catatonia is presented with the aim of assisting emergency department doctors (and clinicians assessing patients in emergency situations) in considering this condition in the differential diagnosis of stupor due to its high frequency of association with organic pathology.

INTRODUCTION

Catatonia is a neuropsychiatric syndrome characterised by psychomotor abnormalities that is observed in a variety of medical, neurological and psychiatric disorders. The concept was introduced by Karl Ludwig Kahlbaum in 1874 who described the presence of stupor, mutism, rigidity, negativism, catalepsy and echophenomena in a group of patients with depression, mania, epilepsy, neurosyphilis and tuberculosis.[1] Catatonia was later incorporated into the nosological entity of schizophrenia, first by Kraepelin and later by Bleuler, although both described catatonia in various organic disorders. However, this observation did not seem to transcend into a wider understanding of catatonia.[2] From its original description, reports of catatonia associated with organic disease were abundant but this did not succeed in removing catatonia from its association with schizophrenia,[2] and this is maintained to date in the various disease classification systems. More modernly, the publication of clinical series of cases of catatonia with an organic aetiology has been frequent, and its prevalence has been reported to range from 7% to 45% in various clinical settings (20%–25% in psychiatric units).[3–7] In 1991, it was proposed that catatonia be considered a distinct diagnostic category[8] and in the Diagnostic and Statistical Manual-IV (DSM-iV), catatonia appears as an entity secondary to a general medical condition for the first time,[9] although its phenomena are not clearly described and their severity not considered.

It has been postulated that the problem relating to the classification of catatonia is one of the reasons why it remains relatively unrecognised by both psychiatrists and other medical practitioners.[10] This causes diagnostic delays that result in an inadequate management and important morbidity and mortality.[11 12]

Given the frequent acute presentation of catatonia in which stupor is a prominent feature, it is often the case that patients with this condition first come into contact with medical practitioners in emergency departments where catatonia is seldom identified or managed adequately and where the presentation is often attributed to psychological problems.[13–15]

METHODS

A literature search of MEDLINE/PubMed was undertaken, ranging from its first registers to July 2011. The following key words were cross-referenced: 'catatonia' and/or 'catatonic syndrome' with 'organic', 'complications', 'emergency' and 'treatment'. Additionally, a manual revision of the literature referred to in the identified articles was undertaken in order to obtain old or non-indexed references. All articles were considered for inclusion if their content was felt to be relevant to this review.

CLINICAL FEATURES

Catatonia usually presents acutely although some forms adopt a chronic course and is characterised by motor, behavioural, affective and autonomic signs. Around 40 signs of catatonia have been described[16] and they can be grouped in two clinical forms: a retarded-stuporous variety, which is the most common, and an excited one. The former presents with stupor, mutism, rigidity, immobility, negativism, catalepsy (and waxy flexibility or rigidity), posturing and echophenomena. Excitement, aggressivity and impulsivity are the main hallmarks of the excited form of catatonia. These two clinical forms may occur in the same patient during the same episode. The underlying cause does not seem to determine the form of presentation but it does seem to determine its prognosis. For example, among the catatonia secondary to medical causes, structural brain damage is associated with a poorer prognosis than other organic disorders.[11 17]

Catatonia can also adopt a very severe form called lethal or malignant when it is associated with hyperthermia, tachycardia, tachypnoea, hypertension or labile blood pressure, diaphoresis and alternating excitement and stupor. This form of catatonia is life threatening,[18] and though not very frequent, it is secondary to a variety of general medical or psychiatric causes, or to drugs and toxic substances,[19] especially antidopaminergic drugs (neuroleptic malignant syndrome)[19] and serotonergic compounds (serotonergic syndrome where, in addition, abdominal pain and diarrhoea, myoclonus, ataxia, tremor and convulsions are noted).[20]

Most patients in a catatonic state, although often demonstrating a flattened affective state,

experience an intense emotional state with high levels of anxiety and fear sometimes thinking they are dead or about to die.[21] They often do not recognise the motor abnormalities affecting them, something known as motor or postural anosognosia.[22]

The assessment of catatonia is undertaken by a clinical interview, observation and direct examination to elicit its signs.[17 23 24] In table 1, there is a description of the main catatonic signs, together with an indication of how to assess them.

It is important to consider that catatonia is associated with a number of medical complications if not treated and managed adequately, and that some of these complications are serious and life threatening. Pulmonary thromboembolism is a frequent cause of death in this patient group.[11 12] Box 1 shows the most frequent medical complications of catatonia.

PATHOPHYSIOLOGY

Given the fact that catatonia has multiple aetiologies, a unique pathophysiological mechanism to explain the whole syndromic complex is lacking. In relation to catatonia secondary to brain disorders, diffuse lesions or dysfunction seem to be more frequently associated with catatonia than localised lesions. This, together with the fact that catatonia occurs in primary psychiatric disorders where no specific brain lesion has been identified, suggests that catatonia is the result of dysregulation of specific neural pathways affecting motor function rather than the result of a single local lesion.[24] These neurochemical pathways relate to circuitry linking the medial frontal and inferior orbital cortices to the basal ganglia and thalami, with connections to parietal lobes, cerebellum and limbic system,[17 22 24] mainly in the right hemisphere.

It has been proposed that the initial neurochemical abnormality would take place in the gamma-aminobutyric acid-A (GABA-A) receptors in cortical areas of the frontal lobes that would cause hypoactivity of this inhibitory neurotransmitter system. In turn, this would cause hypoactivity of dopaminergic transmission in subcortical areas. This top-down or cortico–subcortical dopaminergic dysfunction would cause catatonia and also its malignant form. The neuroleptic malignant

Table 1 Catatonic semiology and its evaluation*

Sign	Definition	Mode of evaluation
Excitement	Extreme and constant hyperactivity without aim	Observation
Immobility and stupor	Extreme hypoactivity, complete immobility, minimal response to stimuli	Observation Stimulation
Mutism	Minimal or absent verbal response	Observation Conversation
Fixation of gaze	Fixed gaze with little or absent response to environment and reduced blinking	Observation
Posturing	Maintenance of a mundane or bizarre posture for long periods of time, even when uncomfortable	Observation
Catalepsy	Same as above but patient is positioned by the examiner	Placing arm, leg or body of patient in a particular position
Grimacing	Production and maintenance of bizarre facial expressions	Observation
Echopraxia and echolalia	Imitation of the movements or speech of a third party or of the examiner	Observation Conversation Examiner scratches own head in an exaggerated manner
Stereotypies	Aimless and repetitive motor activity (the abnormality is not inherent to the act but to its frequency)	Observation
Mannerisms	Directed motor activity undertaken in a strange or exaggerated form (eg, jumping, tiptoeing, waving at passersby)	Observation
Verbigeration	Production and repetition of sentences or words	Observation Conversation
Rigidity	Maintenance of a rigid posture despite attempts to being moved (exclude if tremor or cogwheeling exist)	Examination of muscle tone
Negativism	Unmotivated resistance to instructions or attempts to move or examine the patient, or conduct opposite to that required	Examination of muscle tone Verbal instructions
Waxy flexibility (*flexibilitas cerea*) or waxy rigidity	Initial resistance to passive movement of a limb followed by a facilitation of the movement (similar to the feeling of folding a hot candle)	Examination of muscle tone
Withdrawal	Refusal to eat, drink or to sustain others' gaze	Observation Feeding
Impulsivity	The patient suddenly behaves inappropriately without reason (undressing, running down the corridor, shouting). After the event, unable to explain his or her behaviour	Observation
Automatic obedience	Exaggerated cooperation with the examiner's requests, or repetition of movements or actions that were only required once	Examiner puts hand in pocket while saying to patient: 'stick out your tongue; I am going to prick it with a pin'
Passive obedience (Mitgehen)	Raising of the arm in response to a slight pressure by the examiner's finger despite the instruction not to raise it	Ask patient to extend the arm, place finger under patient's palm and try to raise the arm by gentle pressure while giving the instruction: 'Don't raise your arm'
Gegenhalten	Resistance to passive movement that is proportional to the strength of the stimulus	Passive mobilisation of a limb
Ambitendency	The patient seems to get stuck in indecisive conduct or movements	Observation Give hand to patient as intending to shake his or her hand while instructing: 'don't shake my hand'
Grasping	Grasping reflex that occurs when the patient's palm is stimulated	Examination of the grasping reflex
Perseveration	Repetitive returning to the same topic or movement	Observation Conversation Giving instructions
Combativeness	Unmotivated, undirected and unexplained combative behaviour	Observation
Autonomic abnormalities	In temperature, blood pressure, pulse, respiratory frequency; also, inappropriate perspiration Incontinence (urine/faecal)	Observation Checking of vital signs

*Based on the Bush–Francis catatonia rating scale.[23]

Box 1 Medical complications of catatonia

Cardiac and respiratory
Myocardial infarction, cardiac or respiratory arrest, aspiration, pneumonia, pneumonitis, pulmonary thromboembolism

Gastrointestinal, endocrine and electrolytical
Haemorrhage, dehydration, hypernatraemia, hyponatraemia, malnutrition, chachexis, liver damage, hypoglycaemia

Vascular
Thrombophlebitis, deep venous thrombosis, disseminated intravascular coagulation

Neurological and muscular
Muscle contractures, rhabdomyolisis, neuropathies secondary to posture, convulsions

Renal and urinary
Renal failure, urinary retention, urinary incontinence, bacteriuria, urinary tract infection

Other
Sepsis, oral candidiasis, skin infections, pressure ulcers, burns

syndrome would be caused by subcortical dopaminergic dysregulation (related to dopamine blockade) that would then cause a bottom-up or subcortico–cortical dysfunction in GABA-A transmission in the frontal cortex.[22] Autonomic symptoms in catatonia would be the result of similar GABA-A/dopamine dysregulation in fronto-hypothalamic circuits.[24]

The good response of catatonia to benzodiazepines (positive modulators of the benzodiazepine/GABA receptor)[25] and to electroconvulsive therapy (ECT) (that increases brain concentrations of GABA)[26] is very suggestive of the primary involvement of the GABAergic system in the genesis of catatonia. Glutamate would also have a role in its pathophysiology, being a biological antagonist of GABA, and given the fact that drugs that reduce glutamatergic transmission such as memantine and amantadine have been shown to improve catatonia.[27] Finally, it has been reported that serotonergic and cholinergic hyperactivity could also be involved in the production of catatonic phenomena,[22] but the role of these neurotransmitters is less well understood.

Catatonia has also been conceptualised as an evolutionary response to extreme fear (including that induced by psychopathological phenomena such as hallucinations and delusions) that would cause freezing of the individual to protect himself or herself from predators stimulated by movement.[21] This freezing would be analogous to immobility, catalepsy, stupor and mutism. The catatonic excitement would be analogous to the flight–fight response mediated by the sympathetic nervous system that precedes or follows the freezing.

DIAGNOSIS

There is no consensus on the number of clinical signs needed to diagnose catatonia, although most authors tend to agree that between one and four signs are necessary.[6 9 17 23 24] In a study of catatonia in patients with psychotic symptoms, it was found that with three or more signs a sensitivity and specificity of 99% was reached.[28] The optimal cut-off point could vary according to the clinical setting; in community cases where the prevalence of catatonia is lower, four signs would confer reasonable diagnostic confidence while in inpatient cases or in those in emergency departments, two signs would suffice.[28] The majority of authors agree that two or more signs of catatonia present during 1 h or more, or that can be reproduced in two or more occasions, are sufficient to diagnose catatonia.[17 24]

In any clinical situation, the presence of one catatonic sign should raise the suspicion of catatonia and a full clinical assessment should be undertaken, preferably using a standardised rating scale such as the Bush–Francis Catatonia Rating Scale that also measures the severity of the signs.[23] The use of such scales improves the detection and diagnosis of catatonia. The Bush–Francis Catatonia Rating Scale has also been shown to be sensitive to changes in the severity of catatonia during treatment.[29] Thus, the diagnosis of catatonia is twofold: first syndromic and second aetiological.

As previously mentioned, catatonia has been observed in a wide variety of general medical, neurological and psychiatric disorders as well as associated with drugs and toxic substances[2 17 24 30 31] (box 2). When the patient is initially seen at the emergency department, apart from obtaining a clinical history and details on the patient's past medical, psychiatric and drug/toxic history, a full physical and neurological examination will be needed, as well as a phenomenological description of the clinical signs of catatonia. Since the patient will be, more often than not, unable to assist clinical staff with the above, the interview with relatives, friends or carers of the patient will be of great importance to establish the history and chronology of events and symptoms.

In relation to laboratory tests, it is important to undertake a wide haematological and biochemical screening comprising a full blood count, markers of inflammatory processes such as C reactive protein, glucose, urea and electrolytes, liver function tests, proteins, calcium, thyroid function, and creatinin-phosphokinase and any other test that may be necessary according to the findings of the examination.[17 32 33]

There are no specific abnormalities related to catatonia, but in its malignant form and in the neuroleptic malignant syndrome, raised leucocytes and creatinin-phosphokinase and low serum iron have been often described.[24] In simple catatonia, leukocytosis is generally not seen.[24]

A blood or urine drug/toxic screen will often be needed. In addition, and when neurological disease is suspected, a test of structural neuroimaging such as a CT or MRI scan will be indicated. An EEG is also paramount in the investigation of unresponsive patients, helping with the diagnosis of seizure activity, particularly nonconvulsive status epilepticus[34] that can present with ictal catatonia. It is also of assistance in detecting encephalopathic states and in distinguishing normal wakefulness from sleep.

It is important that clinicians distinguish between medical causes of catatonia and medical complications of catatonia and, hence, that they rule out an organic cause and that they detect possible complications as soon as possible.

MANAGEMENT

Once identified, the treatment of catatonia can be rapidly and dramatically effective, with a complete resolution of clinical signs in 60%–80% of acute cases.[25 35] Due to the clinical signs of acute catatonia, patients are often unable to provide informed consent to the proposed treatment, and so clinicians will need to assess their mental capacity and to undertake a best interests assessment as the consequences of untreated catatonia may be very serious indeed.

Box 2 Aetiologies of catatonia

Neurological
- Bilateral infarction of the parietal lobes, temporal infarcts, thalamic lesions, bilateral lesions in globus pallidus
- Anterior cerebral and anterior communicating artery aneurysms and haemorrhagic infarcts, subdural haematoma
- Hydrocephalus
- Frontal lobe traumatic contusions and neoplasms, paraneoplastic encephalopathy and malignant and benign central nervous system (CNS) tumours
- Encephalitis (including herpes, HIV, postimmunisation and encephalitis lethargica), meningitis and cerebral abscesses
- Postencephalitic states, especially with Parkinsonism, progressive multifocal encephalopathy
- Neurosyphilis, other CNS infections: typhoid fever, tuberculosis, borreliosis, malaria, trypanosomiasis, hidatidosis
- Parkinson's disease and Lewy body disease
- Frontotemporal dementia, Alzheimer's disease, vascular dementia, Creutzfelt–Jakob disease, fatal familial insomnia
- Motor neuron disease, Wilson's disease, Huntington's disease, multiple sclerosis
- Epilepsy (absence seizures, complex partial seizures, generalised and complex partial (focal) status epilepticus, postictal states)
- Brain trauma acute and sequelae, Wernicke's encephalopathy, hepatic encephalopathy, central pontine myelinolisis
- Narcolepsy, Tay–Sachs disease, tuberous sclerosis

Metabolic and endocrine, haematological and immune
- Diabetic ketoacidosis, hypercalcaemia, renal failure, liver failure
- Acute intermittent porphyria, homocystinuria, membranous glomerulonephritis, hyponatraemia and hypernatraemia
- Lysosomal disease, hypothyroidism, hyperthyroidism, hyperparathyroidism, hypoglycaemia, Sheehan's syndrome
- Addison's disease, Cushing's disease, syndrome of inappropriate antidiuretic hormone secretion
- Vitamin B_{12} deficiency, nicotinic acid deficiency, pellagra
- Systemic lupus erythematosus, Paediatric Autoimmune Neuropsychiatric Disorder Associated with Streptoccocus (PANDAS), antiphospholipid syndrome, renal and hepatic transplant, Langerhans carcinoma

Pharmacological, toxic and other
- Typical and atypical antipsychotics (use and withdrawal) including clozapine, levodopa, amantadine, serotonergic drugs, Selective Serotonin Re-uptake Inhibitors ((SSRIs) trazodone, venlafaxine, etc), lithium
- Cephalosporines, ciprofloxacin, levofloxacin, azitromicine, levetiracetam, sodium valproate, gabapentin
- Disulfiram, paracetamol, aspirin, tramadol, hydroxicine, antitretrovirals, Adrenocorticotropic Hormone (ACTH), steroids
- Ciclosporin, chlorphenamine, methylphenidate, morphine, methadone, meperidene, allopurinol
- Benzodiazepines, cocaine, cannabis, lysergic acid diethylamide (LSD), mescalin, ketamine, phenylcyclidine, amphetamines, organophosphates, ethylene, carbon monoxide, severe burns

Psychiatric and neurodevelopmental
- Mania and depression (bipolar disorder), unipolar depression, late-onset depression, schizophrenia and chronic psychoses
- Anxiety disorder, dissociative disorder and Ganser syndrome, adjustment disorders, acute stress reactions, obsessive-compulsive disorder, Prader–Willi syndrome, autistic spectrum disorders

The aetiology, pattern and severity of catatonic signs do not affect response to treatment.[11] The treatment of catatonia should be provided in a hospital setting by a multidisciplinary team and with psychiatric support.[32 33] Benzodiazepines, and especially lorazepam, are the drugs of choice for the acute management of catatonia, and they are widely used for this purpose[17] given their mechanism of action through the GABA-A receptor. There is plenty of literature on the effectiveness of lorazepam in catatonia of various aetiologies, mainly case reports, series and open label studies,[27 36 37] but to date there is no evidence from randomised controlled trials of its use in catatonia secondary to schizophrenia.[38] In this illness, there is controversy on the role of antipsychotic drugs in the management of catatonia, with some authors suggesting that they worsen the latter (except for clozapine)[37] while others reporting that they can be beneficial.[27 39] It would appear that antipsychotics with strong D2 receptor antagonism should be avoided in patients with psychosis and catatonia.[37] There are also reports of other drugs being helpful and effective, sometimes in refractory cases, such as zolpidem, amantadine, memantine, bromocriptine, levodopa, sodium valproate, carbamazepine and topiramate, but they are usually slower than lorazepam in their action.[27] For patients unresponsive to lorazepam and who are in a serious clinical condition, ECT can be very beneficial.[17 40]

It is also very important that in addition to treating the signs of catatonia the treatment of the cause of the catatonia be started as soon as possible in order to ensure a complete resolution of the syndrome and to avoid early recurrences.

A lorazepam trial is the treatment of choice in catatonia,[17 24 25 27 32] especially in the retarded or stuporous forms.[32] It should start with the administration of lorazepam 1–2 mg intramuscular or orally. After 3 h, and if there is no response or if this is partial, lorazepam 1–2 mg intramuscular or orally are administered. This dose can be repeated after another 3 h.[32] An initial positive response usually predicts a more sustained response with further doses of treatment[25] and would confirm the diagnosis of catatonia in 80% of cases.[41] The final effective dose of lorazepam that achieved a complete resolution of the catatonic signs will then be maintained for some days until the treatment of the cause of catatonia is well under way.[32] The dose should be titrated against sedation.[32] Lorazepam should be switched to oral administration as soon as possible. A premature

suspension of treatment with lorazepam is likely to provoke a recurrence of the symptoms.[6 32]

In patients with lethal or malignant catatonia, the dose of lorazepam will be higher and ECT may be needed much earlier.[17]

The initial and subsequent doses of lorazepam may need to be lower in elderly patients[32] or in those with obesity or known respiratory disease.

ECT is considered in those cases in which response to an adequate trial of treatment with lorazepam is not achieved after 5 days or in those with lethal catatonia.[17] In the UK, the National Institute of Clinical Excellence includes catatonia as one of the few indications for ECT in its technology appraisal of this treatment modality.[42] ECT can only be administered by trained psychiatrists with anaesthetic support in accredited services.

During the treatment of catatonia, it is important to ensure that the patient is well hydrated and nursed and that adequate prevention of poor nutrition, infection and thrombosis is given. With improvement, the risk of falls secondary to sedation, especially in older patients, will need to be managed appropriately.

CONCLUSION

Catatonia is a neuropsychiatric syndrome associated with a wide variety of general medical, neurological and primary psychiatric conditions. It is mainly characterised by stupor, mutism, catalepsy, posturing and immobility. It is not infrequent and its presence does not automatically imply a psychiatric cause. The clinical presentation is easily identifiable by the knowledgeable clinician through observation, clinical examination and interview. Most patients with acute catatonia will present to accident and emergency departments where it is crucial that catatonia be diagnosed and where the work up to ascertain an aetiological diagnosis be started. Delays in diagnosis and initial management are associated with increased morbidity and complications, especially when catatonia is wrongly attributed to psychiatric disease.

Given that there is no consistent pattern in the semiology of catatonia to distinguish medical from psychiatric causes, the advice given by Gelenberg to consider medical aetiologies in all patients presenting with acute catatonia regardless of any psychiatric diagnosis still stands.[2]

Treatment with lorazepam is usually effective and improves the patient's clinical condition rapidly in a high proportion of cases.

Catatonia should be considered in the differential diagnosis of altered states of awareness in the emergency department.

Contributors WJA jointly designed the study, reviewed, read and analysed the relevant literature, and jointly wrote the manuscript. JSM jointly designed the study, read and analysed relevant literature, and jointly wrote the manuscript.

Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1. **Kahlbaum KL.** *Catatonia*. Baltimore: John Hopkins University Press, 1973.
2. **Fink M,** Shorter E, Taylor MA. Catatonia is not schizophrenia: Kraepelin's error and the need to recognize catatonia as an independent syndrome in medical nomenclature. *Schizophr Bull* 2010;**36**:314–20.
3. **Gelenberg AJ.** The catatonic syndrome. *Lancet* 1976;**1**:1339–41.
4. **Huang TL,** Ree SC, Huang YC, *et al*. Catatonic features: differential diagnosis and treatments at an emergency unit. *Psychiatry Clin Neurosci* 1999;**53**:63–6.
5. **Barnes MP,** Saunders M, Walls TJ, *et al*. The syndrome of Karl Ludwig Kahlbaum. *J Neurol Neurosurg Psychiatry* 1986;**49**:991–6.
6. **Rosebush PI,** Hildebrand AM, Furlong BG, *et al*. Catatonic syndrome in a general psychiatric inpatient population: frequency, clinical presentation, and response to lorazepam. *J Clin Psychiatry* 1990;**51**:357–62.
7. **Carroll BT,** Anfinson TJ, Kennedy JC, *et al*. Catatonic disorder due to general medical condition. *J Neuropsychiatry Clin Neurosci* 1994;**6**:122–33.
8. **Fink M,** Taylor AM. Catatonia: a separate category for DSM-IV? *Integr Psychiatry* 1991;**7**:2–10.
9. **American Psychiatric Association.** *Diagnostic and Statistical Manual of Mental Disorders*. 4th edn. Washington: American Psychiatric Association, 1994.
10. **Van der Heijden FM,** Tuinier S, Arts NJ, *et al*. Catatonia: disappeared or underdiagnosed? *Psychopathology* 2005;**38**:3–8.
11. **Levenson J.** Medical aspects of catatonia. *Prim psychiatry* 2009;**16**:23–6.
12. **Gross AF,** Smith FA, Stern TA. Dread complications of catatonia: a case discussion and review of the literature. *Prim Care Companion J Clin Psychiatry* 2008;**10**:153–5.
13. **Seethalakshmi R,** Dhavale S, Suggu K, *et al*. Catatonic syndrome: importance of detection and treatment with lorazepam. *Ann Clin Psychiatry* 2008;**20**:5–8.
14. **Valenstein M,** Maltbie A, Kaplan P. Catatonia in the emergency department. *Ann Emerg Med* 1985;**14**:359–61.
15. **Swartz C,** Galang RL. Adverse outcome with delay in identification of catatonia in elderly patients. *Am J Geriatr Psychiatry* 2001;**9**:78–80.
16. **Rogers D.** *Motor Disorder in Psychiatry: Towards a Neurological Psychiatry*. Chichester: John Wiley & Sons, 1992.
17. **Fink M,** Taylor AM. *Catatonia: A Clinician's Guide to Diagnosis and Treatment*. Cambridge: Cambridge University Press, 2003.
18. **Mann SC,** Caroff SN, Bleier HR, *et al*. Lethal catatonia. *Am J Psychiatry* 1986;**143**:1374–81.
19. **Strawn JR,** Keck PE Jr, Caroff SN. Neuroleptic malignant syndrome. *Am J Psychiatry* 2007;**164**:870–6.
20. **Ables AZ,** Nagubilli R. Prevention, recognition, and management of serotonin syndrome. *Am Fam Physician* 2010;**81**:1139–42.
21. **Moskowitz AK.** "Scared stiff": catatonia as an evolutionary-based fear response. *Psychol Rev* 2004;**111**:984–1002.
22. **Northoff G.** What catatonia can tell us about "top-down modulation": a neuropsychiatric hypothesis. *Behav Brain Sci* 2002;**25**:555–77; discussion 578–604.
23. **Bush G,** Fink M, Petrides G, *et al*. Catatonia I: rating scale and standardized examination. *Acta Psychiatr Scand* 1996;**93**:129–36.
24. **Caroff SN,** Mann SC, Francis A, *et al*. *Catatonia: From Psychopathology to Neurobiology*. Washington: American Psychiatric Publishing, 2004.
25. **Bush G,** Fink M, Petrides G, *et al*. Catatonia II: treatment with lorazepam and electroconvulsive therapy. *Acta Psychiatr Scand* 1996;**93**:137–43.
26. **Sanacora G,** Mason GF, Rothman DL, *et al*. Increased cortical GABA concentrations in depressed patients receiving ECT. *Am J Psychiatry* 2003;**160**:577–9.
27. **Carroll B,** Lee JW, Appiani F, *et al*. The pharmacotherapy of catatonia. *Prim Psychiatry* 2010;**17**:41–7.
28. **Peralta V,** Cuesta MJ. Motor features in psychotic disorders II. *Schizophr Res* 2001;**47**:117–26.
29. **Sienaert P,** Rooseleer J, De Fruyt J. Measuring catatonia: a systematic review of rating scales. *J Affect Disord* 2011;**135**:1–9.
30. **Ahuja N.** Organic catatonia: a review. *Indian J Psychiatry* 2000;**42**:327–46.
31. **Duggal HS,** Singh I. Drug-induced catatonia. *Drugs Today (Barc)* 2005;**41**:599–607.
32. **Rosebush PI,** Mazurek MF. Catatonia and its treatment. *Schizophr Bull* 2010;**36**:239–42.
33. **Lahutte B,** Cornic F, Bonnot O, *et al*. Multidisciplinary approach of organic catatonia in children and adolescents may improve treatment decision making. *Prog Neuropsychopharmacol Biol Psychiatry* 2008;**32**:1393–8.
34. **Kaplan PW.** Nonconvulsive status epilepticus in the emergency room. *Epilepsia* 1996;**37**:643–50.
35. **Daniels J.** Catatonia: clinical aspects and neurobiological correlates. *J Neuropsychiatry Clin Neurosci* 2009;**21**:371–80.
36. **Ungvari GS,** Leung CM, Wong MK, *et al*. Benzodiazepines in the treatment of catatonic syndrome. *Acta Psychiatr Scand* 1994;**89**:285–8.
37. **England ML,** Ongür D, Konopaske GT, *et al*. Catatonia in psychotic patients: clinical features and treatment response. *J Neuropsychiatry Clin Neurosci* 2011;**23**:223–6.
38. **Gibson RC,** Walcott G. Benzodiazepines for catatonia in people with schizophrenia and other serious mental illnesses. *Cochrane Database Syst Rev* 2008;(4):CD006570.
39. **Peralta V,** Campos MS, de Jalon EG, *et al*. DSM-IV catatonia signs and criteria in first-episode, drug-naive, psychotic patients: psychometric validity and response to antipsychotic medication. *Schizophr Res* 2010;**118**:168–75.
40. **Zisselman MH,** Jaffe RL. ECT in the treatment of a patient with catatonia: consent and complications. *Am J Psychiatry* 2010;**167**:127–32.
41. **Fink M,** Taylor AM. Catatonia: subtype or syndrome in DSM? *Am J Psychiatry* 2006;**163**:1875–6.
42. **National Institute of Clinical Excellence.** Technology Appraisal No 59: The clinical effectiveness and cost effectiveness of electroconvulsive therapy (ECT) for depressive illness, schizophrenia, catatonia and mania. http://www.nice.org.uk/TA059

Catatonia in the emergency department

Walter Jaimes-Albornoz and Jordi Serra-Mestres

Emerg Med J 2012 29: 863-867 originally published online March 2, 2012
doi: 10.1136/emermed-2011-200896

Notes