The Catatonia Syndrome

Forgotten but Not Gone

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Catatonia is a motor dysregulation syndrome among psychiatric asylum patients that was delineated in 1874. The syndrome was so well characterized that within a few years its prevalence among psychiatric populations was reported from 6% to 38%, averaging 15% of hospitalized patients in the years since.[1] In the mid–20th century, as psychiatric practice shifted from the asylum to the ambulatory clinic with an emphasis on psychotherapy and the prescription of psychotropic agents, the role of the medical examination was degraded and the recognition of catatonia languished.[2] The perceived tight bond between catatonia and the diagnosis of schizophrenia led to the widespread assumption that catatonia is mainly a form of psychosis and is not appropriately classified elsewhere.[3]

Interest in catatonia among clinical neurologists waned with the recognition of catatonia limited to a classic retarded form with posturing, rigidity, staring, immobility, and mutism. A recent case report in the *New England Journal of Medicine* describes a young woman with well-defined catatonia that is neither recognized nor treated except as a passing mention in the case analysis.[4] An ovarian teratoma was removed and within 48 hours of the surgical removal under anesthesia the catatonia syndrome resolved. She was identified as having an anti–*N*-methyl-D-aspartate receptor (NMDAR) antibody–mediated paraneoplastic limbic encephalitis.

A concurrent review of 100 cases of anti-NMDAR encephalitis exquisitely describes the many signs of the catatonia syndrome in almost all the patients.[5] Yet, catatonia is not identified nor effective treatments offered. These experiences prompt this review of catatonia as a definable syndrome that appears in many guises and is eminently treatable.

A writer in 1981 asked: "Where have all the catatonics gone?" suggesting that the disappearance resulted from the increased use of psychotropic agents, preventing the more dire forms of schizophrenia.[6] Yet, studies in the 1970s reported catatonia to be prominent among patients outside schizophrenia. About 10% of the 2500 hospitalized patients at the University of Iowa in 1975 met criteria for catatonia at their index admissions. Among those reexamined at a later date, 40% had, at some point, recovered completely. These patients were not examples of schizophrenia.[7] In patients admitted to a psychiatric unit of a New York City hospital, 55 patients (about 20%) had 1 or more catatonic signs. Only 4 patients satisfied the research diagnostic criteria for schizophrenia, while more than two-thirds met the criteria for affective disorders, usually mania.[8]

The same year, 8 patients receiving high-potency neuroleptic drugs developed an acute syndrome of catatonia and parkinsonism.[9] A few years later, a review of more than 60 cases of neurotoxic responses to neuroleptic drugs described the signs of malignant catatonia using the criteria of hyperthermia, rigidity, altered consciousness, and autonomic instability. The author offered *neuroleptic malignant syndrome* as the name that was quickly adopted.[10]

The reports raised doubts about characterizing catatonia solely as a type of schizophrenia and encouraged interest in the syndrome's characteristics and prevalence in patients in nonpsychiatric environments. The broadened recognition frequently finds catatonia in emergency departments and in general medical and neurologic services.[1,11,12] These reports take on greater significance since clinical science has developed both a useful diagnostic test to verify the diagnosis and rapidly

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Table 1. Principal Features of Catatonia[1]

Feature	Description
Mutism	Verbal unresponsiveness, not always complete nor always associated with immobility.
Stupor	Altered arousal during which the patient fails to respond directly to queries (similar in presentation to the effects of dissociative anesthesia); when severe, the patient is mute and immobile and does not withdraw from painful stimuli.
Negativism (gegenhalten)	Resistance to the examiner's manipulations, whether light or vigorous, with strength equal to that applied, as if bound to the stimulus of the examiner's actions.
Posturing (catalepsy)	Maintaining postures for long periods. Includes facial postures, such as grimacing or schnauzkrampf (lips in an exaggerated pucker). Body postures, such as psychological pillow (patient lying in bed with his or her head elevated as if on a pillow), lying in a jackknifed position, sitting with upper and lower portions of the body twisted at right angles, holding arms above the head or raised in prayerlike manner, and holding fingers and hands in odd positions; prolonged mundane positions are common examples.
Waxy flexibility	The patient's initial resistance to an induced movement before gradually allowing himself or herself to be postured, similar to bending a candle.
Stereotypy	Non–goal-directed, repetitive motor behavior. The repetition of phrases and sentences in an automatic fashion, similar to a scratched record, termed *verbigeration*, is a verbal stereotypy. The neurologic term for similar speech is *palilalia*, during which the patient repeats the sentence just uttered, usually with increasing speed.
Automatic obedience	Despite instructions to the contrary, the patient permits the examiner's light pressure to move his or her limbs into a new position (posture), which may then be maintained by the patient despite instructions to the contrary.
Ambitendency	The patient appears "stuck" in an indecisive, hesitant movement, resulting from the examiner verbally contradicting his or her own strong nonverbal signal, such as offering his or her hand as if to shake hands while stating, "Don't shake my hand. I don't want you to shake it."
Echophenomena	Includes echolalia, in which the patient repeats the examiner's utterances, and echopraxia, in which the patient spontaneously copies the examiner's movements or is unable to refrain from copying the examiner's test movements, despite instruction to the contrary.
Mannerisms	Odd, purposeful movements, such as holding hands as if they were handguns, saluting passersby, or exaggerations or stilted caricatures of mundane movements; odd speech cadences and feigned accents are other examples.

effective treatments, even for the patients with the most severe forms of the syndrome. The experience prompts a change in clinical practice with a promise for better outcomes and a reassessment of the classification of the illness within the psychiatric classification system.

CATATONIA DEFINED

Catatonia is a motor dysregulation syndrome with patients unable to move normally despite full physical capacity. Movements cannot be initiated or stopped and become repetitive, posture is frozen or oddly positioned, and actions become contrary to intent (**Table 1**). Catatonia arises from many roots and is a syndrome analogous to delirium, another behavior syndrome with diverse etiology. The pattern of symptoms and signs defines the catatonia syndrome but is not specific as to the cause.

The main symptoms are mutism (failure to speak), negativism (motor and other behavioral resistance to following simple requests or commands), posturing and rigidity (abnormal body positions), persistent staring, repetitive movements (often self-injurious), automatic obedience (responding to tactile stimuli despite instruction to the contrary), and lack of response to pain. Stupor is a hallmark. Some features of catatonia are also seen in the motor dysregulation states of parkinsonism, compulsions, tics, and seizures.

Disorders in movement were well recognized in the neurology and asylum populations in the 19th century. Stupors, paralyses (eg, neurosyphilis and cerebrovascular disease), repetitive movements (eg, dystonia, dyskinesia, and tremor), and excitement states were among the mix of described conditions. The psychiatrist Karl Kahlbaum delineated catatonia by its motor and behavioral aberrations in a rich text of 26 clinical vignettes. Although all exhibited the signs that we identify as catatonia, 12 patients were depressed, 9 had a seizure disorder, 3 had neurosyphilis, and 2 had tuberculosis.[13]

Emil Kraepelin considered catatonia a core feature of his dementia praecox construct, but he also recognized its presence in manic-depressive illness.[14] He relied heavily on catatonia as a sign of dementia praecox. The psychopathologists Karl Kleist, Carl Wernicke, and Karl Leonhard recognized catatonia as a principal feature of both psychotic and mood-altered states.[15] Kraepelin's connection, however, was widely endorsed and today is accepted in both the American *DSM-IV*[16] and the *International Statistical Classification of Diseases, 10th Revision*.[17]

Kahlbaum described 17 motor signs, but other authors extended the list, some identifying 40 or more phenomena.[1,18-21] The current *DSM-IV* criteria offer a restricted list of signs, with 2 signs sufficient for the diagnosis. Most investigators rely on a greater number of signs and durations of several to 24 hours as necessary for the diagnosis.

Published rating scales facilitate the syndrome's recognition.[18-21] Interrater reliability individually and across instruments is good, with pairwise correlations between 0.9 and 0.96 for all. Several factor analytic and 1 cluster analytic study delineate patterns among the catatonic features, indicating that a syndrome exists.[1,22,23]

WHERE IS CATATONIA RECOGNIZED?

Surveys using standardized rating scales find 7% to 15% of acutely hospitalized psychiatric patients and psychiatric emergency department patients to exhibit the syndrome. Sadly, these patients go largely unrecognized.[1,24,25] In a large Dutch study, for example, while the clinicians identified catatonia in 2% of 139 inpatients, the research team found catatonia in 18%.[26]

Catatonia may begin acutely or develop insidiously and may become life threatening in severity. It appears as stupor and fever of unknown origin, "conversion disorder," "failure to cooperate," and acute neurotoxic syndromes in

general medical and neurology hospital services. It is frequent in patients with mood disorders, appearing as mania and depressive illness in adult and pediatric psychiatry services. Catatonia is common among chronically ill psychotic patients.[1]

The patients are often febrile and delirious, appearing as if they have an infectious encephalopathy.[27,28] The syndrome occurs in patients with systemic infections, such as typhoid fever.[29] Manic excitement, mutism, and repetitive acts complicate lupus erythematosus and other general medical disorders.[30] Deliria and stupors become severe, occasionally requiring intubation and supportive ventilation. Catatonia has recently been identified in patients with paraneoplastic syndromes in which NMDAR antibodies are implicated in the pathophysiology.[4,5,31,32] A history of seizure disorder and evidence of recent seizures is often associated with catatonia; abnormal electroencephalogram recordings are interpreted as evidence of nonconvulsive status epilepticus.[33-35] When catatonia goes unrecognized, fruitless extensive laboratory testing ensues and the illness leads to prolonged hospital care and death.[36]

In pediatric populations, catatonia is reported among those with mental retardation and autistic spectrum disorder.[12] In a large survey of persons with autism and mental retardation, 30 of 506 patients (6%) and 17% of those older than 15 years exhibited catatonia.[37] Self-injurious repetitive motor behaviors are particularly destructive in patients with severe autism but are afforded relief by treatments for catatonia.[38,39]

FORMS OF CATATONIA

A retarded form of catatonia, sometimes referred to as the *Kahlbaum syndrome*, is the most commonly recognized (**Table 2**). Movement is inhibited with posturing, rigidity, mutism, and repetitive actions. Failure to respond to painful stimuli is a feature. Stupors are the more severe form of inhibition and are often an expression of catatonia. Such patients require parenteral feeding and extended nursing care during these life-threatening states.

An excited form of catatonia is characterized as *delirious mania*, sometimes cited as Bell's mania.[40,41] It is marked by restless movements, talkativeness, agitation, and frenzy; the presence of disorientation and confusion is recognized as delirium. Catatonic features are prominent. A peculiar syndrome of confusion has been identified as *oneirophrenia*. These patients are in a clouded state akin to dissociative anesthesia and have other catatonic features.[42] The *Ganser syndrome* is another example.[1,43,44]

Malignant catatonia (also labeled *lethal* or *pernicious*) is a syndrome of acute onset, fever in all but elderly individuals, and abnormal blood pressures, tachycardia, and tachypnea of life-threatening dimensions. It was described well before the introduction of psychotropic agents.[45] It is similar in all respects to the *neuroleptic malignant syndrome* (NMS) and *neuroleptic-induced catatonia*. These syndromes respond well when treated as malignant catatonia.[1] The *toxic serotonin syndrome* is also viewed as malignant catatonia associated with serotonergic drug overdose.[46,47]

A recurrent form of *periodic catatonia* is reported among patients with manic depression, the patient fluctuating between stupor and excitement. This is most likely to occur during a mixed mood state or a period of rapid cycling.[48]

Table 2. The Catatonia Syndromes[1]

Form of Catatonia	Also Referred to As:
Retarded catatonia	
Benign stupor	Kahlbaum syndrome
Excited catatonia	Manic excitement
Delirious mania	Manic delirium
	Bell's mania
Oneiroid state	Onirisme
	Oneirophrenia
Malignant catatonia	Lethal catatonia
	Pernicious catatonia
	Acute fulminating psychosis
Neuroleptic malignant syndrome	Syndrom malin
	Neuroleptic-induced catatonia
Toxic serotonin syndrome	Serotonin syndrome
Periodic catatonia	
Mixed affective state	Rapid cycling mania

HOW IS CATATONIA RECOGNIZED?

Catatonia is a consideration in every patient with dysregulation of motor behavior, particularly those in whom changes in consciousness and mood are also present. These signs are commonly relieved by the intravenous administration of a barbiturate or a benzodiazepine. Intravenous lorazepam, at 1 or 2 mg, temporarily relieves mutism, posturing, staring, rigidity, and repetitive movements. Rapid relief is considered verification of the diagnosis of catatonia and is described as the *lorazepam test*.[1] Zolpidem is reported as an alternative to lorazepam.[49] Mutism and negativism are occasionally relieved when patients are given an anesthetic or sedative for surgery, a magnetic resonance imaging procedure, or to assist tube-feeding.[1,50]

Relief of catatonia with lorazepam was reported in 80% of patients in 1 study.[51] Similar resolution in 75% to 100% of patients was reported in a review of treatments for NMS.[52] The immediate response to benzodiazepines is a useful measure of the syndrome, with an incidence of predictive response that is similar to the use of a scalp-recorded clinical electroencephalogram to identify seizure disorders.[53,54] High serum creatine kinase and low serum iron levels are associated with malignant catatonia/NMS but are less useful in identifying other forms of catatonia.[1]

EFFECTIVE TREATMENTS FOR CATATONIA

Before the era of somatic treatments in psychiatry, the recovery of a catatonic patient was a mystery. Patients, often ill for months or even years, would suddenly be restored to normal behavior after a stressful emotional or traumatic experience or after a febrile episode or spontaneously.[1,13] High doses of amobarbital sodium, the first effective intervention for catatonia, were reported by Bleckwenn[55] in 1930. His films showing the disappearance of mutism, posturing, and rigidity prompted the wide introduction of amobarbital as a life-saving treatment for the syndrome.

Inducing grand mal seizures to relieve psychosis was reported by the Budapest psychiatrist Ladislas Meduna

in 1935.[56] He used camphor or pentylenetetrazol (Metrazol) to initiate seizures. Seeking patients with "schizophrenia," 9 of the first 11 patients he treated had catatonia.[57] His 1937 report prompted worldwide acceptance of convulsive treatment in psychiatric illnesses.[58]

Inducing seizures with electricity was a modification reported in 1938. Electroconvulsive therapy (ECT) was more facile in use, more assured in eliciting effective seizures, and soon widely adopted.[59] It quickly embraced catatonia as an indication for its use and is the definitive treatment for catatonia today.

The treatment algorithm for catatonia is based on severity, offering 2 tracks.[1,11,60] For all patients, potential toxic precipitants should be eliminated and general medical and specific neurologic causes of illness, treated. For patients with retarded catatonia and body temperatures less than 39°C, lorazepam is administered parenterally or orally beginning with 3 mg/d and increasing rapidly to effective resolution. Dosages of 20 to 30 mg/d are occasionally necessary.

For those with higher fevers, in delirium, or at physiologic risk, or who do not quickly respond to lorazepam, bilateral ECT is most effective. It may, however, require daily treatments for 2 to 5 days.[1] The efficacy of lorazepam remitting catatonia was 80% to 100% in 4 studies and ECT, 82% to 96% in 5 studies.[54]

Present treatment practices are not optimal. When catatonia is discerned in general medical and surgical services by psychiatric consultants, the association with schizophrenia and the presence of excitement and delirium compel the prescription of antipsychotic medications, often by parenteral routes and in high dosages. But such medications are reported as precipitating the malignant form of catatonia with severe morbidity and occasional mortality and should be avoided. Identifying catatonia as schizophrenia precludes barbiturates, benzodiazepines, or ECT, as these interventions are not considered in the treatment algorithms.

WHERE DO WE GO FROM HERE?

Patients with catatonia are frequently found in general medical and neurology services and greater attention to its diagnosis and treatment is warranted. Extra efforts at recognizing the syndrome in our teaching and in expanding the guides for differential diagnosis to include catatonia are needed. It is no longer tenable to describe catatonia only as a psychiatric disorder specific to schizophrenia. Catatonia is better considered a movement and behavior syndrome with specific attributes and diverse antecedents. It has the ubiquity of delirium, another behavior syndrome of defined characteristics and varied causes, and warrants a similar independent class in psychiatric classifications.[61] It is a discrete syndrome with clearly specified features, though it presents in many forms. The rapid relief afforded by intravenous barbiturate or benzodiazepine confirms the diagnosis. In contrast to the outcome of treatments for schizophrenia, treatments for catatonia are quickly effective, albeit requiring considerable adjustments in dosages.[1]

Little attention has been paid to the syndrome of catatonia in biological research. Genetic studies, based on the descriptive classification of catatonia by Leonhard, suggest that specific genes, notably *15q15,* are of interest but these studies have not been confirmed.[62,63] An association with catatonia in autism spectrum disorders has been described.[64] Similarly, the brain imaging studies that identify reductions in brain size and in identifiable brain nuclei in schizophrenic patients do not examine patients with the catatonia syndrome.[60,65] It would be useful for these powerful research tools to be applied to catatonic populations defined by catatonia rating scales and verified by a lorazepam test and treatment response. An interesting clue as to the pathophysiology of the catatonia syndrome comes from the association of anti-NMDAR antibodies with catatonia.[4,5,66] The efficacy of sedative anticonvulsants—barbiturates, benzodiazepines, and ECT—in relieving catatonia also encourages studies of the role of seizure thresholds.[1]

In the 1980s, increasing awareness of catatonia as a toxic response to neuroleptic agents led to the identification of NMS. The similarity to malignant hyperthermia suggested treatment trials with dantrolene sodium. The dopamine blockade hypothesis of the action of the neuroleptic drugs suggested treatment with dopamine agonists. Neither approach was useful. Recognition that NMS was indistinguishable from malignant catatonia led to treatment trials with benzodiazepines and ECT. These were successful and NMS is now acknowledged as a form of malignant catatonia with a specific precipitant.

The recognition that catatonia occurs in patients with paraneoplastic syndromes suggests that it would be useful to examine, test, and treat these patients for the catatonia syndrome. The potential upside is great in earlier recognition and effective treatment for catatonia with lesser risk and interference in the management of the neoplastic syndrome.

Increased recognition of catatonia in children and adolescents ill with autism and mental retardation led to treatments for catatonia. Amelioration of much of the disability confirmed the treatment's usefulness. Self-injurious behavior in these children is a repetitive, uncontrollable, damaging stereotypy. Assessing the behavior as catatonia led to treatment trials for catatonia with benefit.[41]

Catatonia has also been identified in patients with Gilles de la Tourette syndrome, epilepsy, and stupors and fevers of unknown origin. Reports of the relief afforded by treatment for catatonia suggest that detailed diagnostic and treatment trials are warranted. These examples of catatonia as an independent motor and behavior syndrome encourage broader queries as to its many faces in neurology, emergency department, and medical settings. Divorcing the catatonia syndrome from its marriage to schizophrenia promises better clinical care and outcomes as well as more homogeneous populations for genetic, brain imaging, and psychopathological studies. In the treatment of seizure disorders, anticonvulsants are prescribed and considered useful regardless of the cause, whether stroke, trauma, tumor, infection, or inheritance. Similarly, we envision the treatment of catatonia regardless of the underlying pathology. As with seizure disorders, the relief of catatonia may be followed by the treatments that are specific for the pathology that elicited catatonia.

The catatonia syndrome has not disappeared. It is well defined in both adult and pediatric acute medical, neurology, and emergency department services. It is eminently treatable. It warrants greater attention as a dis-

tinct syndrome akin to delirium; its recognition outside the construct of schizophrenia is encouraged.[67]

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