Catatonia 1: history and clinical features

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Catatonia is commonly encountered in psychiatric and medical practice but is under-recognized. It occurs in association with a wide range of disorders and drugs. Psychiatric education and textbooks mistakenly only consider catatonia as a subtype of schizophrenia. This article, the first of two, reviews the development of the concept of catatonia, its epidemiology, clinical features and pathophysiology.

In his original monograph, Karl Kahlbaum (1874) described a new neurological motor disorder that was accompanied by psychiatric symptoms. He described this syndrome in association with psychiatric and systemic disease, particularly affective disorder, epilepsy and tuberculosis (Barnes et al, 1986). He drew an analogy with general paresis, implying that the underlying cerebral lesion would be discovered in due course (Johnson, 1993). He specifically stated that it should not be considered merely as a subtype of the contemporary concept of 'degeneration' (Gelenberg, 1976), which was subsequently termed dementia praecox by Kraepelin and schizophrenia by Bleuler.

Despite this, the divergence of psychiatric and neurological practice that followed the work of Freud and Bleuler enshrined Kahlbaum's syndrome within the boundaries of schizophrenia. Catatonic symptoms and signs were interpreted according to the prevailing psychodynamic paradigm of the time — posturing and rigidity were ways of shutting out reality, lethal catatonia was an expression of the death wish (Rogers, 1991). Similarities between catatonia and disorders such as Wilson's disease were noted regularly but treated with disdain and occasionally ridicule by contemporary psychiatry (Rogers, 1992). In subsequent years catatonia was largely ignored, particularly in the American literature, because of its failure to lend itself easily to psychodynamic interpretation. With the advent of social psychiatry, motor symptoms were explained by the concept of institutionalization (Johnson, 1993), and when neuroleptics were introduced they became the universal culprit — all motor symptoms became extrapyramidal side-effects.

Only in the 1960s, with the re-emergence of a brain-based psychiatry, did widespread interest in catatonic symptoms as primary manifestations of disease processes return. In recent years there has been a proliferation of articles describing associations and treatment response as well as attempts to develop diagnostic tools. In their most recent classificatory schemes the World Health Organization (1992) and the American Psychiatric Association (1994) have finally admitted the presence of catatonia outside the boundaries of schizophrenia, although in both schizophrenia remains the primary cause. Similarly standard psychiatric textbooks only consider Kahlbaum's syndrome as a subtype of schizophrenia, ignoring the pleas of the man who described this syndrome so accurately over 100 years ago.

CLINICAL FEATURES

The syndrome of catatonia is defined by the objective presence of motor signs, over 40 of which have been described. There is no standardized list. In their review of the literature, Bush et al (1996) described 23 signs that had been considered catatonic. These are listed in *Table 1*. The symptoms occur against a background of often alternating periods of reduced movement or immobility and excitatory phases of hyperkinesis.

There is no agreed threshold for the number of symptoms or duration of symptoms that should be present to justify a diagnosis of catatonia. Research has suffered from this and studies can rarely be compared with confidence. The new *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) diagnosis of catatonic disorder because of a general medical condition may go some way toward redressing this,

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although as yet there is little published research using these criteria (*Table 2*) (American Psychiatric Association, 1994). The *International Classification of Diseases* (ICD-10; World Health Organization, 1992) has no equivalent category.

Catatonia presents with a spectrum of severity. Transient, isolated motor symptoms occur in many situations but would not be considered to represent a catatonic syndrome by most clinicians. When the motor syndrome is present, it may be termed 'simple catatonia' (Philbrick and Rummans, 1994). This differentiates it from the most severe form — malignant catatonia.

Malignant catatonia is a condition that has been recognized for over 150 years. The same syndrome has received an array of different names including Bell's mania, fatal catatonia, lethal catatonia and acute delirious mania (Mann et al, 1986). It is characterized by a catatonic syndrome in association with hyperthermia and/or signs of autonomic instability (tachycardia, fluctuating blood pressure, diaphoresis) (Philbrick and Rummans, 1994). It progresses to stupor, coma and eventual death.

Laboratory investigations typically, although not invariably, show raised creatine phosphokinase, raised leucocytes, and raised erythrocyte sedimentation rate. The electroencephalogram does not show consistent changes (Singerman and Raheja, 1994). The cause of death is most likely to be acute renal failure consequent upon rhabdomyolysis and myoglobinuria. It is usual for cases of malignant catatonia to be preceded by simple catatonia, implying that there is only a quantitative difference between them and emphasizing the importance of early and appropriate intervention. It is important to note that the neuroleptic malignant syndrome is a variant of malignant catatonia (as discussed in the second of these articles). Therefore antipsychotics are a cause not a treatment of the syndrome.

EPIDEMIOLOGY

There are no population studies evaluating rates of catatonic symptoms. Johnson (1993) considered that catatonia 'remains a common problem', despite the supposed decline in incidence since Bleuler's (1950) estimate that 50% of his patients with schizophrenia had at some time shown catatonic symptoms. This decline in incidence, which has been disputed by some (Rogers, 1991), may be because of improved diagnosis of underlying organic causes of catatonia, leading to reclassification. It is of note, however, that it coincided with the introduction of antipsychotic medication and the consequent recognition of extrapyramidal side-effects.

More recently, Bush et al (1996) prospectively screened a consecutive sample of 215 patients admitted to an academic psychiatric inpatient unit. They found that 15 (7%) patients had three or more catatonic symptoms (from items 1–14 in *Table 1*). Starkstein et al (1996) applied the modified Rogers scale (Lund et al, 1991) to a consecutive series of 79 depressed patients and found that sixteen (20%) had catatonia. Catatonic symptoms were associated with increased age, increased frequency of depressive episodes and more severe impairment of both cognitive function and daily living skills. The mean age of this sample was 54.2 years. Peralta et al (1997) studied 567 consecutive admissions to a psychiatric unit

TABLE 1.
Signs of catatonia

Catatonic sign	Description of sign
Excitement	Non-purposeful hyperactivity or motor unrest
Immobility or stupor	Extreme hypoactivity, reduced response to stimuli
Mutism	Reduced or absent speech
Staring	Fixed, non-reactive gaze, reduced blinking
Posturing	Spontaneous maintenance of posture (the posture itself may or may not be abnormal) for longer than is usual
Grimacing	Maintenance of odd facial expressions
Echolalia	Mimicking of examiner's speech (may be delayed)
Echopraxia	Mimicking of examiner's movements (may be delayed)
Stereotypy	Repetitive, non-goal directed movements
Mannerisms	Odd purposeful voluntary movements
Verbigeration	Repetition of meaningless phrases or sentences
Rigidity	Maintenance of position despite efforts to be moved
Negativism	Apparently motiveless resistance to instructions or attempts to make contact
Waxy flexibility	During reposturing there is initial resistance, then the new posture is maintained
Withdrawal	Refusal to eat, drink or make eye contact
Impulsivity	Sudden inappropriate behaviours with no explanation
Automatic obedience	Exaggerated cooperation with request or continuation of movement requested
Mitgehen	Raising of arm in response to light finger pressure (like an angle poise lamp) despite instructions to the contrary
Gegenhalten	Resistance to passive movement in proportion to strength of stimulus
Ambitendency	Indecisive, hesitant movement
Grasp reflex	Reflex grasping movement of hand in response to stroking palm
Perseveration	Repeatedly returns to same topic or persists with movement
Combativeness	Usually undirected aggression or violent behaviour

From Bush et al (1996)

with functional psychotic illnesses. They defined catatonic syndrome as the presence of at least two motor symptoms or one of severe intensity out of a list of seven. Using these criteria they found that 16.9% of their sample had catatonia.

PATHOPHYSIOLOGY

The pathophysiology of catatonia is poorly understood. Postmortem, brain imaging and biochemical studies are fairly sparse and have failed to reach consistent conclusions (Fink, 1997). The most effective treatments — benzodiazepines, barbiturates and electroconvulsive therapy — are anticonvulsant, acting via gamma-aminobutyric acid (GABA) receptors. On this basis, Fink (1997) suggests that GABAergic systems may be of importance. In malignant catatonia, Philbrick and Rummans (1994) suggest that hypodopaminergia leads to poikilothermia and this may produce hyperthermia via reduced facilitation of hypothalamic temperature reduction. Reduced dopaminergic nigrostriatal facilitation of GABA inhibition may cause rigidity.

Johnson (1993) has reviewed theories of the neural localization of catatonia. Fisher (1989) drew a parallel between akinetic mutism caused by lesions of the midbrain and catatonia. She suggested that interruption of the midbrain ascending pathways might account for both syndromes. Leentjens and Pepplinkhuizen (1998) compared catatonic symptoms and frontal lobe function and described a case of recurrent catatonia owing to frontal lobe seizures. Rogers (1991, 1992) emphasizes the similarities with Wilson's disease and postencephalitic parkinsonism, which were originally noted in the 1920s, and with extrapyramidal side-effects. He suggests that catatonia should be regarded as an extrapyramidal disorder. HM

TABLE 2.
Diagnostic criteria for catatonia disorder caused by a general medical condition

Presence of catatonia — at least one of the following	1	Motoric immobility
	2	Excessive purposeless motor activity
	3	Extreme negativism or mutism
	4	Peculiarities of voluntary movement
	5	Echolalia or echopraxia
Evidence that it is because of a general medical condition		
The disturbance is not better accounted for by another mental disorder		
Disturbance does not occur exclusively during the course of a delirium		

From American Psychiatric Association (1994)

KEY POINTS

- Catatonia is a neuropsychiatric syndrome that occurs commonly in both medical and psychiatric practice.
- It was originally described as a feature of affective disorders and organic illnesses, but subsequently was mistakenly considered to be purely a form of schizophrenia.
- The classical presentation is a triad of mutism, akinesis and waxy flexibility, but a large and unstandardized number of specific symptoms has been described.
- The spectrum of severity ranges from transient, isolated symptoms to a potentially life-threatening syndrome. Simple catatonia may progress to malignant catatonia which has a definite mortality.
- It is vital that catatonia is recognized and appropriately treated promptly.

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