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The many varieties of catatonia

Abstract About 10 % of patients with severe acute psychiatric illness exhibit a cluster of motor signs (mutism, negativism, rigidity, posturing, stereotypy, staring, etc.) that are identified as the syndrome of catatonia. Catatonia responds to sedative anticonvulsant treatment (barbiturates, benzodiazepines) and to electroconvulsive therapy. These treatments raise seizure thresholds. The commonality in response indicates that catatonia, malignant catatonia, neuroleptic malignant syndrome, toxic serotonin syndrome, delirious mania, catatonic excitement, benign stupor, and oneirophrenia are best evaluated as diverse manifestations of one syndrome for clinical and neuroscience research purposes.

Key words Catatonia · Neuroleptic malignant syndrome · Serotonin syndrome · Electroconvulsive therapy · Benzodiazepines

Introduction

After decades of indifference, the psychopathological syndrome of catatonia has surfaced with questions whether it is an identifiable psychiatric syndrome and what is its position in psychiatric nosology. Credit for defining catatonia as a motor syndrome in patients with behavior disorders is usually given to the German psychopathologist Karl Kahlbaum who published *Die Katatonie oder das Spannungsirresein* in 1874. The concept underwent a metamorphosis, notably by such psychopathologists as Kraepelin and Bleuler, who encouraged its restriction to a subtype of schizophrenia.

But many clinicians see the signs of catatonia in patients otherwise identified as suffering from mania, depression, and toxic states. Is catatonia to be treated as a syndrome with many pathologies as described by clinicians or as a single disease entity as described by some psychopathologists? If the latter, in which disorders can catatonia be identified? Are the forms always similar to that described by Kahlbaum or are other entities to be subsumed under the same label? This report aims to identify behavior disorders with the signs of catatonia as aspects of the same pathophysiology. The disorders to be lumped together include catatonia, malignant catatonia, neuroleptic malignant syndrome, delirious mania, toxic serotonin syndrome, and periodic catatonia. Other syndromes, such as oneirophrenia, toxic delirium, akinetic mutism, and von Economo's encephalitis, exhibit the principal signs of catatonia suggesting that they may have similar roots, but for the present our data are insufficient to include them in the same psychopathology.

Such lumping together of entities is consistent with the philosophical rule of parsimony, also known as Occam's razor, that all other things being equal, the simplest solution is usually the correct one. These entities are acute in onset, exhibit the same signs, and have similar responses to defined interventions. This argument for a

Tab. 1 Principal signs of catatonia

Mutism
Stupor
Negativism (Gegenhalten)
Posturing (catalepsy)
Waxy flexibility
Stereotypy
Automatic obedience
Ambitendency
Echophenomena
Mannerisms

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single entity is meant to influence clinical practice and treatment outcome by assuring adequate treatment for a variety of syndromes now treated in a variety of ways.

The observable signs of catatonia were first culled together by Kahlbaum. The list was extended by others so that recent rating scales include from 21 to 40 motor signs. A list of the more classic signs is presented in Table 1.

Catatonia is defined as a syndrome in which at least two of its signs are present for a day or longer (Taylor 1990; Fink 1997). From 6 % to 9 % of patients, admitted to academic hospital units, meet these criteria. For some patients, the signs are transient, disappearing as the disorder remits. For others, the signs persist until defined interventions are applied. In the 1930s, the administration of amobarbital, a sedative anticonvulsant barbiturate, relieved the syndrome dramatically, but often transiently. Barbiturates were replaced by benzodiazepines. These now relieve the syndrome in more than 80 % of the patients treated. When this treatment has failed, convulsive therapy has been effective.

The syndrome is generally of acute onset. When it is fulminant, systemically devastating, and leading to death, it is labeled as *malignant or pernicious or lethal catatonia*. When the catatonic signs wax and wane and the illness is recurrent, it is labeled *periodic catatonia*. When a patient has four or more episodes within one year, a syndrome of 'rapid cycling' has been described as a feature of manic-depressive illness. These patients often exhibit the signs of catatonia during their manic phases.

A neurotoxic state that follows the use of antipsychotic drugs, labeled the *neuroleptic malignant syndrome (NMS)*, has features that are indistinguishable from those of malignant catatonia, other than that the precipitant is specified as an antipsychotic agent. The *toxic serotonin syndrome (TSS)* is indistinguishable from NMS or malignant catatonia except that the precipitant is an antidepressant agent usually identified as a selective serotonin reuptake inhibitor or a monoamine oxidase inhibitor.

Delirious mania, manic delirium, excited catatonia, and catatonic excitement are identified when motor activity is excessive. *Delirious stupor* may appear suddenly without an obvious general medical explanation (*e.g.*, infection) (Table 2).

Tab. 2 Syndromes of catatonia

- Catatonia
- Malignant (pernicious, lethal) catatonia
- Periodic catatonia
- Delirious mania (catatonic excitement, manic delirium, Bell's mania, acute fulminating psychosis, oneirophrenia)
- Neuroleptic malignant syndrome (NMS)
- Toxic serotonin syndrome (serotonin syndrome)
- Benign stupor
- Rapid cycling mania
- Akinetic mutism

The syndrome *of akinetic mutism* is only occasionally described, but the descriptions meet criteria for catatonia. The syndrome is included within this concept of catatonia to suggest that the treatment for catatonia may be usefully applied.

Catatonia

The motor signs of catatonia are features of many psychiatric and neurologic conditions. The sudden onset of mutism, negativism, posturing, and rigidity in a patient with a behavior disorder calls for a complete examination for the main and secondary signs of catatonia. While we lack experimental data to define the syndrome, we take the presence of two or more signs for 24 hours as sufficient basis for the diagnosis. Its identification is facilitated by rating scales that offer methods of examination (Taylor 1990, 1999; Rosebush et al. 1990; Lohr and Wisniewski 1987; Rogers 1992; Bush et al. 1996 a,b; Fink 1997; Northoff et al. 1999; Bräunig et al. 2000).

The reduction or the full relief of catatonia within a few minutes after an intravenous test dose of amobarbital or lorazepam is considered a positive sign of the syndrome. Absence of the response, however, does not rule out its presence as approximately 20 % of subjects do not respond to such a challenge (Bush et al. 1996a; McCall et al. 1992a, b).

In DSM-III and earlier diagnostic classifications the presence of catatonia identified a patient as suffering from schizophrenia, catatonic type (295.2). Such signs today allow for a broader classification of psychiatric illnesses, including catatonia in a medical condition (293.89), or as a specifier in mood disorders (American Psychiatric Association 1994). But even this acknowledgement is inconsistent with the clinical experience that finds catatonia in patients with mania, depression, and diverse toxic, neurologic, and infectious conditions.

The descriptive literature finds catatonia in a long list of systemic disorders (Taylor 1990; Fink 1997). Catatonia is usually transient, but from time to time, it has become the dominant condition in medically ill patients. At such times, the practitioner needs to recognize the syndrome and call for a change from antipsychotic drugs to sedative anticonvulsant benzodiazepines and/or electroconvulsive therapy. In such instances, when ECT is called for, bias against its use and unavailability of adequate facilities militate against the proper and effective treatment of the severely ill (Fink 1999b).

Malignant catatonia

The fatality rate is high when a patient in good general medical health suddenly develops intense excitement, delirium, high fever, catalepsy, mutism, rigidity, stereotypes, and posturing. The syndrome was first termed "tödliche Katatonie" (fatal catatonia) (Stauder 1934). Other authors have labeled the syndrome *Bell's mania,*

manic delirium, delirious mania, acute or fulminating psychosis, and oneirophrenia (Bell 1849; Meduna 1950; Fricchione et al. 2000).

The patients are autonomically unstable with fever, tachycardia, tachypnea, and hypertension. Speech is disorganized and thoughts are delusional. They refuse food and liquids. They appear to have an infectious encephalopathy, are usually subjected to extensive neurologic assessments, but an infectious cause is rarely documented (Penn et al. 1972; Carroll et al. 1994; Caroff et al. 1998).

Prior to the introduction of modern somatic treatments, such patients either recovered spontaneously or died within a few weeks from physiologic collapse (rarely from a specific cause). The disease is fulminant and treatment must be instituted within the first five days to avoid death (Arnold and Stepan 1952; Philbrick and Rummans 1994).

Neuroleptic malignant syndrome

A *syndrom malin* was described soon after the introduction of antipsychotic drugs. Patients exhibited fever, autonomic instability, rigidity, and changes in mood and alertness that was occasionally fatal. Catatonic signs were part of the description but these were dominated by systemic manifestations. The syndrome was given many names but a description in 1980, labeling the syndrome as the '*neuroleptic malignant syndrome*' (NMS) was widely accepted (Caroff 1980). Muscle rigidity, hyperthermia, altered consciousness, and autonomic dysfunction became the syndrome's hallmarks. The rigidity is often associated with mutism, staring, negativism, posturing, verbigeration, and echophenomena (Koch et al, 2000).

About 0.5 % to 1 % of patients treated with antipsychotic drugs develop NMS, usually within the first two weeks of exposure, when antipsychotic drug doses are being increased rapidly. Both typical and atypical antipsychotic drugs are implicated. The same syndrome has been reported to occur when patients are given carbamazepine or valproic acid, and after the rapid withdrawal of levodopa, amantadine, or benzodiazepines (Keck et al. 1989; Rosebush et al. 1990; Berardi et al. 1998).

The syndrome was recognized as a response to antipsychotic drugs whose mode of action was being explained by their blockade of dopamine (D_2) receptors. For treatment, it seemed logical to recommend the dopamine agonists bromocriptine, amantadine, and *l*-dopa. The muscle weakness and fever were not explicable within the dopamine mechanism. These signs seemed so similar to that seen in malignant hyperthermia that the patients were deemed to be suffering a similar syndrome and dantrolene was recommended. The withdrawal of antipsychotic drugs and administration of dopamine agonists and dantrolene then quickly became the main treatment for NMS (Lazarus et al. 1989).

Others concluded, however, that NMS is not a distinct entity, but an example of malignant catatonia triggered by exposure to antipsychotic medicines (Rosebush et al. 1990; White 1992; Philbrick and Rummans 1994; Fink 1996a; Carroll and Taylor 1997). They recommended that the patients be treated with high doses of benzodiazepines and, if these fail, to be treated with ECT. But because NMS can also be induced by other drugs, the term itself is now inappropriate. The agents associated with NMS include disulfiram, corticosteroids, phencyclidine, abrupt withdrawal of anticholinergic and antihistamine drugs, phenelzine combined with lithium or dothiepin, clozapine withdrawal, metoclopramide, carbamazepine, valproic acid, tetrabenazine, cocaine, and cyclobenzaprine. Many prefer to describe these disorders as examples of malignant catatonia.

Toxic serotonin syndrome

A toxic serotonin syndrome occurs in patients exposed to a rapid increase in dosing of a selective serotonin reuptake inhibitor (SSRI) or when an SSRI is combined with other agents affecting the brain's serotonin system (*e.g.*, monoamine oxidase inhibitors). Patients are restless and sleep poorly. They have an altered sensorium and become delirious. Muscular rigidity associated with mutism, staring, negativism, and echophenomena develops (Fink 1996; Keck and Arnold 2000). The skin is flushed and patients complain of sweating, tremor, shivering, lethargy, sialorrhea, nausea, diarrhea, and abdominal pain. Temperature and blood pressure are elevated, tendon reflexes are hyperactive, movements ataxic, and myoclonus appears. These signs and symptoms are identical to those of malignant catatonia with gastrointestinal symptoms. A recent report found that of 28 patients meeting criteria for NMS, 22 (79 %) also met TSS criteria (Carroll et al. 2001).

Although the full-blown toxic serotonin syndrome is infrequently recognized, milder forms occur in patients who receive potent SSRIs. The incidence and severity rises with the use of polypharmacy, but virtually every medicine that potentiates brain serotonin has been implicated. Laboratory findings include leukocytosis, rhabdomyolysis with elevated CPK, myoglobinuria and renal failure. The laboratory features of TSS are similar to those that characterize malignant catatonia. Patients respond rapidly to withdrawal of the offending medicines and supportive care. We lack reports of the merits of benzodiazepines, but ECT has been used successfully.

Delirious mania and excited catatonia

The syndromes of delirious mania and excited catatonia are indistinguishable. It was delineated by Bell (1849) in a 13-year chart review of 1700 admissions to the McLean

Hospital in Boston. Of 40 patients, three-quarters died. The syndrome has received little attention in the psychiatric literature (Fink 1999a).

The syndrome is marked by the acute onset of the excitement, grandiosity, emotional lability, delusions, and insomnia characteristic of mania, and the disorientation and the altered consciousness characteristic of delirium. The patients are excited, restless, fearful, suspicious, and delusional. Negativism, stereotypy, grimacing, posturing, echolalia, and echopraxia are prominent. Garrulousness, flights of ideas, and rambling speech alternate with mutism. They sleep poorly, are unable to recall their recent experiences or the names of objects or numbers given to them, and are disoriented. Fever, rapid heart rate, tachycardia, hypertension, and rapid breathing are common.

The acute onset leads to the search for a toxic or general medical cause. When flights of ideas become incomprehensible, acute schizophrenia is considered. When grandiosity and delusional ideation dominate the picture, mania is more easily recognized. When delirium is the main feature, a full neurologic evaluation, including extensive brain imaging procedures is usually done. Regardless of presumed cause, the presence of the complex syndrome of mania and delirium, with or without catatonia, justifies the syndromal diagnosis of delirious mania.

Delirious mania is a frightening picture that typically leads the emergency-room physician to prescribe an intramuscular antipsychotic medicine. Such intervention, especially when patients are dehydrated and have an electrolyte imbalance, is likely to induce malignant catatonia.

Periodic catatonia

Kraepelin described a condition of periodic excitement and catatonic features alternating with normalcy in young adults (Kraepelin 1913). Gjessing studied the psychopathology of these patients in detail and described periods of stupor alternating with excitement, waxing and waning for years without the mental deterioration that characterized patients with dementia praecox. About 3 % of his patients had a more malignant form that was incorporated into the dementia praecox concept. Gjessing (1938, 1974, 1976) proposed that the condition resulted from a cyclic nitrogen imbalance. He treated patients with thyroid extract, but this work has not been validated. He assumed that he was observing a discrete disease rather than a syndrome (Lindsay 1948; Minde 1966).

Leonhard's detailed descriptions of psychopathological states included patients with periodic catatonia. Strongly influenced by Leonhard's nosology, German investigators at the University of Würzburg now classify periodic catatonia as a separate phenotype of psychosis, related neither to manic-depressive illness nor to systematic schizophrenia (Leonhard, 1979 1995). Two clinical forms of catatonia are described. *Systematic catatonia* has an insidious onset and a progressive chronic course without remissions with a poor response to antipsychotic drugs. Their relatives are at low risk for schizophrenia. *Periodic catatonia*, by contrast, is recurrent, exhibiting a typical "bipolar" course with periods characterized by grimacing, stereotypes, impulsive actions, aggressivity, and negativism, alternating with stupor, posturing, mutism, and waxy flexibility (Stöber et al. 1995; Beckmann et al. 1996). The Würzburg investigators find the two forms of catatonia to be supported by family studies with the relatives of patients with periodic catatonia being at substantial risk for the syndrome (Stöber et al. 2000a, b, c). We are unsure how patients diagnosed with these syndromes respond to the present recommended treatments for catatonia, since the reports do not detail such experiments. One would expect, however, that so long as the major signs of catatonia are prominent and persistent, treatment with high doses of benzodiazepines and with ECT would relieve the syndromes.

A single syndrome?

What encourages the lumping together of these syndromes? The entities were defined in the 19th and 20th centuries on *ad hoc* bases. While dominant symptoms and signs, course of the illness, and family history were offered as criteria, the overlap of entities in our classification systems is severe. For decades we have debated whether manic-depressive illness and schizophrenia can be differentiated, finding many patients who meet the criteria for both diagnoses at one time or another in the course of their illness. The diagnosis of schizoaffective disorder was created to cover the overlap, and yet we have needed other diagnoses as schizophreniform disorder, delusional disorder, brief psychotic disorder, and psychotic disorder not otherwise specified, among others, to cover the presumed phenotypic differences in patient history and description. Incidentally, despite such subdivisions in classification, the dominant treatment offered these patients is antipsychotic drugs.

Psychiatrists have sought laboratory tests to distinguish the varieties of behavior disorders. Tests for syphilis made it possible to diagnose a defined population with the disease, leading to ever more specific treatments, and to the virtual disappearance of patients with neurosyphilis from psychiatric clinics. The electroencephalogram identified patients with brain dysrhythmias, encouraging a spate of effective anticonvulsant treatments, with the loss of these patients from psychiatric clinics. Psychological tests defined varieties of mental handicap, and genetic studies have, in a few of these patients, separated their pathology. But despite the application of blood, neuropsychological, neuroendocrine, sleep EEG, and brain imaging (CAT, MRI, PET, SPECT) tests, we have been unable to demarcate the remaining

types of mental disorders with which our patients are plagued. We are left with attempts to demarcate illnesses by their phenotypic features and/or by their responses in clinical trials.

As each new treatment has been applied for patients with behavior disorders, some patients respond rapidly and well, while others do not. In psychotropic drug studies, chlorpromazine quickly decreased the expression of psychotic thoughts, regardless of the associated psychopathology. Imipramine relieved depressive mood, but it also reduced the symptoms of phobia. Barbiturates reduced the signs of anxiety but also the motor signs of catatonia. We once thought that clinical trials could verify diagnostic classes, as to use the response to lithium to define mania and the response to imipramine to define depression. But such use of treatment response for classification was derided because the patients who responded appeared to be from diverse classes according to preconceived notions of the classification systems.

The dexamethasone suppression test is an example of this failed logic. With methods to measure serum cortisol, fledgling neuroendocrinologists found the highest serum levels with the least diurnal variability and minimal to no suppression by steroids among severely depressed patients. With successful antidepressant treatment, cortisol metabolism normalized, most rapidly in patients treated with ECT. Some patients identified as suffering from mania, schizophrenia, or other psychiatric disorders by DSM criteria also showed abnormal DST responses. Asked whether the test had merit in identifying major depression, the contamination from 'other diagnoses' was taken as a failure of the test, rather than as a failure of the DSM classification system. As a consequence, a unique diagnostic test was derided and discarded. An alternative tack would have been to find the populations with abnormal DST examinations and seek other commonalities among them, defining thereby a more homogeneous population than the overt descriptive behavior called for by DSM systems.

The patients brought together under the rubric of 'malignant catatonia' show a rapid response to intravenous lorazepam or diazepam. Those that show such a response have a good likelihood of responding favorably to high doses of oral benzodiazepines. Not all, however. Those that do not improve with sedative anticonvulsants respond rapidly and dramatically to ECT. Such responsivity to benzodiazepines and ECT characterizes patients with catatonia, malignant catatonia, neuroleptic malignant syndrome, delirious mania, and delirious stupor. There is anecdotal evidence that patients with TSS and toxic delirium also respond rapidly to ECT. The lumping together of these syndromes under the general rubric of malignant catatonia serves a useful therapeutic aim. It directs our attention to a treatment algorithm with a high likelihood of success.

Patients who are now in stupor or delirium on intensive care units or neurological services should be assessed for signs of catatonia. For those with such signs, the rapid introduction of a lorazepam challenge, high dose lorazepam treatment, and ECT will recall many from their life-threatening condition.

For clinical researchers, viewing these syndromes as reflecting a common pathophysiology should encourage greater attention to laboratory tests that define their commonality. Some clues come from reports of abnormalities in serum iron, DST, and EEG recordings.

Neuroscientists who rely on clinical diagnoses to identify populations with a commonality for brain imaging and genetic testing are prone to study populations with mixed genotypic features and fail to make useful contributions. Except for the remarkable success with syphilis and epilepsy, the past century is filled with examples of failed studies. Defining populations of the mentally ill by their response to a medicine may be a more effective way to identify genotypically similar populations.

Conclusion

As it has been useful to identify the presence of delusions and delirium among patients with behavior disorders because such syndromes respond favorably to specific interventions, so too is it useful to identify patients with catatonia. Identifying delusions in the patients with behavior disorders allows effective treatment to be offered regardless of whether the DSM classification is schizophrenia, major depression, mania, or toxic state. The simple expedient of identifying the presence of catatonia will encourage their effective treatment. For some patients, the relief of these symptoms will also relieve the psychiatric syndrome. For others, further treatment for depression or mania or psychosis is necessary, but the identification of catatonia, whether as the dominant sign in malignant catatonia or NMS, or as a feature of excited catatonia, delirious mania, or delirious stupor, is sufficient to serve a clinical purpose. This pragmatic approach to diagnosis and treatment will also serve a scientific purpose.

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