Clinical Practice to Change With Divorce of Catatonia and Schizophrenia

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Catatonia is a motor dysregulation syndrome of acute onset that is marked by stupor, mutism, negativism, refusal of food, posturing, rigidity, and repetitive speech and movement. It follows a mild systemic illness, more often in the young. Some forms are malignant with high fever, delirium, tachycardia, hypertension, and sweating, occasionally leading to death. For all the 20th century, in each revision of the classification of psychiatric disorders, catatonia has only been designated as a type within the poorly conceived concept of “schizophrenia”. Effective treatments for catatonia, sedative anticonvulsant agents and induced seizures (electroconvulsive therapy [ECT]), were developed in the 1930s. Although they were dramatically effective for catatonia, they offered little benefit for schizophrenia. The diagnostic limitation of catatonia only as a type of schizophrenia blocked catatonic patients from effective treatments. Indeed, prescribing neuroleptic drugs for catatonic patients not only offered inadequate treatments but also subjected them to the risks of precipitated malignant catatonia labeled “neuroleptic malignant syndrome.” The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), divorce of catatonia from schizophrenia benefits both patients and clinicians.1,2

Catatonia was delineated by the German psychiatrist Karl Kahlbaum from among the patients in his sanitarium in 1874. He described 26 case examples in a small book titled “Die Katatonie oder das Spannungssiresein”—“Catatonia or the tension insanity”.3 Catatonia was described in patients with thought, mood, and speech disorders and those having the systemic diseases of tuberculosis, neurosyphilis, and epilepsy. Patients were unusually fearful and anxious. He did not connect the syndrome to psychosis or dementia.

Psychiatrists in Europe and the United States, with Kahlbaum’s descriptions in hand, quickly recognized similar cases. In 1893, Emil Kraepelin, then devising a grand classification scheme for behavior and mood disorders, incorporated Kahlbaum’s catatonia as a specific marker for “dementia praecox.”4 Although Kraepelin recognized catatonia in patients having manic-depressive insanity, he pigeonholed catatonia as a principal diagnostic sign of dementia praecox only.

When Eugen Bleuler reshaped dementia praecox into “schizophrenia” in his 1911 textbook, catatonia remained its main sign.5 Although the subsequent literature reflected 2 points of view, catatonia as an independent clinical syndrome and as a singular sign of schizophrenia, the first official diagnostic classification by a committee of the American Psychiatric Association in 1952 cited catatonia only as a type of schizophrenia. Official revisions of the classification in 1968 and 1980 as well as the World Health Association’s International Classifications of Diseases followed suit. For almost all the decades of the 20th century, catatonia has been regarded as a form of schizophrenia, and patients were prescribed interventions deemed effective for that illness.

In the 1970s, clinicians increasingly reported catatonia in patients with depressive and manic mood disorders, the epilepsies, and in systemic medical disorders.6,7 Catatonia was only occasionally recognized as a unique syndrome, and by 1981, the question was raised, “where had all the catatonics gone?” The answer was complex, based in large part on the hiding of catatonia within schizophrenia and in part on the speed with which the signs of catatonia were resolved by available treatments. By 1930, injections of sodium amobarbital quickly relieved catatonia, and clinicians working in acute psychiatric centers aborted catatonia as soon as it was identified.8 In 1934, the acute benefit of induced seizures to relieve catatonia was demonstrated,9 and by the 1940s, electrically induced seizures (ECT) became the almost universal treatment of the severe psychiatric ill, especially those with catatonia.

Soon after potent neuroleptic drugs were introduced to clinical use in the 1950s, a neurotoxic syndrome was recognized with dominant signs of catatonia accompanied by high fever, hypertension, and tachycardia, occasionally progressing to death.10 Labeled the “neuroleptic malignant syndrome,” the first reports hazards treatments based on the belief that neuroleptic drugs inhibited the brain’s...
dopaminergic transmitter system. Dopamine agonists (eg, bromocriptine) and muscle relaxants (eg, dantrolene) were promoted. Those clinicians who recognized the signs of catatonia in NMS applied the known treatments for catatonia—barbiturates, benzodiazepines, or ECT—with rapidly appreciated success when the neuroleptic drug prescription was also discontinued. These treatments successfully relieved NMS more effectively than did the prescription of bromocriptine and dantrolene.1

Once the tie of catatonia to schizophrenia was questioned, other forms of catatonia were identified. Benign stupor, malignant catatonia, delirious mania, and the toxic serotonin syndrome were brought under the catatonia umbrella.1

A limited recognition of catatonia outside schizophrenia was afforded in the 1994 *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*, classification by the addition of a distinct class of “293.89—catatonia secondary to a medical condition.” In the following years, malignant forms of catatonia were often reported in patients with systemic diseases of lupus erythematosus and anti-N-methyl-D-aspartate receptor encephalitis, and most recently in patients with the self-injurious behaviors in autism spectrum disorders.1

The *DSM-5* revised classification deletes the catatonia type of schizophrenia (295.2), adds a new class of “Catatonia Not Elsewhere Classified (299.89),” retains the medical disease association of catatonia introduced in *DSM-IV* (293.89), and accepts catatonia as a specifier of 10 psychiatric diagnoses.3 The authors do not justify this complexity but sign off with the expectation that clinical experience will clarify their formulation.

The DSM diagnoses are made by the presence of a defined number of symptoms and signs from a checklist for each diagnosis. The DSM-5 catatonia diagnosis is made by the presence of 3 or more signs from a 12-item checklist. For catatonia scholars, such criteria are inadequate. A presumptive diagnosis of catatonia is made by the presence of 1 or 2 motor signs for 24 hours or longer.11 The diagnosis of catatonia is verified by the quick relief afforded by the intravenous administration of 1 or 2 mg of lorazepam, 2.5 to 5 mg of diazepam, or the oral administration of 7.5 mg of zolpidem. Lists of catatonia signs include up to 40 behaviors, not 12.2 When the diagnosis is verified, successful treatment with high doses of benzodiazepines (or ECT in malignant cases) validates the diagnosis.

In patients with verified diagnoses, administering high doses of benzodiazepines—6 to 20 mg lorazepam daily, for example—effectively relieves catatonia in more than 80% of subjects.2 The remainder is relieved by ECT. For those with malignant catatonia, ECT is effective, although daily treatments may be necessary.

It is useful to recall this history because clinicians today are indoctrinated in the Kraepelinian dictum that “catatonia = schizophrenia.” This universally adopted formula impedes our ability to recognize catatonia as an independent syndrome. The reflex prescription of neuroleptics to every case of catatonia is no longer acceptable clinical practice.

In systematic surveys, catatonia is found in 10% of patients on psychiatry, neurology, and emergency department units in academic general hospitals. It is increasingly identified in children and adolescents. The signs of catatonia and the methods of evaluation found in catatonia rating scales offer a useful basis for effective recognition.12

The association of catatonia with the animal fear response labeled “tonic immobility” is an intriguing explanation for the onset of the syndrome and its resolution with sedative anxiolytic agents. When Kahlbaum described catatonia, he reported that his patients were unduly fearful. The evidence for the association of catatonia with the animal fear response is compelling.12

Our clinical guidelines identify and differentiate very heterogeneous populations of schizophrenia, bipolar disorder, major depression, and a glossary of childhood disorders. We accept 30% to 50% reduction in the number and severity of symptoms as “improvement” when treating patients with these syndromes. Full relief is rarely attained. By contrast, the proper application of the known treatments for catatonia assures the full and rapid resolution of all its signs. This note alone distinguishes catatonia from other identified behavior disorders and should encourage the education of practitioners to identify catatonia early in the differential diagnosis of patients with peculiar movements, especially in the young.

**AUTHOR DISCLOSURE INFORMATION**

The author declares no conflicts of interest.

**REFERENCES**