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Catatonia Is not Schizophrenia: Kraepelin's Error and the Need to Recognize Catatonia as an Independent Syndrome in Medical Nomenclature

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Catatonia **is a motor dysregulation syndrome described by Karl Kahlbaum in 1874. He understood catatonia as a disease of its own. Others quickly recognized it among diverse disorders, but Emil Kraepelin made it a linchpin of his concept of dementia praecox. Eugen Bleuler endorsed this singular association. During the 20th century, catatonia has been considered a type of schizophrenia. In the 1970s, American authors identified catatonia in patients with mania and depression, as a toxic response, and in general medical and neurologic illnesses. It was only occasionally found in patients with schizophrenia. When looked for, catatonia is found in 10% or more of acute psychiatric admissions. It is readily diagnosable, verifiable by a lorazepam challenge test, and rapidly treatable. Even in its most lethal forms, it responds to high doses of lorazepam or to electroconvulsive therapy. These treatments are not accepted for patients with schizophrenia. Prompt recognition and treatment saves lives. It is time to place catatonia into its own home in the psychiatric classification.**

Key words: history/classification/*DSM*

Introduction

Catatonia is a syndrome of altered motor behavior accompanying many general medical and neurologic disorders. It is more frequently found among patients diagnosed with mania, depression, and neurotoxic syndromes than among those with schizophrenia. Yet, it is mainly classified as a form of schizophrenia. This persistent failure of proper recognition has unfortunate consequences, leading to poor treatment choices with high morbidity and mortality. How did this come about?

In the 19th century, many authors sought to extract identifiable medical conditions from the morass of their patients' behavioral symptoms. The discovery of bacteria made possible a medical diagnostic system that identified symptom complexes that were verified by the presence of specific pathogens. Homogeneous patient populations and improved antibiotic treatments followed. But no such model existed for disturbances in behavior although psychopathologists sought common ground in symptoms and course of illness. Karl Kahlbaum, director of a private psychiatric clinic in the small town of Görlitz in Germany, coined the term *catatonia* in 1874 for symptoms well known to psychiatrists but never coherently delineated. Kahlbaum's catatonia had previously been recognized as *stupor*, and the French called the lack of communication *stupidité*. Nonetheless, the symptoms were not unfamiliar and were thought by subsequent generations of clinicians to occur across a wide range of different disorders.

Kahlbaum, however, went further in his delineation of catatonia as a disease comparable to progressive paralysis or general paralysis of the insane (GPI), later called "neurosyphilis." "I now want to attempt to portray here a clinical picture in which, just as in progressive paralysis, certain somatic—indeed muscular—symptoms are the accompaniment of certain psychiatric phenomena, and in the one disease as in the other [such muscular symptoms] take on an essential role for the conceptualization of the entire disease process."[1(p4)] As for the prognosis of catatonia, certainly in contrast to the grim outlook for GPI, Kahlbaum wrote: "Recoveries are, in general, quite common."[1(p93)]

Despite the familiarity of the symptoms that Kahlbaum bundled together, his disease concept of catatonia elicited an international conflict among psychiatrists. Two schools emerged. One view supported Kahlbaum's proposal of catatonia as a disease of its own. An opposing view was that catatonia was a complication of different pathophysiologies and not a distinctive disease.

Kahlbaum's supporters were numerous and articulate, as he had become a hero to a younger generation of psychiatrists for his willingness to discard older diagnoses based on humoral concepts and to accept as genuine diseases only those that could be clinically ascertained as

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distinctive in their course. Kahlbaum made the clinical course a principal feature of nosological classification. His student, Ewald Hecker, used Kahlbaum's approach to delineate hebephrenia as another distinct disease.[2]

Clemens Neisser, a young staff psychiatrist in a provincial German asylum considered Kahlbaum to be "one of those quite unusual investigators in science who come to conclusions on the basis of their thorough clinical experience, and few break with the old Pseudo-system as brilliantly as Kahlbaum does in catatonia."[3]

Within 3 years, catatonia was recognized in 4 patients with mania and depression.[4] A decade later, 2 forms of catatonia and the cyclic course of illness beginning with an initial stage of melancholia was reported from New York City's Ward's Island.[5] In the same year, catatonia was recognized in mania in a German report.[3(p84–85)]

Doubters, about as numerous as enthusiasts, considered catatonic symptoms complications of different psychiatric illnesses without possessing any particular syndromic quality.[6] Carl Wernicke, a most influential thinker in German psychiatry before the First World War, wrote: "One sees the value of Kahlbaum's work essentially therein, that he brought together a number of important building blocks for the construction of his [catatonia] edifice, while the edifice itself is not tenable." For Wernicke, catatonia was configured as an "akinetic motility psychosis."[7]

The Kraepelin Position

Emil Kraepelin's third image offered catatonia as a complex of symptoms associated with dementia praecox. Rather than catatonia being an independent illness entity or a complication of many different illnesses, Kraepelin believed that catatonia was, along with hebephrenia and paranoid dementia, a basic presentation of dementia praecox. He did not reject Kahlbaum's and Hecker's notions as much as he incorporated them into the single disease of dementia praecox: "I got the starting point of the line of thought which in 1896 led to dementia praecox being regarded as a distinct disease, on the one hand from the overpowering impression of the states of dementia quite similar to each other which developed from the most varied initial clinical symptoms, on the other hand from the experience connected with the observations of Hecker that these peculiar dementias seemed to stand in near relation to the period of youth."[8]

Also, "I kept Kahlbaum's and Hecker's ideas in mind and tried to collect those cases, which inclined towards dementia as 'mental degeneration processes.' Apart from Kahlbaum's catatonia, I differentiated between dementia praecox, which essentially corresponded with hebephrenia, and dementia paranoides with hallucinations, which quickly developed into mental deficiency."[9] And "Special importance in the establishing of dementia praecox has, not without justification, been attributed to the demonstration of the so-called 'catatonic' morbid symptoms."[8(p257)]

Kraepelin, professor of psychiatry in Heidelberg and then after 1903 in Munich, is a founding parent of modern psychiatric nosology. His influence is manifest in the popularity of successive editions of his textbooks, the first of which appeared in 1883.[10] Catatonia was not mentioned in this first edition, and a friend wrote him to ask why he had not included it.[11]

Once Kraepelin became interested in catatonia, he first agreed with Kahlbaum that it was an independent illness entity. In the fifth edition of his textbook in 1896, Kraepelin described catatonia as one of the "metabolic disorders leading to dementia," alongside dementia praecox and dementia paranoides. Even though he did not agree with Kahlbaum in all points, he said, "I nonetheless see myself obliged, by extensive experience, to view the great majority of these cases as examples of a distinctive form of illness [catatonia]."[12]

Kraepelin's clinical impressions changed often. By the time of the sixth edition of his textbook in 1899, catatonia had become a category of dementia praecox. There were no data to support this evolution in his thinking; Kraepelin had changed his mind. He now devoted more attention to the catatonic form of dementia praecox than he did to presenting dementia praecox itself.[13] It was in this 1899 edition that he definitively separated dementia praecox and manic-depressive illness.

The catatonia of Kraepelin differed markedly from Kahlbaum's. As Eric Arndt, a staff physician in the Heidelberg psychiatric clinic, put it in 1902: "In Kraepelin's view, we are dealing with the occurrence [in catatonia] of peculiar conditions that end mostly in dementia accompanied by stupor or with agitation accompanied by negativism, stereotypies and suggestibility in expressions and actions. The emphasis here is no longer on clinical course and coarse motor phenomena, but on termination in dementia. It is above all the prognosis that influences the diagnosis."[14] Kraepelin's catatonia was truly no longer Kahlbaum's catatonia with its differentiated clinical courses but an effort to bring catatonia into his vision of dementia praecox.

The eighth edition of Kraepelin's textbook in 1913 was the last on which he actively worked. (His death in 1926 interrupted completion of a ninth edition after the War.) By 1913, catatonia had become 1 of the 8 subgroups into which dementia praecox was divided and clearly subordinated to the larger diagnosis. Kraepelin said, "Later experience has shown that catatonic symptoms may in no way be sharply distinguished from the other forms of Dementia praecox." He also opined that nothing in the clinical course or the pathological anatomy made one think these were different diseases: "At any rate we may consider Kahlbaum's catatonia for the most part as a

distinctive clinical course of Dementia praecox.'' And, ''We must limit the designation [catatonia] to those cases alone in which the pathological process of Dementia praecox is at work.''[15]

In 1920, Kraepelin's position turned once again, expressing doubts as to the meaningfulness of separating dementia praecox and manic-depressive illness because the 2 presentations and clinical courses intermingled.[16] This renunciation of much of his life's work reinforces the view that Kraepelin's entire system was impressionistic, including the rather arbitrary shifts about catatonia. For him—and for the rest of the world—catatonia remained firmly part of what Eugen Bleuler had coined in 1908 as ''schizophrenia.''

The Ensuing Debates

Kraepelin's dictum that catatonia equaled schizophrenia was not immediately accepted among German nosologists. In 1898, Schüle[17] acknowledged catatonia a new entity with 6 subtypes, criticizing Kraepelin's incorporation of catatonia within dementia praecox. In the same journal, Aschaffenberg[18] reported an experience with 227 psychiatric patients, finding distribution ratios for catatonia among men and women (men to women, 2:3) different from those with dementia paralytica (3:1).

An active academic industry commenting on Kahlbaum's concept developed among German, French, and American authors. Each effort, in samples of 1–12 patients, confirmed Kahlbaum's descriptions, often discussing ''somatic'' and ''psychologic'' explanations for the disorder.[19] By 1912, a monograph on catatonia by Urstein[20] related an experience with 30 patients, faulting Kraepelin's adoption of catatonia within dementia praecox, finding catatonia in patients with syphilis and other infectious diseases, toxic states, depression, mania, and delirium.

In his 1913 textbook, Karl Jaspers, a leading psychopathologist at the Heidelberg clinic, portrayed catatonia as an illness sui generis characterized by opposing pairs of symptoms (negativism vs automatic obedience, eg). Jaspers' main interest was in the psychology of catatonia, which he found unknowable: ''Sometimes it seems as though the patient is like a dead camera: He sees everything, hears everything, understands everything and yet is capable of no reaction, of no affective display, and of no action. Even though fully conscious he is mentally paralyzed.'' Jaspers put the accent on inhibition, not on a clinical course trending catastrophically downward; in his account of catatonia, he did not mention Kraepelin.[21]

After Jaspers lost interest in psychiatry and turned to philosophy, the mantle of authority in psychopathology fell on Kurt Schneider, first in Cologne and then in Heidelberg. Schneider thought catatonia a complication of many illnesses and rejected Kraepelin's formulations.[22] Another author, Lange[23] reported an experience with 200 patients meeting Kraepelin's constructs for manic-depressive illness and dementia praecox in follow-up studies covering more than 10 years of illness. He found catatonia to be more common among the manic-depressive patients than among those with dementia praecox.

It was Eugen Bleuler, professor of psychiatry in Zurich, who brought Kraepelin's view that catatonia equaled schizophrenia to North America. In his 1916 textbook, Bleuler[24] assimilated catatonia within schizophrenia. He had a milder view of schizophrenia, anticipating many recoveries and not the inevitable course to dementia. Bleuler's optimistic view was more enthusiastically received in the United States than was Kraepelin's pessimism. Bleuler's acceptance was bolstered by a therapeutic optimism within psychiatry engendered by psychoanalysis and the strong anti-German sentiment that followed World War I.

Bleuler envisioned the catatonic patient as suppressing unpleasant memories by silence (mutism), tenseness and rigidity (holds back acts that are compelled by memories), refusal to obey commands, and displacing rising emotions and tension into motor acts that shut out reality (posturing, grimacing, staring, stereotypes). Lethal catatonia was an expression of the death wish. After Bleckwenn's description of the relief of catatonia with amobarbital, Bleuler considered this effect a ''release'' of blocking.[25]

Kraepelin had one more influential disciple abroad. Willi Mayer-Gross, who had been at the Heidelberg clinic before fleeing to England in 1933, landed at the Maudsley Hospital in London and was soon acknowledged as a highly influential figure in British psychiatry. In 1954, he became the lead author—in collaboration with Eliot Slater and Martin Roth—of that era's principal English language psychiatry textbook.[26] Mayer-Gross's position on catatonia was resolutely Kraepelinian that catatonia was a type of schizophrenia. ''Schizophrenia sometimes begins with a sudden outburst of wild excitement…. These cases, formerly called 'delirium acutum' may begin out of the blue without any obvious premonitory signs …. The restlessness and excitation may exceed everything known in psychiatry, except perhaps some epileptic furors. The patient cries, hits, bites, breaks and destroys everything he can lay hand on, runs up and down, fights everybody and keeps moving day and night. It is impossible to establish any rapport with him, he continues to rage when left alone ….''[26(p250)] Kahlbaum and Jaspers would have recognized such patients as catatonic; today we might see them as examples of *malignant catatonia* (MC) or *delirious mania*.[19]

In contrast, neurologic images by French and other continental authors viewed catatonia as one among many motor syndromes, similar to dystonia, Parkinsonism, and dyskinesia.[27–31] The neurologic connection was also central to the studies of epidemic encephalitis by von Economo[32] who described catatonia in many patients in the acute and chronic phases of the illness.

Gjessing[33] described a periodic form of catatonia with hormonal connections. In the absence of effective treatment, he observed his patients for long periods, reported their spontaneous relapses and remissions, and associated the cycles with changes in nitrogen metabolic balance. He described an occasional treatment success with thyroid extracts and concluded that periodic catatonia was a metabolic disorder. Similar reports of a periodic form of catatonia with a relationship to thyroid metabolism dot the literature.[34–36]

Another form of catatonia, with an acute onset and a malignant outcome, was described by Stauder[37] in 27 patients in 1934. He labeled the disorder *Die tödliche Katatonie*, a term that is best translated as *lethal* or *malignant catatonia* (MC). Young adults between 18 and 26 years of age were reported to suddenly become mute, rigid, and either stuporous or severely excited. Fever and autonomic dysfunction were severe, and the outcome was quickly fatal. The syndrome has been described by many authors and is best known today as MC.[19(ch3),38–41] A subtype of the syndrome associated with exposure to antipsychotic drugs is widely recognized as the *neuroleptic malignant syndrome* (NMS) or *neuroleptic induced catatonia*.[19(ch3)] The *serotonin syndrome* is another manifestation of a medication-induced catatonia.[42]

Conflicts in America

Adolf Meyer, Smith Eli Jelliffe, and William Alanson White, leaders in American psychiatry following the images set by Bleuler, viewed schizophrenia and especially its catatonic form as evidence of the psychological basis for the psychoses.[43] Their views became the basis for the 1952 *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) classification that described abnormal behaviors as reactions to psychological and physical stressors and not as defined syndromes.[44]

Contrasting views in America were expressed by George Kirby who pictured catatonia as typically occurring among patients with manic-depressive illness.[45] He argued that Kraepelin had drawn the boundaries of schizophrenia much too broadly. In a monograph titled *Benign Stupors*, August Hoch described 25 psychiatric patients in stupor. Thirteen with manic-depressive illness had a favorable prognosis and 12 with general medical illnesses or schizophrenia had a poor prognosis.[46]

Among psychopathologists, catatonia continued to be recognized within other disorders. In 1969, Pauleikhoff[47] described an extensive 35-year experience with 552 hospitalized psychiatric patients with 64% suffering from 1 of 5 forms of catatonia. Deliria were present in his patients, and he concluded that catatonia was a syndrome of many forms, most with favorable outcome; catatonia was not only a phase of a progressive disorder with a dementia outcome.

Catatonia in the Nomenclature

Despite these many descriptions of catatonia in association with manic depression and general medical and neurologic conditions, a separate nosologic entity of catatonia was not included in psychiatric classifications.

At the beginning of the 20th century, in the absence of an agreed-upon nomenclature, each psychiatrist developed his own descriptive terms for the illnesses of his patients. In an effort to standardize medical diagnoses, the New York Academy of Medicine held a meeting in 1928 on the nomenclature of disease, from which emerged the view that catatonia was a subtype of schizophrenia.[48] This document, as well as a parallel effort by the American Medico-Psychological Association (as the American Psychiatric Association was then known), influenced the first "*DSM*" disease classification of the American Psychiatric Association, published in 1952. In this version, catatonia is recognized only as *schizophrenic reaction: catatonic type* (000-x23).[44(p83)]

In 1948, the sixth edition of the World Health Association's *International Classification of Diseases* (*ICD*) recognized a "catatonic type" among the "schizophrenic disorders."[49] The tenth edition in 1992 was essentially unchanged, except that clinicians in developed lands were encouraged to ignore the subject: "For reasons that are poorly understood, catatonic schizophrenia is now rarely seen in industrial countries, though it remains common elsewhere."[50]

As the *ICD* was undergoing revision in the 1960s, the American Psychiatric Association converted the *schizophrenic reaction* to *schizophrenia* in the second edition of 1968.[51] Catatonia was recognized as a type of schizophrenia, with excited and withdrawn subtypes.

Soon after the 1968 *DSM* edition, successive breaches in the wall of catatonia only as a form of schizophrenia called for renewed debate. Taylor and Abrams, in 4 publications between 1973 and 1979, reported catatonia to be more common among manic and depressed patients than among those with schizophrenia, challenging the limited recognition of catatonia only as schizophrenia.[52–55] Follow-up studies by Morrison[56] found catatonia in more than 10% of 500 patients, most commonly among those with mood disorders. Gelenberg[57] described catatonia among patients with neurologic and general medical illnesses.

The *Diagnostic and Statistical Manual of Mental Disorders* (Third Edition) classification of 1980 ignored these reports and again catalogued catatonia as a type of schizophrenia (295.2).[58] As a logical consequence catatonia, as schizophrenia, called for treatment with neuroleptic medications.

Additional Evidence Against the Catatonia-Schizophrenia Link

Several developments supported the disconnection of catatonia from schizophrenia. The first occurred in 1980 with descriptions of a toxic response to neuroleptic agents and

the identification of the NMS.[59] The patients were mute, rigid, posturing, and in stupor, accompanied by fever, tachycardia, hypertension, and tachypnea. The early authors saw a similarity to malignant hyperthermia and suggested treatment with dantrolene. They also accepted dopamine blockade as the central action of these compounds and recommended treatment with dopamine agonists. Neither approach was useful. In time, NMS was appreciated as a form of MC with a specific precipitant.[19,60] Successful treatment trials with benzodiazepines and electroconvulsive therapy (ECT), the known effective treatments for MC, confirmed their identity.[61]

Based on these reports and a plea that catatonia deserved a home of its own in the classification, the 1994 revision of *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition) additionally recognized catatonia as a disorder due to a general medical condition with a numeric designation of 293.89.[62] Catatonia was also hesitantly accepted as a features specifier in mood disorders.

The renewed interest in a catatonia syndrome encouraged the development of rating scales and effective examination procedures. From 9% to 17% of patients in academic psychiatric inpatient units and psychiatric emergency rooms met criteria for catatonia, more often among patients with mood disorders or toxic states than with schizophrenia.[19]

Catatonia was vouchsafed in many guises.[19(ch3),63] It was reported in children and adolescents ill with autism and mental retardation; treatment trials for catatonia reported quick clinical benefit.[64,65] Self-injurious behavior is a repetitive, uncontrollable, damaging stereotypy in children that is ameliorated by treatment for catatonia.[66] Catatonia is identified in patients with Gilles de la Tourette syndrome,[67] epilepsy, stupors, and fevers of unknown origin[19] and in patients with paraneoplastic syndromes.[68,69]

The syndrome of catatonia has 2 attributes that further separate it from schizophrenia. The signs quickly respond to intravenous amobarbital or benzodiazepines offering clinicians an affirmative test of the syndrome. The same agents are effective treatments, fully resolving catatonia although requiring higher dosages than ordinarily prescribed. About 70% of catatonic patients respond to lorazepam alone, while few respond to antipsychotic agents, another indication that the pathophysiology of catatonia is distinct from that of patients with schizophrenia. ECT is another effective treatment for catatonia in each of its guises and even in its malignant forms. Neither the sedative anticonvulsants nor ECT is considered in treatment algorithms for schizophrenia, further endorsing the divorce of the syndrome from schizophrenia.

Resolving the Error

Many authors, including contemporaries of Kraepelin, recognized the fallacy of regarding catatonia mainly as a type of schizophrenia, and that view is supported by the data collected over the 20th century. Nevertheless, some modern clinicians who adhere to Kraepelin's writings and the supporting opinions of Bleuler, Meyer, Kleist, and Leonhard continue to accept Kraepelin's image of catatonia as schizophrenia, however else they differ in their formulations.

In 1981, a writer asked where the catatonics had gone, suggesting that the widespread use of antipsychotic drugs may be responsible.[70] A better explanation comes from the early 20th century shift in psychiatric practice from the asylum, where catatonia was common, to office practice and ambulatory clinic where it is not. The mutism, negativism, motor abnormalities, and stupors of catatonic patients are not treatable in office settings.[71] Sadly, even in inpatient psychiatry settings, these patients go largely unrecognized. In a Dutch study, clinicians identified catatonia in 2% of 139 inpatients, but the research team identified catatonia in 18%.[72]

Failure to recognize catatonia is also a response to deterioration in the teaching of psychopathology. A clinician cannot recognize what he has not been taught. Classification manuals offer limited pictures of catatonia and list only a handful of more than the 40 recognized catatonic features. Once the linchpin of training, psychopathology now focuses on how to recognize the clinical features needed to apply *DSM* or *ICD* labels from a limited number of symptoms listed in symptom checklists.[73,74] The death of phenomenology following adoption of the *DSM* nomenclature is ably described by Andreasen.[75]

Catatonia is singularly identified with schizophrenia in clinical teaching with the unfortunate consequence that antipsychotic medications are immediately prescribed. These medicines offer limited relief and indeed risk converting the catatonia syndrome to its malignant form.[19] Authors who adopt the Kraepelin equation that catatonia is schizophrenia defend the use of antipsychotics in adolescents and in chronic psychotic patients. They disregard the risks of neuroleptic precipitation of MC and accept delayed resolution of symptoms. Their testimonials challenge the consideration of lorazepam and ECT.[76–78] More critical, however, is the reality that equating catatonia with schizophrenia precludes the use of barbiturates, benzodiazepines, or ECT.

For more than 125 years after Karl Kahlbaum categorized catatonia as a distinct psychopathologic entity, it has been overwhelmingly reported in association with many conditions. Yet, catatonia is allocated a position of dependency in the construct of schizophrenia. Catatonia's divorce from schizophrenia and its recognition as an independent syndrome, akin to delirium, are needed in the next psychiatric classification. The evidence that compels this consideration is extensive.[79,80] A century of well-documented clinical experience cannot be ignored. Catatonia deserves a home of its own in the classification.

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