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COMMENTARY ON FINK ET AL ARTICLE

The Catatonia Conundrum: Evidence of Psychomotor Phenomena as a Symptom Dimension in Psychotic Disorders

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To provide a rational basis for reconceptualizing catatonia in *Diagnostic and Statistical Manual of Mental Disorders* (Fifth Edition), we briefly review historical sources, the psychopathology of catatonia, and the relevance of catatonic schizophrenia in contemporary practice and research. In contrast to Kahlbaum, Kraepelin and others (Jaspers, Kleist, and Schneider) recognized the prevalence of motor symptoms in diverse psychiatric disorders but concluded that the unique pattern and persistence of certain psychomotor phenomena defined a "catatonic" subtype of schizophrenia, based on intensive long-term studies. The enduring controversy and confusion that ensued underscores the fact that the main problem with catatonia is not just its place in *Diagnostic and Statistical Manual of Mental Disorders* but rather its lack of conceptual clarity. There still are no accepted principles on what makes a symptom catatonic and no consensus on which signs and symptoms constitute a catatonic syndrome. The resulting heterogeneity is reflected in treatment studies that show that stuporous catatonia in any acute disorder responds to benzodiazepines or electroconvulsive therapy, whereas catatonia in the context of chronic schizophrenia is phenomenologically different and less responsive to either modality. Although psychomotor phenomena are an intrinsic feature of acute and especially chronic schizophrenia, they are insufficiently recognized in practice and research but may have significant implications for treatment outcome and neurobiological studies. While devising a separate category of catatonia as a nonspecific syndrome has heuristic value, it may be equally if not more important to re-examine the psychopathological basis for defining psychomotor symptoms as catatonic and to re-establish psychomotor phenomena as a fundamental symptom dimension or criterion for both psychotic and mood disorders.

Key words: catatonia/schizophrenia/classification/psychopathology

Introduction

The revision of *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) offers a critical opportunity to address the nosological status of catatonia. In past iterations, "catatonic" symptoms have been vaguely defined, incomplete, and included as a subtype or modifier for schizophrenia, mood disorders, or medical conditions, without a clear theoretical or evidentiary basis. This shortcoming of *DSM* reflects the neglect of classical literature and descriptive psychopathology in contemporary training, practice, and research.

To provide a rational basis for the nosological status of catatonia in *Diagnostic and Statistical Manual of Mental Disorders* (Fifth Edition) (*DSM-V*), we briefly review historical sources, the complexities of the psychopathology of catatonia, and the validity of catatonic schizophrenia. Available evidence indicates that catatonic symptoms are described in association with numerous brain disorders, supporting the conceptualization of catatonia as a nonspecific syndrome as reviewed by Fink et al[1] in this issue. While we agree that catatonia does not necessarily imply schizophrenia, it is also true that catatonic and other persistent psychomotor phenomena comprise an intrinsic symptom dimension of psychotic disorders, with implications for treatment, prognosis, and neurobiological research.

Kahlbaum and Kraepelin: Contributions to the Concept and Nosology of Catatonia

Every major modern and classical writer acknowledged the ubiquitous nature of motor signs and symptoms in patients with severe mental illness.[1–5] Although Kahlbaum proposed catatonia as a single disorder, he instead described a loosely defined syndrome appearing in a variety of conditions.[1] Berrios[4] aptly remarked that "There is a difference between what Kahlbaum actually described and what he thought he was describing at the time." Of the 26 patients Kahlbaum included in his

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monograph,[1] 11 had "neurological" signs and 9 had epileptic seizures; etiologically, there were 2 cases each of tuberculosis and general paresis, 1 case of peritonitis complicated by delirium and 1 case was too brief to draw any conclusion. A factor analysis of Kahlbaum's cases clearly delineated a neurological and a "psychotic depression" factor.[4] Although Kahlbaum advocated longitudinal observations, few of his cases were followed and the outcome was far from benign: only 8 of his cases remitted, whereas 8 died or became chronic and in 10 cases no follow-up information was provided.[4]

Kraepelin had a sophisticated view of the psychopathology of catatonia. He did not arbitrarily subsume catatonia under dementia praecox without supporting evidence. Starting in 1891, he systematically followed a large number of patients meticulously recording careful observations.[6] He published the first longitudinal study involving 63 cases of catatonia followed for up to 4 years.[7] Only 24 patients remitted from the index episode and 14 of these relapsed within a short period. Nine of the remaining 10 remitted patients continued to display mild disturbances. Because of its recurring course and poor prognosis in his follow-up study, Kraepelin regarded catatonia as a subtype of dementia praecox in successive editions of his textbook.

It is important to note that Kraepelin considered catatonic symptoms as "secondary accompanying phenomena"[8(pp248/255)] and not fundamental clinical manifestations of dementia praecox. By the eighth edition of his textbook, Kraepelin described 11 clinical forms of dementia praecox, only 1 of which was called catatonia ("Catatonia, excitement, stupor [melancholia attonita]") though catatonic symptoms appeared in other forms as well.[8] Predominantly, catatonic types formed 19.5% of all dementia praecox cases in his series.[8(p153)] Despite their frequency, he could not distinguish well-circumscribed catatonic syndromes within dementia praecox: "the catatonic symptoms may be present in the morbid picture in all possible grades and groupings".[8(p254)]

Kraepelin did not simply equate catatonia with dementia praecox. He was fully aware that motor symptoms occur in other psychiatric and medical conditions, emphasized that catatonic symptoms were not sufficient to diagnose dementia praecox, and had only a preliminary place in his classification system: "So far as judgment on the subject is possible today, we may regard the catatonia of Kahlbaum as in the main a form, though peculiar, of dementia praecox. On the other hand, catatonic morbid phenomena are undoubtedly also observed in many quite different morbid processes to a greater or lesser extent, so that its appearance alone does not justify the conclusion that catatonia in the sense just indicated is present".[8(p132)] However, in no other psychiatric conditions were catatonic symptoms observed "in such extent and multiplicity"[8(p257)] than in dementia praecox. Of the catatonic symptoms, negativism and mannerism were of diagnostic importance being "more characteristic … [and] scarcely accompanying any other morbid process in a pronounced form throughout a long period",[8(p258)] while waxy flexibility and echophenomena were "not rare" in manic-depressive illness nor was stupor combined with cataplexy.[9(pp36,79,106)] It is important to mention here that catatonia in Kraepelin's sense meant *persisting motor phenomena*; following continuous observation of the patient and based on the opinion of several clinicians, the initial diagnosis was made within 4 weeks of admission in his clinic,[6] with further observations continuously added to refine the diagnosis until the case was closed.[10(pp61–62)]

Addressing the presence of catatonic symptoms in manic-depressive illness, Kraepelin elaborated on the *specific quality* of the catatonic phenomena that distinguish dementia praecox from manic-depressive illness.[8(pp261–270)] Thus, for Kraepelin, the justification for keeping catatonia as part of dementia praecox was its *unique psychopathological pattern and duration*. Motor signs and symptoms in other illnesses including mania and depression similar to those seen in dementia praecox were mentioned without the catatonic adjective as opposed to the "genuine catatonic morbid symptoms" in dementia praecox.[8(p258)] Having examined transient catatonic states in healthy individuals and a variety of psychiatric and neurological conditions, Schneider[11] reached the same conclusion that fleeting motor symptoms in acute psychoses do not justify the catatonic adjective in the diagnosis. A comparison between catatonic and manic excitement observed in 100 patients in each group seemed to support Kraepelin's view despite some overlap of symptomatology: blocking (29 vs 2), waxy flexibility (15 vs 2), stereotyped speech (29 vs 7), mutism (48 vs 8), and negativism (52 vs 8) predominated in agitated schizophrenia patients.[12] Clearly, the distribution of catatonic symptoms in functional psychoses depends on where the division between schizophrenia and mood disorders is drawn and how catatonia is defined; both decisions are, to a large extent, arbitrary. Broadly defined mania and catatonia results in a high frequency of catatonic symptoms, [13–15] whereas the opposite is expected using a restrictive (Schneiderian) concept of.[16]

For Kraepelin, persistent motor symptoms defined a catatonic subtype of dementia praecox just as enduring negative symptoms define the current concept of deficit syndrome in schizophrenia.[17] The psychopathological aspect of the Kraepelinian legacy has been largely overlooked by modern catatonia research with few exceptions.[18]

Psychopathological and Diagnostic Issues of Catatonia

In our opinion, the main problem is not the place of catatonia in *DSM* but its lack of conceptual clarity. Few modern writers realize that catatonia has no clear definition.[19,20] Lohr and Wisniewski noted that most

investigators assumed that catatonia was a ''coherent, well-defined syndrome … without ever defining exactly what it was''[19(p204)] and ''catatonia has not been fully defined''.[19(p208)]

Based on the broad methodological principles of ''understanding'' and ''explanation'', Jaspers provided a general psychopathological definition of catatonia that remained the guiding principle in most schools of European psychiatry: ''Somewhere between the neurological phenomena, seen as disturbances of the motor apparatus, and the *psychological* phenomena, seen as sequelae of psychic abnormality with the motor apparatus intact, lie the *psychic motor phenomena*, which we register without being able to comprehend them satisfactorily one way or the other''.[21(p179)] For Jaspers, catatonic signs and symptoms constituted the psychic motor phenomena,[21(p179)] whereas the psychological phenomena, such as hysterical or depressive stupor, were ''not conceived to be primary motor phenomena but are actions and modes of expressions which have to be understood''.[21(p179)] This is the theoretical viewpoint that essentially restricted catatonia to the schizophrenic psychoses[21(p183)] but acknowledged the presence of a variety of motor symptoms in other psychiatric disorder without calling them catatonic. Similar to Kraepelin, Kleist[22] and the Wernicke-Kleist-Leonhard school of psychiatry regarded only qualitatively abnormal psychomotor phenomena as catatonic.

Modern psychiatry ignores Jaspers' concept of catatonia but makes no attempt to redefine it in psychopathological terms, which is the most significant stumbling block in modern catatonia research. There is no guiding principle of what makes a motor symptom catatonic, and consequently, the boundaries of catatonia have become unclear. For example, ''denudativeness'' or ''prankishness'' is sometimes included in catatonia[19(p205)] and recently priapism[23] and ''catatonic coma''[24] were mentioned, whereas, loquaciousness,[1] blocking,[8] or ''bizarreries'',[25] three classical symptoms have nearly disappeared from its symptomatology. One may ask, for instance, why akathisia—described in 1901 as a motor disorder accompanied by psychological symptoms[26]—is not subsumed under catatonia, at what point psychomotor retardation in depression becomes catatonic, or why ''grasp reflex'' is part of catatonia but not other signs of frontal lobe dysfunction?[27,28] Similarly, excitement, a core catatonic symptom, has not been systematically examined in modern studies.

There is still no consensus on which signs and symptoms constitute a catatonic syndrome or syndromes.[20] Bleuler questioned the existence of a catatonic syndrome: ''I would not insist that these symptoms are intrinsically more related to each other than to other symptoms.''[29(p180)] Lohr and Wisniewski[19(p211)] pointed out that ''Because catatonia at the present time is not well-defined as a clinical syndrome, it is questionable whether all the different clinical descriptions in the many case reports of catatonia represent the same clinical state. For example, many patients have been called catatonic on the basis of mutism and rigidity, and in many cases, it is not clear what the authors considered to be catatonic (as opposed to say, parkinsonian) signs.'' Factor and latent class analytic studies suggest that beyond the classical retarded-agitated division, there are further syndromes within the domain of catatonia depending on the type of patients and the number of catatonic symptoms examined.[18,30–33] Preliminary evidence indicates that the symptom profile of catatonia observed in chronic psychotic patients[34–36] appears to be different from acutely emerging mostly stuporous catatonic states and may constitute a separate condition or a group of conditions as proposed by the Wernicke-Kleist-Leonhard school.[37]

Owing to the conceptual confusion, the existing diagnostic systems and catatonia rating scales significantly differ from each other in terms of the time-frame of the examination and the number, composition, and definitions of signs and symptoms that would form the catatonic syndrome(s). *Diagnostic and Statistical Manual of Mental Disorders* (Fourth Edition) (*DSM-IV*)[38] and *International Classification of Diseases, Tenth Revision* (*ICD-10*)[39] list 11 and 9 signs and symptoms, respectively, for catatonia. In modern studies, symptoms assessed for catatonia range from 8 to 40.[14,18,27,28,32,35,36,40–43] The definitions of the same items on different scales are inconsistent even for such basic symptoms as mannerism or rigidity. In addition, the symptom threshold for the diagnosis of catatonia varies from 1[14,39] to any combinations of 2 to 4 symptoms.[15,19,28,36,38,41,42]

Modern rating scales provide a cross-sectional assessment, although catatonic symptoms fluctuate over time such that longer periods of observation are needed to obtain the full picture.[8,29,37] This potential bias in describing catatonic phenomena was demonstrated by a recent study[43]: Significantly more symptoms (8.4 ± 3.3 vs 4.1±1.8) were rated in chronic psychotic patients based on long-term observation as opposed to the standard cross-sectional assessment. A further problem is that all rating scales were validated on acute, mostly akinetic catatonic patients. Consequently, one such scale had to be significantly modified to adjust it to chronic catatonic patients.[44]

While classical texts provide excellent starting points in the current debate on catatonia, eventually modern studies using sophisticated assessment methods are necessary to determine its nosological position. The ideal methodological requirements would be the prospective, independent screening, and evaluation of motor and other psychiatric symptoms with standardized rating instruments in a large sample with mixed diagnoses coupled with neurobiological tests and long-term follow-up. Currently, there are no extensive clinical or follow-up studies

attempting to delimit the clinical boundaries and course of catatonia that meet these criteria.

Treatment Response and the Heterogeneity of Catatonia

There are consistent clinical reports that benzodiazepines are effective in acute catatonic syndromes, particularly stuporous conditions but no placebo-controlled randomized studies have been published.[45] Due to its favorable pharmacokinetic properties,[46] lorazepam is widely recommended as the drug of choice in catatonia but other benzodiazepines or other agents may also be effective.[46–50] However, the efficacy of lorazepam in treating acute catatonic symptoms in schizophrenia seems to be limited compared with catatonia associated with mood or other disorders.[51,52] According to a recent review, only 20%–30% of patients with catatonic symptoms in the context of schizophrenia respond to benzodiazepines.[45]

Enduring catatonic symptoms in chronic psychoses have received less attention. The only controlled study testing the efficacy of lorazepam in persistent catatonia was a 12-week long randomized placebo-controlled crossover trial involving chronic schizophrenia patients with prominent catatonic symptoms.[53] None of the 16 patients showed noticeable improvement in catatonia on lorazepam. Similarly, in a 5-year open study, benzodiazepines were ineffective in chronic catatonic schizophrenia.[54] The lack of response to lorazepam of catatonia observed in chronic psychotic patients raises again the possibility that acute and chronic catatonic conditions may have different pathophysiology.

Case series and retrospective reviews indicate that acute catatonic syndromes also respond well to electroconvulsive therapy (ECT).[55,56] However, catatonia associated with schizophrenia was less likely to respond than that with mood or medical disorders[57,58] but ECT is still superior to benzodiazepines in these patients.[59,60] The only controlled study involving patients with chronic catatonic schizophrenia that met the methodological criteria for inclusion into the Cochrane analysis compared ECT, nonconvulsive stimulation, and sham ECT.[61] No significant differences between the effects of the 3 methods were found with regard to short-term outcome.

In summary, while benzodiazepines and ECT are often dramatically effective in acute stuporous catatonia as discussed by Fink et al,[1] preliminary results in patients with catatonia associated with chronic psychoses are discouraging and suggest that catatonic symptoms may be heterogeneous in both psychopathology and neurobiology.

Catatonic Schizophrenia

There is overwhelming evidence from the preantipsychotic era as well as from studies of drug-naive patients that a variety of psychomotor symptoms are an integral part of the acute[40,51,62,63] and particularly in the chronic phase of schizophrenia.[34,36,64,65] Preliminary evidence suggests that the symptom profile of catatonia in schizophrenia is different from that of other catatonic disorders,[18] a notion that harks back to Kraepelin and Bleuler.[29(p211)] Limited data also indicate that catatonia in the chronic phase of schizophrenia is phenomenologically different from what is usually observed in an acute episode; chronic patients display more stereotypes, mannerisms, automatic movements and bizarre postures, and less immobility, mutism and vegetative symptoms.[33,36,66] Significant associations have been found in schizophrenia between catatonia and sex, younger age, parkinsonian and negative symptoms, and more severe psychopathology.[36,42,66–69] Interestingly, residual but not the acute motor symptoms were correlated with clinical variables.[42]

Although catatonia was routinely and erroneously equated solely with schizophrenia in the United States, early in the last century, recent evidence is less convincing that patients with catatonia are mostly or disproportionately diagnosed with schizophrenia today. In a review of 11 studies surveying the rates of disorders underlying catatonia, we found a mean of 28% of catatonic patients diagnosed with schizophrenia (range 7%–67%) vs 44% diagnosed with mood disorders (range 28%–71%).[70] In fact, contrary evidence suggests a dramatic decline in the diagnosis of catatonic symptoms among patients with schizophrenia over the last century. Although the recognition of catatonia among psychiatric patients in general has increased, the diagnosis of catatonia attributed to schizophrenia has nearly vanished in the United States. In a recent unpublished analysis using a national database, we found that fewer than 0.4% of patients discharged from hospitals in the United States with schizophrenia were diagnosed with the catatonic subtype (http://hcupnet.ahrq.gov), significantly less than the 7%–17% having catatonia in general psychiatric populations.[70] During the 20th century, the percentage of patients with the catatonic subtype of schizophrenia dropped to single digits in the developed world.[71,72] Studies comparing different periods of time at single sites using consistent criteria showed a significant decline in the diagnosis of catatonic schizophrenia ranging from 28.8% to 77.0% since the Kraepelinian era.[71,73–76]

Closer inspection of published data reveals that acute catatonia has been noted only intermittently in the course of schizophrenia, with the rate increasing in proportion to the length of follow-up.[77,78] On the other hand, Stompe et al[71] and Astrup[79] showed that the decline in catatonic schizophrenia is primarily due to the fall-off in diagnosis of catatonia in chronic schizophrenia (mainly "systematic schizophrenia" in the Leonhard system). This finding conflicts with the fact that catatonic symptoms were more frequently observed in patients with chronic schizophrenia in the historical record and are more common among chronically ill populations in

institutional settings and in developing nations.[51,69,80–82] Although numerous reasons have been proposed for the decline in the recognition of catatonic schizophrenia, we favor the hypothesis that catatonic symptoms classically associated with chronic schizophrenia are no longer recognized because of changing diagnostic fashions and the de-emphasis of descriptive psychopathology. The influence of diagnostic bias was demonstrated by Stompe et al,[83] who showed a direct correlation between the number of catatonic signs used in classification systems (57 in Leonhard vs 11 in *DSM-IV*) and the prevalence of catatonic schizophrenia (23.6% vs 9.5%, respectively). Applying *ICD-10*, *Diagnostic and Statistical Manual of Mental Disorders* (Third Edition Revised), and historical criteria to the same cohort of patients with schizophrenia, Hoffler et al[84] diagnosed the catatonic subtype 3 times more often using traditional classifications.

The influence of catatonia on treatment response and outcome is also rarely considered in treatment trials of schizophrenia, even though rating instruments are available and preclinical data on the second-generation antipsychotic drugs predicted a reduction in liability for inducing or worsening catatonia.[85–87] Catatonic schizophrenia was diagnosed in 7.4% of patients in a trial of clozapine and 5% of patients in a trial of risperidone, but neither treatment response nor worsening of catatonia was reported in either study.[88,89] In a trial by Naber et al,[90] 11.5% of patients were diagnosed with the catatonic subtype of schizophrenia and showed a favorable response to clozapine. Martenyi et al[91] reported an ameliorative effect of olanzapine on catatonic-like symptoms in schizophrenia. In contrast, Girish and Gill[60] showed greater improvement in psychotic and catatonic symptoms from ECT compared with risperidone in a randomized trial of acute catatonic schizophrenia. Evidence from case reports is mixed, with reports of second-generation antipsychotics reducing catatonia, causing catatonia, or precipitating NMS in some patients.[92,93] Several authors published data suggesting that antipsychotic response varies depending on the clinical presentation and duration of symptoms; acute catatonic schizophrenia has a favorable prognosis and response to antipsychotic treatment,[71,76,79,94,95] whereas catatonia correlated with severity, poor treatment response, and chronicity of schizophrenia in other studies.[73,77,95,96]

Similarly, although catatonic symptoms have been invoked as positive predictors of response to ECT in schizophrenia, [97–99] treatment results are limited and mixed. Catatonic symptoms are neglected in modern trials of ECT in mood disorders as well as in schizophrenia.[100,101] However, in a retrospective study, Wells[102] reported preferential response to ECT in patients with catatonic or schizoaffective subtypes of schizophrenia. Dodwell and Goldberg[103] noted "perplexity" as a positive predictive factor. Suzuki et al[59] reported a positive response to ECT in all 11 patients diagnosed with chronic catatonic schizophrenia unresponsive to medications; Thirthalli et al[104] reported that patients with catatonic schizophrenia responded faster to ECT than other subtypes. However, ECT in combination with clozapine reduced symptoms significantly more in schizoaffective patients than in catatonic or hebephrenic subgroups in a study by Gazdag et al[105] Thus, while psychomotor phenomena are an intrinsic feature of acute and chronic schizophrenia, they are insufficiently recognized in clinical practice and research but may have significant implications for treatment outcome and neurobiological studies.

Conclusion

In response to a proposal to include catatonia as a separate disorder in *DSM-IV* made 18 years ago,[106] it was pointed out that "a consensus definition of diagnostic criteria for catatonic syndrome with guidelines for determining the threshold for the presence of the symptoms requires input from the growing number of investigators"[20]. This suggestion still rings true today. Due to the lack of conceptual clarity and well-designed prospective follow-up studies, several important issues concerning the psychopathology, diagnosis, treatment, and classification of catatonia remain uncertain such that classifying catatonia in *DSM-V* remains arbitrary.[107]

In our opinion, the most parsimonious options, consistent with current knowledge and historical evidence, would be the following: first, catatonia could be added as a proposed independent entity either in the text of *DSM-V* as proposed by Fink et al[1] or in the appendix among other syndromes for further research; second, to revise and extend the list of psychomotor symptoms defined as catatonic based on unifying and rational psychopathological principles; and third, include revised psychomotor symptoms as a fundamental symptom dimension or criterion for major psychotic and mood disorders. At this juncture, dissociation of catatonia completely from the major psychotic and mood disorders may have unintended consequences; separation may lead to further marginalization of catatonia as a nonspecific syndrome and to continued exclusion from vital research on the significance of psychomotor phenomena as a symptom dimension of psychotic and mood disorders.

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