

Catatonia in DSM 5: controversies regarding its psychopathology, clinical presentation and treatment response

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Over the past two decades, there has been an upsurge of interest in catatonia, which is reflected in the attention it received in DSM 5, where it appears as a separate subsection of the Schizophrenia Spectrum and Other Psychotic Disorders (APA, 2013). This commentary argues that due to the lack of solid scientific evidence, the extended coverage of catatonia in DSM 5 was a premature, and consequently, a necessarily ambiguous decision. The psychopathological foundations of the modern catatonia concept are lacking therefore its boundaries are fuzzy. There are only a few, methodologically sound clinical, treatment response and small-scale neurobiological studies. The widely recommended use of benzodiazepines for the treatment of catatonia is based on case reports and open-label studies instead of placebo-controlled, randomized trials. In conclusion, the catatonic concept espoused by DSM 5 is necessarily vague reflecting the current state of knowledge.

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The diagnosis of catatonic syndrome (catatonia hereafter) has vitally important clinical implications. A sizeable proportion of catatonic patients has serious underlying medical or psychiatric conditions (Fink and Taylor, 2003). A time lag of days or weeks between the appearance of the first catatonic signs/symptoms and adequate symptomatic treatment is not uncommon (Tuerlings et al., 2010). If not treated early and vigorously, life-threatening medical complications such as venous thromboembolism, aspiration pneumonia, urinary tract infection, dehydration, and electrolyte disturbances could ensue (Clinebell et al., 2014). The diagnosis of catatonia is significantly associated with higher rates of mortality (Tuerlings et al., 2010) and suicide (Kleinhaus et al., 2012).

CATATONIA IN DSM 5

Psychiatrists who do not closely follow recent research on catatonia would be surprised to learn that approximately 10% of all acute psychiatric admissions could present with sufficient number of catatonic signs/symptoms to qualify for the diagnosis of catatonia (Fink, 2009). Thus, for many clinicians, one of the unexpected new features of DSM 5 was the inclusion

of catatonia under a separate heading in the chapter on Schizophrenia Spectrum and Other Psychotic Disorders (APA, 2013). This was not the original plan of the expert committee responsible for the psychoses chapter, but the committee members changed their minds following the repeated requests and arguments of a group of catatonia scholars. In fact, these scholars made a case for separate category for catatonia as a *distinct clinical-diagnostic entity* (Francis et al., 2010). This broad, all-inclusive view of catatonia was unsuccessfully challenged (Ungvari et al., 2010) and it eventually found its way into DSM 5 although in disguise and not without ambiguity. According to the architects of the chapter on Schizophrenia Spectrum and Other Psychotic Disorders, the expansion of catatonia in DSM 5 was justified by its frequency, the need to facilitate its wider recognition and its excellent treatment response to benzodiazepines (Tandon et al., 2013). None of these pragmatic arguments, however, amounts to a criterion to devise a nosological category.

Catatonia appears in three forms in DSM 5: “Catatonia associated with another mental disorder (catatonia specifier),” “Catatonic disorder due to another medical condition” and “Unspecified catatonia.”

The list of signs/symptoms is identical in the first two subcategories, while the third is a “wastebasket” category for catatonia of uncertain origin or incomplete presentation (APA, 2013). When no underlying psychiatric or medical conditions can be detected, the category of “Unspecified catatonia” could lead to catatonia being regarded as a distinct nosological entity, although it is not stated as such in the DSM 5 text. Thus, while DSM 5 does not endorse catatonia as an independent clinical diagnosis, it leaves the door ajar to it by establishing a loosely defined, independent category. The common perception is that catatonia has become a separate clinical entity although not yet an independent clinical diagnosis (Parker, 2014). This ambiguity in DSM 5 clearly demonstrates the uncertainty surrounding the entire concept of catatonia.

PROBLEMS WITH THE DEFINITION AND DIAGNOSIS OF CATATONIA

There are fundamental problems with this all-encompassing concept of catatonia, which is likely to become the prevailing view in the coming years due to the huge influence that DSM 5 will exert on psychiatric practice and research. This paper argues that giving such prominence to catatonia was probably a premature decision, as it is not in keeping with recently available data, nor with the tenets of classical descriptive psychopathology on catatonia that had underpinned its place in psychiatric classifications until DSM-III appeared in 1980. The traditional psychopathological view of catatonia still predominates in some European schools of psychiatry, particularly the sophisticated catatonia classification of the Kleist-Leonhard system that has heuristic value but never been adequately validated (Leonhard, 1957).

The major difficulties with the current concept of catatonia and its position in DSM 5 are as follows.

The central problem is the lack of a psychopathological definition of catatonia. Current definitions such as “motor and mood dysregulation syndrome” (Fink, 2011), “disturbed motor functions amid disturbances of mood and thought” (Fink, 2009) and “concurrent motor, emotional, vegetative and behavioral symptoms” (Naranayaswamy et al., 2012) are non-specific, if not tautological. Admittedly, however, even the best definitions are merely crude approximations of the very complex phenomena of psychomotor disturbances. Among modern authors, only Lohr and Wisniewski (1987) have pointed out that most authors assume catatonia to be a “coherent, well-defined syndrome,” “without ever defining exactly

what it was.” (Lohr and Wisniewski, 1987, p. 204). Without a generally agreed upon definition, what makes a sign/symptom “catatonic” is an entirely arbitrary decision. Consequently, catatonia is becoming boundless and its clinical presentation is increasing, for instance, with “priapism” (Fischel et al., 2009) and “coma” (Freudenreich et al., 2007) recently joining the ever-increasing symptom profile of catatonia. We return to this problem later in the text.

DSM 5 requires the presence of at least 3 out of 12 symptoms to define catatonia, while published rating scales include 23-40 symptoms (Sienaert et al., 2011). The 7 published rating scales together comprise 54 partly overlapping symptoms. A further problem is that the existing rating instruments not only differ in the number of symptoms they measure, but also in the definitions thereof and the number of symptoms sufficient for the diagnosis of catatonia (Kirkhart et al., 2007; Carroll et al., 2008; Sienaert et al., 2011). The most frequently used instrument, the Bush-Francis Catatonia Rating Scale (BFCRS; Bush et al., 1996), stipulates that 2 of the 23 symptoms should be met within 24 hours to diagnose catatonia. The BFCRS requires a 5-minute examination, although it also recommends a review of case notes and unobtrusive observation. This brief examination is clearly inadequate because such cross-sectional assessment cannot capture the whole gamut of catatonic symptomatology (Ungvari et al., 1999a), particularly as classical studies based on year-long personal observations clearly attest to the fluctuation of catatonic symptomatology (e.g. Bleuler, 1911/1950; Leonhard, 1957). A further difficulty is that these generic rating scales may not accommodate catatonic symptoms of different durations and origins (Wong et al., 2007). To sum up, the descriptive features and diagnoses of catatonia as they appear in contemporary diagnostic guidelines and rating scales vary widely and reflect the overall uncertainty surrounding the concept of catatonia.

“KRAEPELIN’S ERROR”

An oft-repeated argument justifying catatonia as a separate diagnostic category is that although Kahlbaum (1874) originally described it as a disease entity, creating the catatonia subtype of dementia praecox (schizophrenia) Kraepelin narrowed catatonia to dementia praecox (schizophrenia) and lost sight of the clinical reality that catatonic symptoms do occur in other diagnostic categories. We believe that the argument regarding ‘Kraepelin’s error’ is flawed. Kahlbaum (1874) claimed to have described a new disease entity,

but a re-analysis of his 26 cases clearly showed that what he described was a host of peculiar, simple and complex speech, motor, and behavioural signs and symptoms occurring in a variety of medical, neurological and psychiatric conditions (Berrios, 1994). That catatonic symptoms were not pathognomonic to any psychiatric illness was common knowledge by the 1880s. Supporting this view, Seglas and Chaslin's (1890) magisterial review cites numerous references to this effect. The omnipresent nature of short-lived catatonic symptoms was reasserted in a landmark paper published 100 years ago (Schneider, 1914). This was well-known to Kraepelin as he cited many of the pertinent papers in his textbook (Kraepelin, 1913).

Kraepelin was a far too methodical and thorough clinical observer and scientist to misunderstand catatonia's significance in psychiatric nosology. In fact, Kraepelin relied on research findings when devising his classification. He followed up a cohort of 63 patients presenting with *persistent* catatonic signs/symptoms for up to 8 years (Kraepelin, 1896). Of the 63 patients, 53 never remitted and ended up in a "defect" state, similar to his hebephrenic and paranoid dementia praecox cases, while the remaining 10 showed signs of subtle to mild motor disturbances. Thus, Kraepelin had good reason to create the catatonic subtype ("Krankheitsformen") of his dementia praecox group. However, Kraepelin also emphasized that only patients with *enduring bizarre motor phenomena* qualified for inclusion in the catatonic form of dementia praecox (Kraepelin, 1913, p. 257). Kraepelin noted that short-lived motor symptoms did occur in a variety of other psychiatric illnesses including manic-depressive illness, *but he classified only the persistent motor signs and symptoms as "catatonic"* (Kraepelin, 1913, p. 258).

THE TRADITIONAL AND CURRENT CONCEPT OF CATATONIA

Opposing to what is perceived as "Kraepelin's error" (Fink and Taylor, 2010), the currently prevailing view, promoted mainly by American authors from the 1970s (Gelenberg, 1976), is that catatonia is a ubiquitous syndrome, most frequently associated with mood disorders (Fink, 2009; Tandon et al.; 2013). Following this line of argument, it would have been illogical to retain catatonic schizophrenia. Poor reliability and validity were the other reasons given for the removal of the traditional catatonic subtype of schizophrenia from DSM 5. While catatonic schizophrenia has never been validated and was kept in classifications

because of traditions and clinical 'wisdom', it is undeniable, however, that motor symptoms and signs are frequently observed in schizophrenia, justifying the term "schizophrenia with prominent catatonic features" instead of "catatonic schizophrenia," as we suggested (Ungvari et al., 2005). In this respect, the 'catatonia' specifier for psychoses in DSM 5 is in keeping with the available scientific evidence.

Kraepelin's early follow-up study and his refined concept of catatonic motor phenomena seem to have been glossed over by most modern authors. The psychopathological roots of the catatonia concept in classical European psychiatry, which reached its pinnacle in Kraepelin and Bleuler's nosology, were evident in Jaspers' definition: "Somewhere between the neurological phenomena, seen as disturbances of the motor apparatus, and the psychological phenomena, seen as the sequel 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" (Jaspers, 1963, p. 179). In Jaspers' conceptualization, hysterical (conversion) or depressive stupor belonged to the psychological domain because they "were not conceived to be primary motor phenomena but are actions and expressions which have to be understood" (Jaspers, 1963, p. 179). In short, Jaspers' definition of catatonia includes only those simple or complex motor phenomena that cannot be understood in the context of the patient's life and current situation or cannot be explained by biological causation. Jaspers' definition is dynamic as it implies that the circle of catatonic symptoms should shrink as neuroscience develops. To ascertain catatonia according to Jaspers' psychopathological principle in clinical practice requires repeated personal examinations to enable the clinician to carefully observe and analyze the motor phenomena, their duration and relation to other signs and symptoms and the patient's subjective experiences in the context in which they arose. This complex assessment is not required by modern rating instruments. The current, simplified and over-arching approach to catatonia is illustrated by the renaming of conversion disorder ("psychogenic stupor") as "psychogenic catatonia" (e.g. Salam and Kilzieh, 1988) based on the superficial resemblance of cross-sectionally ascertained signs and symptoms, thereby diluting the classical concept of catatonia (Ungvari et al., 1994). This is a typical example of the positivist, "atheoretical" approach taken to its extreme.

The contemporary concept of catatonia is based on cross-sectional evaluation. DSM 5 does not specify

a timeframe for assessing catatonia. As mentioned, the most frequently used rating scale, the BFCRS (Bush et al., 1996), requires only a 24-hour observation to reach the diagnosis of catatonia. Within such a short period, many catatonic symptoms that wax and wane and fluctuate in intensity cannot be reliably ascertained (Ungvari et al., 1999a). Thus, the current catatonia diagnosis does not overlap with that of classical authors who saw patients several times over a considerably longer period than 24 hours, and hence could identify the *enduring motor phenomena*.

EVIDENCE SUPPORTING THE MODERN CONCEPT OF CATATONIA

A further problem with the modern concept of catatonia as embodied in DSM 5 is, that data to justify catatonia as a distinct diagnostic category are few and far between. Apart from a few exceptions (e.g. Peralta and Cuesta, 1999; Docx et al., 2012), well-designed, methodologically sound clinical studies using comprehensive assessment are lacking. Contemporary follow-up studies are rare and short-term (Francis et al., 1997) and their methodology does not stand up to rigorous scrutiny. Another difficulty is that nearly all modern studies focus on retarded (stuporous) catatonia, ignoring the traditional subtype of excitement, mainly because there are no firm psychopathologically informed signposts to help identify the catatonic nature of excitement and differentiate it from, for instance, the aggressive outbursts of an impulsive person with antisocial personality disorder.

Further, no systematic studies have examined the subjective experiences of catatonic patients and the association of such experiences with the clinical forms of catatonia. Similarly, systematic studies exploring the biological markers of catatonia are lacking although testable hypotheses concerning its pathophysiology are available (Asztalos et al., 2014). Modern research has not continued pioneering biological investigations (e.g. Gjessing 1974), apart from the systematic genetic studies conducted by Leonhard's disciples at Wurzburg (Schanze et al., 2012).

It is frequently claimed that lorazepam has "near-specific" therapeutic effects in catatonia (Rosebush and Mazurek, 2010). Indeed, lorazepam and other benzodiazepines do suspend short-lived catatonic symptoms temporarily or permanently, mostly in stuporous cases occurring in a variety of medical and psychiatric conditions, but are less effective for relieving the psychomotor symptoms of schizophrenia (Rosebush and Mazurek, 2010; Naranayaswamy et

al., 2012). Yet, there have been no large-scale, double-blind, controlled studies comparing lorazepam to placebo or other benzodiazepines, or other psychotropic drugs that case reports or small case series have found effective for catatonia, such as clozapine (Chattopadhyay et al., 2012), carbamazepine (Kritzinger and Jordaan, 2001) and topiramate (McDaniel et al., 2006). The only placebo-controlled, double-blind, cross-over trial with lorazepam that targeted only catatonic symptoms in chronic schizophrenia found that lorazepam had no effect on any of the catatonic phenomena (Ungvari et al., 1999b).

To conclude, at this point there is no compelling evidence to justify classifying catatonia as a separate diagnostic category. The poorly defined catatonia concept in DSM 5 is likely to extend its boundaries even further, thus rendering catatonia a vaguely circumscribed, etiologically heterogeneous group and hindering future clinical and biological studies. The catatonic concept espoused by DSM 5 reflects the current state of knowledge.

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Katatónia: ellentmondások a pszichopatológiával, klinikai megnyilvánulással és a kezelésre adott válasszal kapcsolatban

Az utóbbi két évtizedben megélénkült az érdeklődés a katatónia iránt. Ez abban is tükröződik, hogy a tünetegyüttest a DSM 5 Szkizofrénia és Egyéb Pszichotikus Zavarok fejezetében önálló alfejezetbe sorolták. Jelen tanulmány mellett érvel, hogy a katatóniának kiemelt helyet adni a DSM 5-ben – megalapozott tudományos bizonyítékok hiányában – túl korai, és következőképpen megkérdőjelezhető döntés volt. A modern katatónia-koncepciónak hiányos a pszichopatológiai megalapozottsága, ezért bizonytalanok a határai. Az irodalomban nagyon kevés a megfelelő módszerrel végzett patofiziológiai, klinikai és terápiás hatékonyságot vizsgáló kutatásról szóló közlemény. A katatónia kezelésére széles körben javasolt benzodiazepinek használata esetismertetésekben és nyílt klinikai megfigyeléseken, nem pedig randomizált, placebo kontrollált vizsgálatokon alapul. Összességében a DSM 5 által elfogadott katatónia-koncepció szükségképpen elnagyolt, és tudásunk jelenlegi szintjét tükrözi.

Kulcsszavak: DSM 5, katatónia, pszichopatológia