Psychomotor Psychoses – The Enigmatic Catatonic Phenotype

Willem MA Verhoeven,[1] Frank MMA van der Heijden,[2] Bruno Pfuhlmann[3] and Gerald Stöber[4]

1. Director, Residency Training and Head, Centre of Excellence for Neuropsychiatry, Vincent van Gogh Institute for Psychiatry and Part-time Professor, Department of Psychiatry, Erasmus University Medical Centre; 2. Vice-director, Residency Training and Chief, Specialised Outpatient Department for Psychotic Disorders, Vincent van Gogh Institute for Psychiatry; 3. Professor of Psychiatry and Vice-chair, Department of Psychiatry, Psychosomatics and Psychotherapy, University of Würzburg; 4. Assistant Professor of Psychiatry and Vice-chair, Department of Psychiatry, Psychosomatics and Psychotherapy, University of Würzburg

Abstract

Since the second half of the nineteenth century, disorders of psychomotor behaviour have been described as part of psychotic states. In this respect, the descriptions of catatonia by Kahlbaum and of the motility symptom complex by Wernicke form the historic start of the catatonia concept. In both the German and French psychiatric traditions, psychoses characterised by motor abnormalities and a polymorphic psychopathological picture have been diagnosed in clinical practice until now. In contrast, the current internationally used taxonomies cover catatonia as a subtype of schizophrenia, secondary to a general medical condition or as a specifier of a mood disorder. In this article, the differential diagnosis of the catatonic syndrome will be outlined, with special emphasis on the Wernicke–Kleist–Leonhard classification system. In addition, catatonia in autism spectrum disorders and as part of a genetic syndrome is outlined. Finally, the prevalence of catatonia is discussed. It is advocated to include catatonia as a new diagnostic class in the psychoses chapter of the *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition (DSM-5).

Keywords

Catatonia, Leonhard, schizophrenia, affective disorder, autism, Prader-Willi syndrome, Kleefstra syndrome, *Diagnostic and Statistical Manual of Mental Disorders*

Disclosure: The authors have no conflicts of interest to declare.
Received: 17 October 2011 **Accepted:** 22 November 2011 **Citation:** *European Psychiatric Review*, 2011;4(2):78–83
Correspondence: Willem MA Verhoeven, Vincent van Gogh Institute for Psychiatry, Stationsweg 46, 5803AC Venray, The Netherlands. E: wverhoeven@vvgi.nl

Since the second half of the nineteenth century, motor abnormalities have been associated with severe psychotic states. In this respect, the description by Carl Wernicke in 1900 of the so-called motility symptom complex, later termed motility psychosis, should be mentioned.[1] About two decades before, in 1874, Karl Ludwig Kahlbaum had already published his well-known monograph, *Die Katatonie oder das Spannungsirresein*, in which he delineated catatonia as a separate neuropsychiatric disorder with disturbances in motor functionality and a characteristic alternating course. Previously, such catatonic states were designated as stupor, which in France was termed 'stupidité'. Kahlbaum identified motor signs in terms of mutism, psychotic negativism, catalepsy, stereotypies, verbigeration and muscular symptoms. In addition, Kahlbaum stressed the combined occurrence of depressive and manic symptoms as part of the different stages of the disease and, based on symptomatology, severity and prognosis, he differentiated three subtypes termed catatonia mitis (melancholia with stupor), catatonia gravis (mixed affective state) and catatonia protracta (chronic course). In the sixth edition of his textbook in 1899, Emil Kraepelin incorporated catatonia in his concept of dementia praecox with its poor prognosis in general, but stressed at the same time that motor symptoms could also be part of his concept of a manic-depressive illness with good prognosis and psychotic features, or may coincide with a great variety of somatic diseases. In 1911, Paul Eugen Bleuler broadened Kraepelin's concept of schizophrenia and attributed psychological explanations to catatonic symptoms. In his view, posturing and grimacing, for example, were signs of avoiding reality, whereas mutism was regarded as a suppression of undesirable thoughts. In 1917, Karl Bonhoeffer conceptualised the so-called exogenous reaction types associated with medical conditions which could lead to catatonic symptoms.[2]

Although Kraepelin's restrictive view that catatonia equals schizophrenia still dominates the psychiatric diagnostic systems, it should be underlined that, since the beginning of the last century, several other disorders have been described in which a motor symptom complex with a phasic course dominates the clinical picture.[3] In 1901, Alexander Pilcz wrote *Die periodischen Geistesstörungen*, in which he made a distinction between the periodic course of, among others, mania, melancholia, amentia, paranoia and circular psychosis.[4] In 1932, Rolv Gjessing described a catatonic syndrome with a phasic course in which periods with stupor and excitement alternate in strict longitudinal regularity. This periodic catatonic phenotype was hypothesised to originate from nitrogen metabolic imbalances.[5] As reviewed in 1974 by Leiv Gjessing, this periodic syndrome may last for decades or disappear automatically and the duration of the periods varies individually from days to weeks or months.[6] Also, in the French psychiatric tradition, catatonia is, apart from schizophrenia, a well-recognised diagnostic entity associated with transient and polymorphic psychotic disorders, such

as the 'bouffées délirantes polymorphes'.[7] Factor and cluster analytical studies of catatonia have demonstrated that it can be viewed as symptom, syndrome or separate nosological entity.[8,9] Until it is clear which symptoms are most relevant for a diagnosis of catatonia according to the International Classification of Diseases (ICD) and the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), a screening instrument should be used systematically. In this respect, the most appropriate scale seems to be the Bush–Francis Catatonia Rating Scale (BFCRS), although a variety of scales exist.[10] In 1996, Bush et al. defined catatonia by the presence of a spectrum of motor symptoms and described 23 signs (see *Table 1*).[11] They emphasised that symptoms occur in retarded and excited forms.

The idea that catatonia has to be considered as a subtype of schizophrenia or associated with affective or medical conditions has persisted until now in the international psychiatric classification systems, the DSM, fourth edition (DSM-IV) and ICD-10. Both taxonomies follow a nosological approach and comprise cross-sectional diagnoses without prognosis and with arbitrary symptom patterns. *Table 2* summarises which BFCRS items are included in the DSM-IV and ICD-10.

The DSM-IV contains five symptoms of catatonia (stupor, excitement, negativism/mutism, stereotypy and echophenomena), of which two are required for the diagnosis of catatonic type. According to the ICD-10, for a diagnosis of catatonic schizophrenia, only one or more of seven symptoms should dominate the clinical picture (stupor/mutism, excitement, posturing, negativism, rigidity, waxy flexibility, command automatism/perseveration). In both taxonomies, catatonia is primarily considered as a subtype of schizophrenia or occurring secondary to a general medical condition. Besides, catatonic signs may serve as specifiers of a mood episode.

In addition to the Kraepelin-based classification, Wernicke, Kleist and Leonhard included in their classification of psychoses, based on aetiology and prognosis, various nosological entities within the catatonic syndrome and advocated the necessity to distinguish a quantitative increase in psychomotor activity from qualitative psychomotor disturbances. Three different forms of psychomotor psychosis were delineated: 1) motility psychosis characterised by purely quantitative psychomotor disturbances; 2) periodic catatonia with akinetic and hyperkinetic phases; and 3) the group of systematic catatonias.[12]

Differentiated psychopathology, as advocated by Leonhard, has always emphasised the importance of a detailed description of signs and symptoms of psychiatric disorders.[13] This viewpoint contrasts with the American opinion, which is heavily driven by psychoanalytic theories and loss of phenomenology.[14] In addition to the catatonic syndromes as included in the DSM-IV and ICD-10, it should be emphasised that catatonia may present as a co-morbid syndrome of autism and is rather frequently observed in conjunction with developmental disorders. Concerning catatonia as a co-morbidity of autism, early childhood catatonia has to be considered as a more complex differentiation of autism.

In the following sections, the catatonic syndromes from the Wernicke–Kleist–Leonhard (WKL) tradition and catatonia in autism spectrum disorders and as part of a so-called behavioural phenotype will be elaborated concisely. In addition, some epidemiological data will be presented. Potential treatment strategies will not be discussed in this primarily psychopathological review.

Table 1: Signs of Catatonia According to Bush et al.

Sign	Description
1. Excitement	Non-purposeful motor hyperactivity
2. Immobility or stupor	Extreme hypoactivity, minimally responsive to stimuli
3. Mutism	Reduction or absence of speech
4. Staring	Fixed, non-reactive gaze, reduced blinking
5. Posturing	Maintenance of posture for longer than usual
6. Grimacing	Maintenance of odd facial expressions
7. a. Echolalia	Mimicking of examiner's speech
b. Echopraxia	Mimicking of examiner's movements
8. Stereotypy	Repetitive, non-goal-directed motor activity
9. Mannerisms	Odd, purposeful voluntary movements
10. Verbigeration	Repetition of meaningless phrases or sentences
11. Rigidity	Maintenance of position, despite efforts to be moved
12. Negativism	Resistance to instructions or contact
13. Waxy flexibility	Maintenance of new posture after reposturing
14. Withdrawal	Refusal to eat, drink or make eye contact
15. Impulsivity	Inappropriate behaviours without explanation
16. Automatic obedience	Exaggerated co-operation
17. Mitgehen	Raising arm in response to light finger pressure
18. Gegenhalten	Resistance to passive movement
19. Ambitendency	Indecisive, hesitant movement
20. Grasp reflex	Reflex grasping in response to palm striking
21. Perseveration	Repeatedly returns to same topic or movements
22. Combativeness	Undirected aggression or violent behaviour
23. Autonomic abnormality	Temperature, blood pressure, pulse, respiratory rate, diaphoresis

Items 1–14 have to be used for screening for the presence or absence of catatonia (at least two signs are needed to establish its presence); items 1–23 are used to measure the severity of catatonia (scored on a 0–3 scale). Source: Bush et al., 1996.[11]

Table 2: Catatonic Items According to Bush et al. as Represented in the DSM-IV and ICD-10

BFCRS Item	DSM-IV	ICD-10
1. Excitement	+	+
2. Immobility or stupor	+	+
3. Mutism	+	+
4. Staring	-	-
5. Posturing	+	+
6. Grimacing	+	-
7. a. Echolalia	+	-
b. Echopraxia	+	-
8. Stereotypy	+	-
9. Mannerisms	+	-
10. Verbigeration	-	-
11. Rigidity	+	+
12. Negativism	+	+
13. Waxy flexibility	+	+
14. Withdrawal	-	-
15. Impulsivity	-	-
16. Automatic obedience	-	+
17. Mitgehen	-	-
18. Gegenhalten	-	-
19. Ambitendency	-	-
20. Grasp reflex	-	-
21. Perseveration	-	+
22. Combativeness	-	-
23. Autonomic abnormality	-	-

+ = included; - = not mentioned.
BFCRS = Bush–Francis Catatonia Rating Scale; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders, fourth edition; ICD-10 = International Classification of Diseases-10.

Table 3: The Three Forms of Psychomotor Psychoses and their Symptomatology According to Leonhard

Hyperkinetic and Akinetic Motility Psychosis	
a. Hyperkinesia in expressive and reactive motions; severe distractibility by environmental conditions with continued senseless motor activity	
b. Hypokinesia with disappearance of reactive motions, stiffness of expressive motions, reduction or standstill of voluntary movements (akinesia/stupor)	
c. Unarticulated sounds of expressive character (in hyperkinetic episodes)	
d. Possibly mood alterations, hallucinations, delusions	
Periodic Catatonia	
a. Acute onset and unipolar or (usually) bipolar course with hyperkinetic and akinetic episodes	
b. Qualitatively distorted psychomotor activity with a characteristic admixture of hyperkinetic and akinetic traits during acute episodes and residual states	
c. Characteristic psychomotor disturbances with iterations, stereotypies, grimacing, parakinesia, stiff and jerky movements with loss of natural grace, rigidity of posture, impulsive actions and negativistic behaviour	
d. Incomplete remissions, development of residual states of varying degrees with prominent psychomotor disturbances	
e. Possibly mood alterations, hallucinations, delusions	
Systematic Catatonias	
a. Insidious beginning, chronic course without remissions	
b. Development of irreversible, sharply delimited residual states	
Six different subphenotypes, each characterised by specific symptom constellations:	
Parakinetic	Parakinetic (bizarre expressive/reactive) movements of the whole body; agrammatical sentences; jumpiness of thought; carefree mood
Manneristic	Mannerisms within complex movements; progressive stiffness of psychomotor activity; impairment of voluntary motor activity
Proskinetic	Proskinesia (abnormal tendency towards automatic movements reactive to external stimuli); impulse-automatism ('Anstossautomatie, Mitgehen, Gegengreifen'); verbigerations
Negativistic	Negativism (resistance, omissions); ambitendency; negativistic excitement with aggressiveness; contorted postures; impulse-automatism
Speech-prompt	Only talkative when a stimulus comes from outside; autism; quick, premature answers (short-circuiting in speech); talking past the point ('Vorbeireden'); vacant facial expression
Sluggish	Sluggish verbalisations; continuous hallucinations; introverted with distracted facial expression; extinguished initiative

Source: Leonhard, 1957.[16]

Catatonia in the View of Carl Wernicke, Karl Kleist and Karl Leonhard

Introduction to the Concept

Affective disorders, thought disorders and disorders of psychomotor behaviour constitute the main categorical disease entities within the WKL classification systems.[1,15,16] Psychomotor disturbances were separated early by Wernicke into episodic, remitting syndromes, which affected psychomotility ('Motilitätspsychose'),[1] psychomotor phenomena due to organic brain lesions[17,18] and specific chronic catatonic diseases.[19-21] In the group of psychomotor diseases, the label 'catatonia' was restricted only to those subphenotypes with a chronic course in the so-called endogenous psychoses.[13]

According to Wernicke, the term 'psychomotor disturbances' refers to primary abnormalities of involuntary (psychomotor) movements and the speech act, which are therefore not secondary to disorders of thought and affectivity.[1] It is also important to distinguish purely quantitative alterations of psychomotor activity from qualitative psychomotor disturbances. As previously mentioned, three independent forms of psychosis have been differentiated by Leonhard whith a symptomatology primarily characterised by psychomotor disturbances (see *Table 3*).

Motility Psychosis

Motility psychosis represents a subform of the cycloid psychoses, which run a bipolar, phasic and remitting course. In hyperkinetic episodes, a quantitative increase, especially of reactive and expressive movements, occurs, whereas, in akinetic episodes, these movements are reduced. Expressive movements are involuntary movements appearing in connection with certain mental states (e.g., facial expressions, gestures), whereas reactive movements represent immediate and involuntary motor reactions to sensory impressions (e.g., beckoning). In severe cases, voluntary movements are also affected up to an akinetic stupor. Polymorphous and fluctuating affective, delusional or hallucinatory symptoms may accompany the psychomotor symptoms without being prominent. In motility psychosis, the psychomotor disturbances are purely quantitative in nature, differentiating this condition from catatonic psychoses in the strict sense, which are characterised by various qualitative psychomotor abnormalities.

Periodic Catatonia

Periodic catatonia shows a bipolar and polymorphous symptomatology with hyperkinetic and akinetic states. The onset is often acute and the course is typically intermittent, with acute exacerbations followed by incomplete remissions. During acute episodes, hyperkinetic and akinetic distortions of psychomotor activity are characteristically intermingled. As a consequence, and in contrast to motility psychosis, hyperkinesis in periodic catatonia displays a peculiar rigidity of all movements due to the combination with akinetic traits. Motion sequences lack a natural harmonious interplay, so that natural grace of motion gives way to a jerky clumsiness, with movements appearing stiff and becoming repetitive and monotonous in the sense of stereotypes or iterations. These qualitative disturbances imply that reactive and expressive motions lose their meaningful content; e.g., distorted facial expressions turn to grimaces, which occur particularly in the upper part of the face. The clinical picture may also include mood alterations, delusions and hallucinations, which usually disappear after the acute exacerbation. After one or more acute episodes, adynamic residual states of varying degree occur. Psychomotor symptoms remain identifiable at any stage of the residual syndrome.

Systematic Catatonias

Systematic catatonias belong to the systematic schizophrenias, which usually begin insidiously and run a chronic course leading to stable and irreversible catatonic residual states. In the beginning, there may be fluctuations in the intensity of the symptoms but, within a few years, the characteristic symptom cluster of the specific residual states is visible continuously. The distinctly characterised residual states of systematic catatonias can be clearly distinguished from periodic catatonia and can be differentiated into six subforms (see *Table 3*). Each of these subforms represents a characteristic clinical syndrome with a specific constellation of symptoms. In each fixed symptom constellation, a specific qualitative disturbance of psychomotor activity (such as parakinetic restlessness of movements, mannerisms

with rigidity in gesture and motion, negativism, proskinesis, speech-inactivity with sluggishness and speech-promptness with abnormal readiness to answer) constitutes the central feature.

Summary

In the view of WKL psychopathology, the diagnosis of catatonia is based on a precise ascertainment and differentiation of cardinal catatonic symptoms in characteristic catatonic syndromes. This is in polar opposition to recent conceptions which have defined catatonia cross-sectionally as a (more or less arbitrary) combination of a minimum number of catatonic symptoms, which may include any kind of motor abnormalities.[22,23] Catatonia rating scales and the international classification systems do not consider the interrelationship of the motor symptoms to each other nor the significance of motor abnormalities within distinct symptom constellations. The importance of a differentiation between qualitative and quantitative psychomotor disturbances is also neglected in current definitions. This is a major impediment, since only psychomotor abnormalities in the strict sense, occurring independently from disorders of affect or thought, can provide a basis for a differentiated diagnostic classification of catatonic psychoses beyond diagnostically non-specific catatonic syndromes.

Diagnostic differentiation between periodic catatonia and the group of systematic catatonias is highly reliable with quite considerable stability of diagnosis.[24,25] Empirico-genetic and molecular studies confirm the scientific value of the differentiation. In most cases, systematic catatonias appeared to be sporadic and, among potential environmental risk factors, the excess of mid-gestational maternal infections indicates early neurodevelopmental disturbances. In contrast, periodic catatonia is strongly transmitted within pedigrees, with linkage studies and genome-wide association studies showing evidence of genetic heterogeneity.[26–28] Therefore, a more complex classification of catatonia seems to provide a useful tool for further neurobiological and therapeutic research.

Catatonia in Autism, Genetic Syndromes and Other Conditions

Autism

Autism is a developmental disorder defined as a severe and persistent restriction in communication skills, including lack of social and emotional reciprocity, as well as stereotyped and repetitive behaviours. Early infantile autism was originally described in 1943 by the Austrian paediatric psychiatrist Leo Kanner as 'autistic disturbance of affective contact'. One year later, the Austrian paediatrician Hans Asperger reported comparable findings under the title 'Die "Autistischen Psychopathen" im Kindesalter'. Both Kanner and Asperger considered autism to be a contact disorder of children with severely impoverished relations with the environment (i.e., 'autistic aloneness'). Wing introduced the term 'autism spectrum disorder', which can be described on the basis of information from three domains: (a) social reciprocity; (b) verbal and non-verbal communication and imagination; and (c) a restricted, stereotyped pattern of interests and activities.[29] These elements still constitute the diagnostic criteria from the DSM-IV category of pervasive developmental disorders.[30]

Catatonic features are frequently observed in autism and related disorders, especially in the case of intellectual disability. The most prominent symptoms are disorders of speech and behaviour and psychomotor abnormalities. With respect to abnormal movement patterns, slowing or stopping activities are mostly seen, which may be accompanied by short episodes of excitement and impulsivity. Bizarre behaviours, such as odd gait and stiff postures, are also regularly present, sometimes with psychotic phenomena such as paranoid ideation.[31] It should be stressed, however, that catatonia in the so-called autism spectrum disorder is generally not equivalent to the current definitions of catatonia in non-autistic patient groups. Therefore, the term 'autistic catatonia' was proposed as more appropriate, in which, for at least one month, the following should be present: freezing and being resistant to prompting, very slow voluntary motor movements, and stopping in the course of movement.[32] It is nowadays suggested to restrict the diagnosis of catatonia in autism to those cases in which stupor or mutism occur that may be accompanied by an increase in pre-existent features, such as stereotypies and echophenomena.[33] This viewpoint is in contrast with the description by Leonhard of the early childhood catatonias.[34]

Genetic Syndromes

Already at the beginning of the 19th century, stuporous conditions were observed in patients with intellectual disabilities. In 1809, the French psychiatrist Philippe Pinel regarded stupor and idiocy as identical. Over the past decades, rapid developments in clinical genetics have elucidated the genetic underpinnings of several syndromes. Parallel to this development, motor abnormalities have been demonstrated to be integrated in the behavioural phenotype of a variety of genetic syndromes. The most recent example is the 9q subtelomeric deletion syndrome, nowadays called Kleefstra syndrome, which is caused by haploinsufficiency of the euchromatin histone methyltransferase (EHMT1) gene and of which the phenotype, from adolescence on, is associated with severe motor symptoms that culminate in an apathy syndrome.[35,36] Prader–Willi syndrome (PWS), caused by a lack of expression of genes on the paternally derived chromosome 15q11–q13, has been reported to be associated with recurrent atypical psychoses that may present with psychomotor/catatonic symptoms.[37] Until now, however, it is not clear whether the genetic defect in these two and in other genetic syndromes is pathogenically involved in the motor dysfunctions within the catatonic spectrum.

Other Conditions

As outlined above, catatonic features can be observed in a variety of psychiatric diseases (schizophrenia, mood disorders, autism and genetic syndromes). Apart from these disorders, catatonia may be an element of several general medical and neurological conditions and, for example, inborn errors of metabolism. Some examples of the latter are porphyria and cerebrotendinous xanthomatosis.[38] Relevant other disorders associated with catatonia are, for example, epilepsy, endocrine dysfunctions, infections and vascular anomalies.[39]

Prevalence and Epidemiology

As mentioned earlier, disturbances in psychomotor behaviour, particularly catatonic symptoms, may occur in a variety of psychiatric conditions, such as motility psychosis, periodic catatonia and systematic catatonia. In addition, catatonic symptoms may be part of the psychopathological picture of psychiatric and somatic disorders and may manifest in genetic syndromes. Given the scope of this article, emphasis will be given to the rates of catatonia in patients who meet a diagnosis of schizophrenia, affective disorder or autism.

Schizophrenia

Originally, catatonia was considered as a schizophrenic subtype and, consequently, all epidemiological data were collected from prevalence studies in schizophrenia. It has to be stressed, however, that the prevalence figures obtained over a period of about one century are heavily influenced by changes in diagnostic criteria in psychiatry, differences in patient samples, the technical developments in the differentiation between somatic and neurological disorders and the introduction of antipsychotic treatment in the early 1950s.[40-43] Moreover, in the past decades, in addition to the introduction of atypical antipsychotics, a wide scale of rehabilitation programmes have been rolled out, and both have led to better symptom control.[44-46]

As reviewed by several authors, in the first half of the last century, high prevalence rates of catatonia were reported that varied between 10 and 30 %, with some reported figures reaching 50 %.[47,48] Thereafter, reported prevalence figures dropped to 2–10 %, which, however, most probably does not reflect a real reduction of catatonic features in schizophrenia but is more likely related to the under-recognition of the catatonic symptom profile.[49,50]

Affective Disorders

According to the original descriptions given by Kahlbaum, and as a consequence of the evolution and expansion in diagnostic criteria, the automatic tendency to attribute catatonia to schizophrenia has been broken through by the addition of a diagnostic class of mood disorders in the DSM-IV and ICD-10, in which catatonic features serve as specifiers for a subtype. According to these criteria, catatonic symptoms can be established in both depression[51,52] and mania.[52-54] These studies mention prevalence figures of 20 % and 27–31 % in depression and mania, respectively. It should be stressed that these percentages are obtained from rather small patient samples. As compared with schizophrenia, however, the catatonic syndrome seems to be more frequently observed in affective disorders.

Autism

The prevalence figures of catatonia in autism are rather scarce since most publications deal with summarised case reports. Most data come from patient samples that were primarily referred for the differential diagnosis and treatment of severe autism.[55] As far as can be deduced from the available data obtained from either cross-sectional or prospective analyses, the prevalence figure of catatonia in autism varies between 12 and 17 %.[31,56] Also, here, the broadening of the concept of autism has resulted in an increase in the proportion of catatonic features observed in patients.[57]

Conclusions and Future Directions

As can be inferred from the previous sections, catatonia may be a disorder in itself or may occur either in the context of a psychiatric or somatic disease, or in association with a genetic syndrome such as PWS. In his *Grundriss der Psychiatrie* from 1946, Leonhard already presented a detailed description of psychomotor symptoms as part of organic brain diseases and dementia.[58] It should be stressed that catatonia can be regarded as a separate diagnostic entity, as in periodic catatonia, or can be considered as a psychopathological dimension associated with other conditions. Catatonia may start acutely or have a gradual, progressive course; it can even have a life-threatening outcome.

As elegantly stated by Ungvari et al., there is still no consensus on which signs and symptoms constitute the catatonic syndrome and the authors advocated the necessity to re-establish psychomotor phenomena as a fundamental symptom dimension or criterion for both psychotic and mood disorders.[48] The categorical approach is reflected by the detailed clinical descriptions of psychotic disorders with catatonic features by Leonhard,[13] which has been demonstrated to have major implications for treatment and prognosis. In this top-down concept, research is focused on putative pathophysiological mechanisms. Following the bottom-up orientation, the analysis of the puzzling interplay between biological and contextual systems may ultimately lead to more dimensional and/or functional classifications of mental disorders, including catatonia.[59]

With respect to the developmental trajectory towards the DSM, fifth edition (DSM-5), two proposals for catatonia have been made.[60,61] The first suggests the inclusion of a new diagnostic class for catatonia in the psychoses chapter, which will lead to more focus on the recognition of catatonic features and therefore to a better understanding of its aetiology (e.g., catatonia in psychotic disorder, catatonia in mood disorder, catatonia in autistic disorder). The second suggests to add catatonia as a specifier to a categorical diagnostic entity (e.g., schizophrenia with catatonia as a specifier, etc.). Since catatonia/catatonic features may occur as a separate disorder or as target symptoms of other co-morbid conditions, the first proposal seems to be most appropriate for clinical and scientific use.

In conclusion, given the combination of medical and psychiatric problems the patient with catatonia often presents, the psychiatrist should have a central position in the differential diagnosis and choice of treatment of these disorders. As recently stated by Klosterkötter, today, diagnostic procedures in psychiatry do not differ essentially from those used in the days of Wernicke and Kraepelin.[62] ■

1. Wernicke C, *Grundriss der Psychiatrie in klinischen Vorlesungen*, Leipzig: Thieme, 1900.
2. Bonhoeffer K, [The exogenous reaction types], *Arch Psychiatr Nervenkr*, 1917;58:50–70.
3. Fink M, Shorter E, Taylor MA, Catatonia is not schizophrenia: Kraepelin's error and the need to recognize catatonia as an independent syndrome in medical nomenclature, *Schizophr Bull*, 2010;36:314–20.
4. Pilcz A, *Die periodischen Geistesstörungen, Eine klinische Studie*, Jena: Fischer, 1901.
5. Gjessing R, [Contributions to the knowledge of the pathophysiology of the catatonic stupor I and II], *Arch Psychiatr Nervenkr*, 1932;96.319–473.
6. Gjessing LR, A review of periodic catatonia, *Biol Psychiatry*, 1974;8:23–45.
7. Ey H, *Études Psychiatriques: Structure des Psychoses Aiguës et Déstructuration de la Conscience*, Paris: Desclée de Brouwer et Cie, 1954.
8. Peralta V, Cuesta MJ, Motor features in psychotic disorders I. Factor structure and clinical correlates, *Schizophr Res*, 2001;47:107–16.
9. Peralta V, Cuesta MJ, Motor features in psychotic disorders II. Development of diagnostic criteria for catatonia, *Schizophr Res*, 2001;47:117–26.
10. Sienaert P, Rooseleer J, De Fruyt J, Measuring catatonia: a systematic review of rating scales, *J Affect Disord*, 2011;135:1–9.
11. Bush G, Fink M, Dowling F, et al., Catatonia. I. Rating scale and standardized examination, *Acta Psychiatr Scand*, 1996;93:129–36.
12. Pfuhlmann B, Stöber G, The different conceptions of catatonia: historical overview and critical discussion, *Eur Arch Psychiatry Clin Neurosci*, 2001;251:S1–7.
13. Leonhard K, *Classification of endogenous psychoses and their differentiated etiology*, 2nd revised and enlarged edition, Vienna/New York: Springer, 1999.
14. Andreasen NC, DSM and the death of phenomenology in America: An example of unintended consequences, *Schizophr Bull*, 2007;33:108–12.
15. Kleist K, [The structure of the neuropsychiatric disorders], *Monatsschr Psychiatr Neurol*, 1953;125:526–54.
16. Leonhard K, *Aufteilung der endogenen Psychosen*, Berlin: Akademie-Verlag, 1957.
17. Kleist K, *Untersuchungen zur Kenntnis der psychomotorischen Bewegungsstörungen bei Geisteskranken*, Leipzig: Klinkhardt, 1908.
18. Kleist K, *Gehirnpathologie*, Leipzig: Johann Ambrosius Barth-Verlag, 1934.
19. Kleist K, [The catatonias], *Nervenarzt*, 1943;16:1–10.
20. Leonhard K, [Towards the subdivision and genetic biology of schizophrenia. First section: the 'typical' sub-forms of catatonia], *Allg Z Psychiatr*, 1942;120:1–23.
21. Leonhard K, [Towards the subdivision and genetic biology of schizophrenia. Second section: combined-systematic and periodic catatonia], *Allg Z Psychiatr*, 1942;121:1–35.
22. Lohr JB, Wisniewski AA, Catatonia. In: Lohr JB, Wisniewski AA (eds), *Movement disorders: a neuropsychiatric approach*, New York: Guilford Press, 1987;201–27.
23. Rosebush PI, Hildebrand AM, Furlong BG, et al., Catatonic syndrome in a general psychiatric inpatient population: frequency, clinical presentation, and response to lorazepam, *J Clin Psychiatry*, 1990;51:357–62.
24. Franzek E, Beckmann H, Reliability and validity of the

Leonhard classification tested in a five year follow-up study of 50 chronic schizophrenics. In: Ferrero FP, Haynal AE, Sartorius N (eds), *Schizophrenia and affective psychoses. Nosology in contemporary psychiatry*, New York: John Libbey, 1992;67–72.
25. Pfuhlmann B, Franzek E, Stöber G, et al., On interrater reliability for Leonhard's classification of endogenous psychoses, *Psychopathology*, 1997;30:100–5.
26. Stöber G, Saar K, Rüschendorf F, et al., Splitting schizophrenia: periodic catatonia susceptibility locus on chromosome 15q15, *Am J Hum Genet*, 2000;67:1201–7.
27. Stöber G, Seelow D, Rüschendorf F, et al., Periodic catatonia: confirmation of linkage to chromosome 15 and further evidence for genetic heterogeneity, *Hum Genet*, 2002;111:323–30.
28. Stöber G, Reis A, Periodic catatonia. In: Lang F (ed), *Encyclopedia of Molecular Mechanisms of Disease*, Heidelberg: Springer, 2009;1615–6.
29. Wing L, Gould J, Severe impairments of social interaction and associated abnormalities in children: epidemiology and classification, *J Autism Dev Disord*, 1979;9:11–29.
30. Kumbier E, Domes G, Herpertz-Dahlmann B, et al., [Autism and autistic disorders], *Nervenarzt*, 2010;81:55–65.
31. Wing L, Shah A, Catatonia in autistic spectrum disorders, *Br J Psychiatry*, 2000;176:357–62.
32. Hare DJ, Malone C, Catatonia in autistic spectrum disorders, *Autism*, 2004;8:183–95.
33. Dhossche DM, Shah A, Wing L, Blueprints for the assessment, treatment, and future study of catatonia in autism spectrum disorders, *Int Rev Neurobiol*, 2006;72:267–84.
34. Leonhard K, [On catatonia in children], *Psychiatr Neurol Med Psychol* (Leipz), 1960;12:1–12.
35. Verhoeven WMA, Kleefstra T, Egger JIM, Behavioral phenotype in the 9q subtelomeric deletion syndrome: a report about two adult patients, *Am J Med Genet B Neuropsychiatr Genet*, 2010;153B:536–41.
36. Verhoeven WMA, Egger JIM, Vermeulen K, et al., Kleefstra syndrome in three adult patients: further delineation of the behavioural and neurological phenotype shows aspects of a neurodegenerative course, *Am J Med Genet A*, 2011;155A:2409–15.
37. Verhoeven WMA, Tuinier S, Prader-Willi syndrome: atypical psychoses and motor dysfunctions, *Int Rev Neurobiol*, 2006;72:119–30.
38. Sedel F, Baumann N, Turpin JC, et al., Psychiatric manifestations revealing inborn errors of metabolism in adolescents and adults, *J Inherit Metab Dis*, 2007;30:631–41.
39. Fink M, Taylor MA, The differential diagnosis of catatonia. In: Fink M, Taylor MA (eds), *Catatonia: A Clinician's Guide to Diagnosis and Treatment*, Cambridge: Cambridge University Press, 2003;71–113.
40. Guggenheim FG, Babigian HM, Catatonic schizophrenia: epidemiology and clinical course, *J Nerv Ment Dis*, 1974;158:291–305.
41. Hare E, Schizophrenia as a recent disease, *Br J Psychiatry*, 1988;153:521–31.
42. Stompe T, Ortwein-Swoboda G, Ritter K, et al., Are we witnessing the disappearance of catatonic schizophrenia?, *Compr Psychiatry*, 2002;43:167–74.
43. Meyer PS, Bond GR, Tunis SL, et al., Comparison between the effects of atypical and traditional antipsychotics on work status for clients in a psychiatric rehabilitation program, *J Clin Psychiatry*, 2002;63:108–16.
44. Woodward ND, Purdon SE, Meltzer HY, et al., A meta-analysis of neuropsychological changes to clozapine, quetiapine and risperidone in schizophrenia, *Int J Neuropsychopharmacol*, 2005;8:457–72.
45. Taylor TL, Killaspy H, Wright C, et al., A systematic review of the international published literature relating to quality of institutional care for people with longer term mental health problems, *BMC Psychiatry*, 2009;9:55.
46. Kurzban S, Davis L, Brekke JS, Vocational, social, and cognitive rehabilitation for individuals diagnosed with schizophrenia: a review of recent research and trends, *Curr Psychiatry Rep*, 2010;12:345–55.
47. Fink M, Catatonia: a syndrome appears, disappears and is rediscovered, *Can J Psychiatry*, 2009;54:437–45.
48. Ungvari GS, Caroff SN, Gerevich J, The catatonic conundrum: evidence of psychomotor phenomena as a symptom dimension in psychotic disorders, *Schizophr Bull*, 2010;36:231–8.
49. Van der Heijden FMMA, Tuinier S, Pepplinkhuizen L, et al., Catatonia: the rise and fall of an intriguing psychopathological dimension, *Acta Neuropsychiatr*, 2002;14:111–6.
50. Van der Heijden FMMA, Tuinier S, Arts NJM, et al., Catatonia: disappeared or under-diagnosed?, *Psychopathology*, 2005;38:3–8.
51. Starkstein SE, Petracca G, Tesón A, et al., Catatonia in depression: prevalence, clinical correlates and validation of a scale, *J Neurol Neurosurg Psychiatry*, 1996;60:326–32.
52. Krüger S, Bagby RM, Höffler J, et al., Factor analysis of the catatonia rating scale and catatonic symptom distribution among four diagnostic groups, *Compr Psychiatry*, 2003;44:472–82.
53. Bräunig P, Prevalence and clinical significance of catatonic symptoms in mania, *Compr Psychiatry*, 1998;39:33–46.
54. Dunayevich E, Keck PE, Prevalence and description of psychotic features in bipolar mania, *Curr Psychiatry Rep*, 2000;2:286–90.
55. Kakooza-Mwesige A, Wachtel LE, Dhossche DM, Catatonia in autism: implications across the life span, *Eur Child Adolesc Psychiatry*, 2008;17:327–35.
56. Billstedt E, Gillberg C, Gillberg C, Autism after adolescence: population-based 13- to 22-year follow-up study of 120 individuals with autism diagnosed in childhood, *J Autism Dev Disord*, 2005;35:351–60.
57. Ohta M, Kano Y, Nagai Y, Catatonia in individuals with autism spectrum disorders in adolescence and early adulthood: a long-term prospective study, *Int Rev Neurobiol*, 2006;72:41–54.
58. Leonhard K, *Grundriss der Psychiatrie*, Stuttgart: Enke, 1946.
59. Verhoeven WMA, Tuinier S, Van der Burgt I, Top-down or bottom-up: contrasting perspectives on psychiatric diagnoses, *Biologics*, 2008;2:409–17.
60. Heckers S, Tandon R, Bustillo J, Catatonia in the DSM. Shall we move or not?, *Schizophr Bull*, 2010;36:205–7.
61. Aboraya A, Recommendation for DSM-V: A proposal for adding causal specifiers to Axis-I diagnoses, *Psychiatry (Edgmont)*, 2010;7:24–8.
62. Klosterkötter J, Problems in psychiatric classification, *Fortschr Neurol Psychiatr*, 2011;79:265–6.