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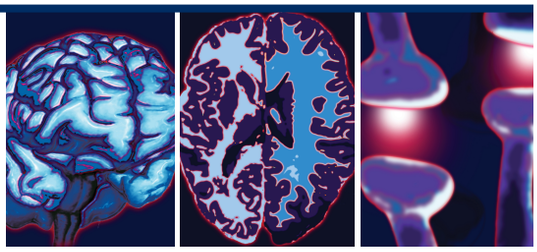

New DSM-5 category 'unspecified catatonia' is a boost for pediatric catatonia: review and case reports

Dirk M Dhossche*[1], Michal Goetz[2], Gabor Gazdag[3,4] & Pascal Sienaert[5]

Practice points

- Pediatric catatonia occurs regularly in patients with autistic and developmental disorders, tic disorders and Tourette's syndrome, and various other disorders outside of major psychotic, affective and medical disorders.
- Pediatric catatonia is a treatable syndrome, regardless of any underlying disorders, and, like in adults, its primary treatments are benzodiazepines and electroconvulsive therapy.
- Pediatric catatonia is marred by poor recognition and treatment delays or no specific treatment at all.
- The creation of a new category 'unspecified catatonia' in DSM-5 aspires to increase early recognition and appropriate treatment of catatonia in patients without diagnosable major psychotic, affective or medical disorders.
- Unspecified catatonia may be particularly beneficial to boost recognition and treatment of catatonia in children and adolescents.
- Unspecified catatonia should be considered when catatonic symptoms develop in children and adolescents with autistic and other developmental disorders, autoimmune encephalitides, tic disorders (with or without self-injury) and Tourette's syndrome, Kleine–Levin syndrome, post-traumatic reactions, pervasive refusal syndrome and various other disorders.
- The aspirations of unspecified catatonia to reduce morbidity and mortality and to increase further research are bound to be tested in the field of pediatric catatonia over the next few years.

[1]Department of Psychiatry, University of Mississippi Medical Center, 2500 North State Street, Jackson, MS 39216, USA
[2]Department of Child Psychiatry, University Hospital Motol, Charles University in Prague, 2nd Faculty of Medicine, V Uvalu 84, Prague 5, 15006, Czech Republic
[3]Center for Psychiatry & Addiction Medicine, Szent István & Szent Laszló Hospitals, Gyali Utca 17–19, 1097 Budapest, Hungary
[4]Department of Psychiatry & Psychotherapy, Faculty of Medicine, Semmelweis University, Balassa Utca 6, 1083 Budapest, Hungary
[5]Department of Psychiatry, University Psychiatric Center – Catholic University of Leuven, Campus Kortenberg, Leuvensesteenweg 517, 3070 Kortenberg, Belgium
*Author for correspondence: Tel.: +1 601 984 5805; Fax: +1 601 984 6965; dirkdhossche@gmail.com

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SUMMARY The creation in DSM-5 of a new category 'unspecified catatonia' aspires to improve diagnosis and treatment of catatonia in patients of all ages without diagnosable major psychotic, affective or medical disorders. Unspecified catatonia may be particularly beneficial in boosting recognition and treatment of pediatric catatonia that often occurs outside these disorders. Until now, pediatric catatonia has been poorly recognized. Benzodiazepines and electroconvulsive therapy are underutilized in pediatric catatonia, yet are safe and effective treatments without the risk of worsening catatonia or precipitating neuroleptic malignant syndrome. Unspecified catatonia should be considered when catatonic symptoms are present in children and adolescents with autistic and other developmental disorders, autoimmune encephalitides, tic disorders (with or without self-injury) and Tourette's syndrome, Kleine–Levin syndrome, post-traumatic reactions, pervasive refusal syndrome and other disorders. The aspirations of the 'unspecified catatonia' category to reduce morbidity and mortality and to increase further research are bound to be tested in the field of pediatric catatonia over the next few years. Changes in DSM-5 regarding the classification of catatonia are reviewed. Case reports are presented to illustrate the use of the new category, unspecified catatonia, in children and adolescents.

Catatonia, a potentially life-threatening but treatable syndrome that warrants prompt diagnosis and treatment [1–3], also occurs in children and adolescents [4–6] with a variety of associated disorders **(Box 1)**. Adult catatonia is often associated with major affective and psychotic disorders, yet these disorders are thought to be less frequent in pediatric patients. However, it is an important finding that catatonia is found in 12–17% of adolescents and young adults with autism spectrum disorders. Pediatric catatonia also emerges in patients with other developmental disorders, tic disorders, including Tourette's syndrome, and a variety of other disorders. Estimates of the frequency of catatonia in some of these disorders are lacking. Prevalence studies of pediatric catatonia since 1996 are listed in **Table 1**. Estimates vary widely across studies and are likely due to differences in study samples. Overall, there is evidence supporting that catatonia may not be as rare in younger patients as previously thought, at least in selected diagnostic groups and when applying uniform assessments **(Table 1)**.

Box 1. Child and adolescent disorders associated with catatonia.

Developmental disorders
- Autistic disorders [38–41]
- Childhood disintegrative disorder [9,42]
- Mental retardation [4] including Down's syndrome [43]
- Prader–Willi syndrome [44]

Medical and neurological disorders
- Catatonia due to a general medical condition (brain structural damage, seizures, metabolic, endocrine and autoimmune disorders) [45–48]

Psychiatric disorders
- Psychotic disorders [49]
- Mood disorders [49]
- Substance-induced disorders [45]
- Medication-induced movement disorder [50]
- Tic disorder and Tourette's syndrome [51,52]

Other disorders and conditions
- NMDAR encephalitis [17,53,54], pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections [46], encephalitis lethargica [55] and other aseptic encephalitides [56]
- Kleine–Levin syndrome [9]
- Psychogenic catalepsy [57]
- Anaclitic depression [58]
- Pervasive refusal syndrome [37]

Clinical experience and case reports support benzodiazepines and electroconvulsive therapy (ECT), including maintenance ECT, as safe and effective treatments for pediatric catatonia that do not carry the risk for precipitating neuroleptic malignant syndrome (NMS), but controlled studies are lacking. From our clinical experience and case reports, we propose that catatonia should be suspected early on when psychomotor retardation, agitation or other motor symptoms are prominent. A medical work-up should include blood work with basic hematologic and metabolic measures, comprehensive drug testing, brain imaging, autoimmune antibodies (lupus, pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections and anti-NMDAR encephalitis) and other tests guided by clinical examinations. In our experience, antipychotics should be avoided due to the risk of worsening catatonia or precipitating NMS. A rating scale can be used to assist diagnosis and estimate severity. A lorazepam test should be considered for diagnostic validation of catatonia. If catatonia improves considerably after a 1- or 2-mg test dose (*per os*,

Table 1. Prevalence studies of pediatric catatonia since 1996.

Study (year)	Sample size (n)	Design, sample population	% with catatonia	Ref.
Moise and Petrides (1996)	13	Retrospective, ECT	46	[59]
Wing and Shah (2000)	506	Prospective, autism	17	[38]
Thakur *et al.* (2003)	198	Prospective, psychiatric clinics	18	[49]
Cohen *et al.* (2005)	4976	Prospective, psychiatric clinics	0.6	[60]
Billstedt *et al.* (2005)	120	Prospective, autism	12	[61]
Ohta *et al.* (2006)	69	Prospective, autism	12	[62]
Consoli *et al.* (2009)	199	Meta-analysis, ECT	6	[33]
Ghaziuddin *et al.* (2012)	101	Retrospective, at-risk inpatients	18	[63]
Goetz and Dhossche	92	Retrospective, first-break adolescent psychosis	33	[Goetz M, Dhossche DM, Unpublished Data]

ECT: Electroconvulsive therapy.

intramuscularly or, ideally, intravenously [iv.]) of lorazepam, we continue lorazepam and titrate rapidly for optimal response and maintenance of improvement, often to doses between 10 and 20 mg per day. If there is no response, bilateral ECT becomes an option. Based on our experience, we advocate the use of bilateral (bitemporal or bifrontal) electrode placement as the most effective method. The number and frequency of treatments is often higher in the treatment of catatonia than in affective disorders. Sometimes, treatments are applied daily for 3–5 consecutive days in severe cases. We recommend courses of at least ten to 20 treatments before substantial improvements can be expected. Acute ECT is often followed by continuation of ECT to avoid relapse. After a few more treatments, some patients do not require any further treatment. Others require maintenance ECT to avoid relapse. Alternatively, associated psychiatric or medical conditions may require separate treatment.

These findings are imminently relevant for classification where catatonia has previously been restricted to sections of the psychotic, affective or medical disorders. In DSM-IV, catatonia features as a subtype of schizophrenia, as a specifier (without a separate code) of primary mood disorder (bipolar I disorder and major depressive disorder), and as a separate category 'mental disorder due to a general medical condition' [7].

DSM-5 has recently been published and catatonia features in three main categories: catatonia associated with another mental disorder (code 293.89); catatonic disorder caused by another medical condition (code 293.89); and unspecified catatonia (codes 781.99 and 293.89) [8]. All three diagnoses use the same definition of catatonia requiring that three out of 12 criteria are present. The catatonic subtype of schizophrenia has been deleted, five more catatonia specifiers have been added in addition to those already existing for six primary diagnoses of affective disorder and these specifiers concern schizophrenia, schizoaffective disorder, schizophreniform disorder, brief psychotic disorder) and substance-induced disorders

A new passage in DSM-5 highlights the growing support for NMS as the drug-induced form of malignant catatonia: "Catatonia can be a side effect of medication. Because of the seriousness of the complications, particular attention should be paid to the possibility that catatonia is attributable to NMS. In rare instances, individuals with schizophrenia or a mood disorder may present with malignant catatonia, which may be indistinguishable from NMS. Some investigators consider NMS to be a drug-induced form of malignant catatonia" [7].

Unspecified catatonia aspires to document the presence of catatonia outside the major psychotic and affective diagnoses that have a catatonia specifier, in situations where the underlying medical or psychiatric condition may not be identified initially yet the primary presentation of catatonia warrants acute treatment and in situations where there is no associated major affective or psychotic disorder (e.g., in patients with autistic disorders). The creation of the unspecified catatonia category purports to be the safest approach to ensure proper diagnosis and treatment of this syndrome in patients of all ages and the best approach to promote research [6,8]. In this article, we show that this new category may be particularly beneficial to increase early recognition and appropriate treatment of pediatric catatonia.

Pediatric catatonia suffers from poor recognition & treatment delays

Until now, pediatric catatonia has been poorly recognized [9]. Although a principal culprit for this has been remedied to some extent in DSM-5, that is, the total lack of an independent status of catatonia in psychiatric classification, several other barriers remain. They include:

- Unfamiliarity with the diagnosis and treatment of catatonia;
- The effects of overshadowing, that is, the false attribution of catatonic symptoms to another disorder both in children with autistic or other developmental disorders, and in children with complex and poorly understood medical or neurological entities, such as idiopathic encephalopathy, aseptic encephalitis or anti-NMDAR encephalitis [9];
- The segregation of severely ill psychiatric patients and patients with developmental disorders in long-term facilities;
- The perceived lack of effective treatments for catatonia;
- The neglect of physical and neurological examination by (child) psychiatrists.

Another pervasive misunderstanding concerns the role of psychogenic, social or traumatic factors in the development of catatonia that has been viewed increasingly as a purely 'organic' disorder or brain disorder. A case in point is the tendency to view those with catatonia as suffering from autoimmune, limbic encephalitis, for example, anti-NMDAR encephalitis when anti-NMDAR antibodies are found, leaving little or no role for psychogenic, social or traumatic risk factors. However, the literature clearly supports the view that deprivation, abuse and trauma can precipitate catatonia in children, adolescents and adults [10]. Conversely, catatonic symptoms may be falsely attributed to various psychogenic disorders or syndromes related to deprivation, abuse or trauma, and labeled as anaclitic depression, quasi-autism after early deprivation, reactive psychosis, post-traumatic stress disorder (with psychosis), dissociative disorders or pervasive refusal syndrome (PRS) [10]. Children (and adults) with symptoms of catatonia should, therefore, be assessed for traumatic and abusive events in family and broader environments in addition to medical causes of catatonia.

Diagnostic and therapeutic errors of omission are also instrumental in poor recognition of pediatric catatonia given the considerable stigma and ambivalence surrounding high-dose benzodiazepine treatment and pediatric ECT in the general and medical community [11]. While errors of commission, such as operating on the wrong knee, may be more blatant, errors of omission undoubtedly have an enormous but more covert impact on patient outcomes including or especially in psychiatry [12]. Such errors in pediatric patients concern the failure or avoidance to diagnose a perceived high-risk diagnosis such as catatonia, or withholding high-dose benzodiazepines or ECT, treatments that are perceived as high risk and laden with stigma. Other deterrents are legal barriers to access pediatric ECT [11,13].

Children and adolescents with catatonia nowadays often follow convoluted diagnostic and treatment trajectories. In our practices, we often observe that lorazepam and other benzodiazepines are prescribed fortuitously for catatonic patients on an as-needed basis for 'agitation', not for catatonia, yet are found to allow the patient to start communicating, eating and drinking for a few hours. The dose prescribed is usually low and for too short a period, resulting in incomplete resolution of catatonia. Another common strategy is to prescribe antidepressants or antipsychotics in order to alleviate any possible underlying affective or psychotic disorders of which catatonia is a manifestation. However, in our experience, it is more effective to treat catatonia first and vigorously as the primary source of psychiatric impairment, and to postpone other treatments until catatonia resolves and residual symptoms are assessed to guide further treatment selection. Often there is a delay in starting ECT when benzodiazepines are not or only partially effective. When ECT is finally pursued, it very often proves to be the definitive treatment for catatonia whose symptoms were clearly present in the early stages of illness.

Unspecified catatonia not elsewhere classified is a boost for pediatric catatonia

The decision to create a separate DSM-5 category – unspecified catatonia – is significant and may be the best for 'the sake of the children' [5], especially patients with autism and other developmental disorders and pediatric patients with a wide range of disorders other than the major affective or psychotic disorders associated

with catatonia (Box 1). Unspecified catatonia should allow experience to refine the place of pediatric catatonia in clinical care and to offer the primary treatments for catatonia to a greater number of pediatric patients.

Studies in adults support a response rate of catatonia of approximately 80% to benzodiazepines and an almost universal response to ECT in those who fail with benzodiazepines [1,2]. Similar studies have not been carried out in children and adolescents. Prompt recognition of catatonia, and in some cases treatment with ECT, is likely to prevent medical complications, such as deep vein thrombosis, pulmonary emboli [14,15], dehydration, malnutrition, physical exhaustion and death.

The following sections describe case reports of children and adolescents diagnosed with autism, tic disorder, Kleine–Levin syndrome (KLS), anti-NMDAR-encephalitis, post-traumatic psychosis and PRS in whom catatonia emerged as a separate syndrome treatable with benzodiazepines and/or ECT. These cases, selected from the literature, illustrate the utility of the new category, unspecified catatonia, to increase early recognition and appropriate treatment in pediatric patients.

Malignant catatonia in an adolescent with autism

A 15-year-old adolescent previously diagnosed with high-functioning autism slowly developed full-blown catatonia. Initially, his symptoms of increased slowness of movements, decreased speech and muteness, inability to complete tasks, muscle stiffness, posturing, waxy flexibility, refusal to eat and urinary incontinence were attributed to autism and cognitive disability. On psychiatric examination, there were no signs of affective or psychotic pathology, and a diagnosis of catatonia was made only after 10 months of onset of clear catatonic symptoms. His condition had worsened to the point of the need for cardiorespiratory monitoring due to severe autonomic instability. Coverage for ECT was denied by his insurance carrier based on the absence of a psychotic or affective illness. The parents of the patient opted to start ECT nonetheless, and all catatonic symptoms remitted after 15 bilateral ECT sessions followed by maintenance ECT for several months. Exhaustive subsequent psychiatric evaluations failed to reveal any hint of affective or psychotic pathology. This patient presented with malignant catatonia alone, and lack of rapid diagnosis and treatment nearly cost him his life [16].

■ Comment

In future cases, the coded listing of unspecified catatonia in DSM-5 should avoid the administrative difficulties of obtaining coverage for ECT (especially in the USA) because the patient has neither a defined major psychotic, affective nor medical illness.

Catatonia in tic disorder with self-injury

A 17-year-old adolescent with normal development was brought to the emergency room because of gradual onset of tic-like spitting, facial tics and grimacing, repetitive hand washing, lip smacking, face slapping, head banging and repetitive finger flexing. These symptoms had worsened over the course of 4 months. On examination, he was withdrawn with a depressed mood, answered questions in a whispering voice and expressed suicidal ideas. Affect was restricted and eye contact was poor. Thought processes were ruminative and obsessive. The patient was admitted to the general hospital where a medical work-up and head computerized tomography were normal. He was transferred to the psychiatric inpatient unit where ECT was started after trials of antipsychotics combined with antidepressants failed. Full resolution of most affective and psychomotor symptoms, including tics and self-injurious behavior, occurred after two bilateral ECT treatments. Before stopping acute ECT, the patient was started on haloperidol 5 mg daily, benzotropine 1 mg twice daily, bupropion 100 mg daily and modafanil 100 mg daily. The patient was then discharged but continued to receive ECT on an outpatient basis, at first every 2 weeks, and later once a month, over a period of 1.5 years. The patient dropped out of treatment for 5 months, but relapsed with reappearance of tics and depressed mood, despite compliance with haloperidol, bupropion and modafanil. Outpatient ECT was restarted with resolution of symptoms. To date, he has received 33 bilateral treatments since the start of his illness. He attends school and is free of psychiatric symptoms. He continues with maintenance treatments every 2 weeks in combination with the above-mentioned medications for relapse prevention [17].

■ Comment

A remarkable feature in this case is that tics emerged together with classical catatonic

symptoms such as psychomotor retardation, stereotypies and grimacing, and that all symptoms, including self-injurious tics, responded to ECT. Motor and vocal tics are often self-injurious and emerge prominently in some patients with catatonia and underlying affective, psychotic or autistic disorders [13]. In some cases, tics with or without self-injury may be signs of catatonia. Therefore, patients with tics, Tourette's syndrome or intractable self-injury [18,19] warrant assessment for catatonia. If catatonia is present, unspecified catatonia may be diagnosed in conjunction with tic disorder, stereotypic movement disorder or another disorder if applicable, and ECT should be considered as a safe alternative to pharmacotherapy, psychosurgery or invasive brain stimulation in the treatment of severely disabling and, at times, life-threatening tics or self-injury.

Catatonia in KLS

A 13-year-old boy with normal development and without a personal or family psychiatric history was brought to the emergency room with a 3-month history of altered behavior and responsiveness following a flu-like illness. The mother reported the occurrence of 10-day-long episodes of bizarre behavior, decreased speech and eating, confusion, mild aggression and agitation, and episodes of hypersomnolence lasting up to 24 h. While not incontinent, he would urinate in unusual places. After each 10-day episode, the patient resumed normal function and remembered little of his behavior other than feeling 'odd.' The patient was admitted to the pediatric hospital for a neurological work-up, including comprehensive laboratory studies and brain MRI. These tests were within normal limits, and he also had a normal awake and sleep EEG. He was diagnosed with KLS. After failed trials of anticonvulsants and stimulants a psychiatric consult was requested. On examination, the patient was unresponsive to verbal commands, and displayed echolalia and stereotypies. As criteria for catatonia were met, a test dose of 1 mg of lorazepam was administered by mouth. Within the next few hours, the patient began to speak normally and regained normal behaviors and cognition. He was discharged from the hospital the following day. Three relapses over the next 6 months were cut short by administering 1 mg of lorazapam two or three times per day [9].

■ Comment

KLS is a poorly understood syndrome, occurring mostly in male adolescents, that is characterized by recurrent episodes of excessive sleep and behavioral abnormalities, such as hyperphagia or hypersexuality, in which altered diencephalic function is considered a central feature [20]. Patients have normal alertness, cognitive functioning and behavior between the episodes. One of us (Dhossche) previously proposed that KLS is a type of episodic adolescent-onset catatonia based on the symptom overlap between KLS and catatonia and on the excellent response of all symptoms, including 'hypersomnia', to lorazepam [9]. The diagnosis of unspecified catatonia will be instrumental in future cases in clarifying the relationship between KLS and catatonia and treatment response to benzodiazepines.

Catatonia in pediatric anti-NMDAR encephalitis

A 14-year-old girl became acutely psychotic without clear precipitants, and without a previous medical or psychiatric history. She experienced one seizure, but her EEG showed frontal slowing and no epileptic activity. Over the next week, full-blown catatonia developed and the patient was prescribed lorazepam 1 mg iv. as needed, with some relief. Prednisolone and iv. immunoglobulin were started for presumed autoimmune encephalitis. After 6 weeks of the illness, cerebrospinal fluid was found to be positive for anti-NMDAR antibodies. Full-body imaging studies showed no malignancies. The patient was started on risperidone 1 mg every 8 h as needed by nasogastric tube. Catatonia worsened and lorazepam 8 mg per day iv. brought temporary, but dramatic relief. Rituximab was tried but the patient developed autonomic instability in addition to motor symptoms of catatonia. ECT was started with significant improvement after seven treatments. ECT was stopped and she was treated next with plasma exchange, followed by rituximab, and then cyclophosphamide in conjunction with risperidone 4 mg per day and lorazepam 0.5 mg per day. She improved slowly over the next 8 months [21].

■ Comment

Many cases of pediatric anti-NMDAR encephalitis and other autoimmune encephalitides meet criteria for catatonia, but the diagnosis remains unacknowledged or catatonia is presented as a mere 'symptom' of underlying encephalitis [22,23]. The case report here is an exception in that catatonia is recognized as a full syndrome and attempts are made to initiate moderate-dose benzodiazepine treatment and ECT.

Pediatric cases with autoimmune encephalitides, including the new anti-NMDAR encephalitis, that meet criteria for catatonia may be better viewed in the future as suffering from unspecified catatonia, for which benzodiazepines and ECT are indicated as first-line treatments. Studies comparing benzodiazepines or ECT, including maintenance ECT, with immune therapies in children, adolescents and adults who meet criteria for catatonia and who test positive for autoimmune antibodies, including anti-NMDAR antibodies, are urgently needed [9] as several of such cases have insufficient or sluggish responses to immune treatments when benzodiazepines or ECT are not used. The benefits of immune treatments once the acute presentation resolves with benzodiazepines or ECT are unclear and need further study.

Post-traumatic catatonia

A 14-year-old girl, previously an excellent high school student without prior psychiatric history, was admitted because of disorganized behavior and decreased need for sleep following significant emotional turmoil caused by cyberbullying by her schoolmate. She was diagnosed with acute polymorphic psychotic disorder and was started on 1 mg of risperidone. A few days later she developed frank catatonia characterized by agitation, increased gesturing of the hands and the fingers, mutism, immobility, posturing, refusal to eat and drink, and incontinence. The girl had to be fed intravenously. Risperidone was stopped and daily bilateral ECT was initiated, enhanced by adjunct amantadine iv. from day 6 of ECT treatment. Catatonia completely resolved after 12 ECT sessions. She was stabilized on low-dose aripiprazole in outpatient treatment. There have been no relapses during the 2-year follow-up and she successfully resumed her high school studies [24].

■ Comment

Traumatic events in childhood and adolescence represent risk factors for the later development of severe mental disorders [25–27]. This case extends findings that adolescent bullying increases the risk for psychotic experiences by linking cyberbullying, an intrusive form of bullying, to onset of catatonia. This supports the notion that severe traumatic events are risk factors for catatonia [28] and that catatonia may represent a primitive evolutionary-based freezing response to a perceived threat [29]. It remains possible that the antipsychotic medication used at the beginning of the hospital stay worsened the patient's clinical status into severe catatonia. Finally, the dynamic of the response to ECT shows that severe catatonia requires intensive and longer treatment courses of ECT than five to seven sessions, which is usually sufficient for depression.

Catatonia in PRS

An 11-year-old girl was admitted because of a 2-month history of refusing to eat any food and drinking of minimal amounts of fluids, lethargy, reduced speech with episodes of mutism and poor self-care. No organic cause was found. Her personal and family history was unremarkable. There were no known medical or traumatic precipitants. Individual and family therapy, as well as antidepressant trials, were prescribed as the patient was thought to suffer from PRS, but with no relief. Supportive measures included a nasogastric feeding tube. Lorazepam was used for extreme agitation, up to 4 mg per day without many overall effects. ECT was recommended for the catatonic presentation of the patient but the parents refused this option. After 18 months of inpatient admission, the patient suddenly and spontaneously started to improve, eat and drink adequately, and communicate verbally with staff and family members. She resumed normal school and social function, and has remained well at a follow-up of 6 years [30].

■ Comment

The authors express contentment and surprise about the unexpected and sudden improvement in the patient yet wonder whether the protracted and complicated 18-month inpatient treatment course could have been shortened if catatonia had been the primary working diagnosis, given the symptom overlap, for which trials of high-dose lorazepam (higher than 4 mg per day) and ECT could have been implemented. Unfortunately, treatment studies in PRS of benzodiazepines and ECT are lacking. Positive effects of these treatments in PRS would support that PRS is a type of catatonia. PRS is a syndrome, absent from the American literature, that was first described by Lask and colleagues in 1991 in four British girls aged 9–14 years as a potentially life-threatening condition characterized by refusal to eat, drink, walk, talk or care for themselves over a period of several months [31]. A literature search found 24 cases published between 1991 and 2006 [32].

The clinical descriptions of contemporary refugee children with PRS are also similar to those of classic catatonia [9,33,34]. It has been previously stated that PRS may be better viewed as pediatric catatonia and should be treated as such [35]. So far, no case of PRS has been treated with high-dose benzodiazepines or ECT as PRS is claimed to be different from catatonia. However, the contention that PRS and catatonia are different conditions is questionable, and may be due to misunderstanding the symptoms and signs of catatonia [9]. Nunn *et al.* have recently proposed to rename PRS as pervasive arousal withdrawal syndrome based on a biological model implicating autonomic dysfunction [36], suggesting a role for medications with beneficial effects on the autonomic nervous system and GABAergic system (but without any mention of benzodiazepines or ECT, known to enhance GABAergic function and reduce autonomic dysfunction). The authors maintain that PRS is different from catatonia – "in that, although in PRS there is mutism, there is an absence of unresponsiveness, stereotypies and waxy flexibility and the capacity to engage with the patient remains, often combatively, intact." It is of note that stereotypies and waxy flexibility never were obligatory symptoms of catatonia and that in DSM-5, presence of three of 12 catatonic symptoms meets criteria for catatonia. It is arguable that there is no unresponsiveness in patients with PRS as many cases are portrayed as very withdrawn, stuporous and without spontaneous movements [37]. The capacity to engage with catatonic patients is preserved at certain times as the severity of catatonia often fluctuates during the course of illness. Unfortunately, little rapprochement is found in the literature on PRS toward a unifying framework with catatonia and its treatments. Hopefully, the new unspecified catatonia category will be useful in future cases to clarify the psychopathological presentation of PRS in relation to catatonia in order to optimize treatment strategies in this serious condition.

Conclusion & future perspective

The new DSM-5 unspecified catatonia category aims to reduce morbidity and mortality in patients with catatonia and to increase further research. Unspecified catatonia may be particularly useful to increase early recognition and appropriate treatment of pediatric catatonia. As major affective and psychotic disorders are not as frequent in children as in adult patients, pediatric catatonia seems to emerge more often outside these disorders previously rendering the diagnosis of pediatric catatonia, as it occurred in developmental, psychogenic or unspecified medical/neurological conditions as a moot issue. The new DSM-5 unspecified catatonia category should be considered when catatonic symptoms are present in children and adolescents with autistic and other developmental disorders, autoimmune encephalitides, tic disorders (with and without self-injury) and Tourette's syndrome, KLS, post-traumatic reactions, PRS and various other disorders. The aspirations of unspecified catatonia to reduce morbidity and mortality and to increase further research are bound to be tested in children and adolescents in the field of pediatric catatonia over the next few years.

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