Making childhood catatonia visible, separate from competing diagnoses

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The prevailing belief in the rarity of pediatric catatonia is incorrect. The condition was frequently diagnosed in the past, but under other labels. In the remote past, catalepsy and stupor were diagnostic terms for some symptoms of what Karl Kahlbaum described in 1874 as ‘catatonia’. However, even after Kahlbaum, catatonia in children was often submerged by global competing diagnoses such as ‘schizophrenia’, ‘autism’, and ‘hysteria’. Recently, however, catatonia in children has begun to emerge as an independent diagnosis.

Accurate diagnosis is important because catatonia responds readily to well-defined treatments such as benzodiazepines and electroconvulsive therapy, whereas the larger diagnoses that have for many years concealed it are often less responsive to treatment.

The concept of catatonia dates back to the Ancients (1, 2). The signs and symptoms of pediatric catatonia do not differ significantly from those of the adult form. However, the history of catatonia in children is of interest because, for many years it was believed that they did not suffer from catatonic symptoms. The purpose of this article is to show that, historically, catatonia in children was not uncommon. As it was described under other terms, however, its existence has been hidden from previous observers. In 1973, two pediatric researchers asserted, ‘Reports of catatonic episodes in childhood are exceedingly rare in the English-language literature’ (3).

In light of the earlier historical literature, such skeptical assessments are in need of revision. The problem is that catatonic behavior was not formerly called ‘catatonia’. It was called ‘catalepsy’ (meaning waxy flexibility of the limbs), ‘stupor’, or even ‘coma’, depending on the main presentation. This would of course make sense before Kahlbaum coined the term in 1874 (4). However, even afterward, the pediatric equivalent was not acknowledged, and remained a collection of loose symptoms easily submerged within larger entities such as dementia praecox/schizophrenia, hysteria, and autism (see Table 1 for an overview).

In the past, pediatric catatonia manifested itself mainly in the form of ‘hysterical attacks’ of stupor or catalepsy, although catalepsy often entailed stupor as well. Cases are numerous of patients who went into ‘death spells’. Such patients often risked being buried alive. Pierre Pomme, an 18th-century French physician, cites in 1761, the case of a medical colleague, who ‘delayed the funeral services of a girl from the lower classes [une fille du peuple], because her color had not changed at all. And she recovered several hours later. The girl... would already have been buried several times, if we had not been familiar with her attacks of hysterical vapors’ (5). This account is typical of a large body of medical literature from the 18th and early 19th centuries (6).

Catatonia *avant la lettre* – before the label

Part of the waxing and waning of catatonia from view results from fashion, both in diagnosis and symptoms among patients. It is important to understand that cultural suggestion can cause patients to present catatonic symptoms in some epochs, but not in others, whereas changes in diagnostic fashion determine whether physicians make the diagnosis or not.

The two predominant forms of pediatric catatonia have been stupor and catalepsy. Catatonia was called stupor (*stupidité*, *Starrsucht*), when the sensory functions were blocked, and catalepsy when motor symptoms predominated (7). When accompanied by severe mood symptoms, some variant of ‘melancholia’ was often the diagnosis instead. Indeed, this led several authorities to confuse the diagnoses of melancholia and (catatonic) stupor. Kayser–Petersen, in a 1924 review of the literature on ‘depressive disturbances of consciousness’ going back to Antiquity, gives a comprehensive account of stupor without even mentioning catatonia (8). In addition, the early medical literature abounds with patients who had both stupor and catalepsy. These combined forms could be known under either label.

Kahlbaum was thus not the first to describe catatonia in 1874, although he coined the term. Rather, he brought these traditional symptoms into direct association with major psychiatric illness. He also supplied a medically legitimate name for these symptoms after the term ‘catalepsy’ had been downgraded to the equivalent of ‘hysteria’ (6).

In a typical case, from around 1800, a 5-year-old girl had a ‘cataleptic spasm’ at the dinner table: ‘She was highly shocked that her sister had stolen a choice morsel that she herself desired. The hand that she stretched out toward the platter, with her spoon, remained in this position... And even though people spoke loudly to her and shook her firmly, she heard nothing. She moved neither her mouth nor her lips; she walked when she was pushed or taken by the hand; her arm, when it was drawn high or low, or transversally, remained in the same position; you would have thought her a wax statue.’ (9).

Thus, a word of caution is in order. Catalepsy has always been influenced by a cultural climate of suggestion, in which, essentially, patients produce the symptoms expected of them. In an era that associated catalepsy with somnambulism, hypnotism, and other psychological phenomena, non-organic catalepsy cases abounded (10). In the last quarter of the 19th century, hypnosis underwent a revival in medicine. Many cases of childhood catalepsy responded well to hypnosis, and the medical hypnotist Ambroise-Auguste Liébeault, a village doctor in France, prided himself on curing the condition. In December 1860, Liébeault attended a girl of 13 ‘immobile in her bed and without initiative... During the interview, we discovered that her arms retained the position in which they were placed’. He treated her successfully with ‘negative suggestion’. Liébeault (11) was well aware of the climate of ‘suggestion’ that enveloped the young patient.

Even after Kahlbaum defined catatonia in 1874, catalepsy continued as a separate diagnosis. Finally in 1928, Henri Claude, the professor of psychiatry in Paris, and his associate Henri Baruk, completed the process of removing hysteria from catatonia: They distinguished between ‘hysterical catalepsy’ and ‘catatonic catalepsy’, the latter characterized by the ‘less paroxysmic form; by the presence of more complex motor disturbances in which, in addition

to immobility and stupor, may be added negativism and some automatic movements; by the reduced role of suggestion and reactive stress; and, finally, by the existence of severe psychiatric disturbances that accompany and frequently follow the acute event' (12). This distinction was the beginning of the end of 'cultural' catalepsy.

However, not all catalepsy was the product of suggestion. Many young patients with chronic psychoses were also simultaneously 'cataleptic'. 'In many cases of melancholy with stupor', wrote London psychiatrist George Savage in 1876 about youthful inpatients at the Bethlem Royal Hospital, 'one sees a more or less cataleptic state, but with memory and knowledge of what was going on around' (13). The patients he described were in their 20s, not impressionable youngsters, but young men with the chronic psychotic illness of 'melancholica attonita'.

The other important catatonic symptom in children was stupor. Much catatonia was buried under the traditional term 'stupidity', not meaning the state of being stupid, as understood in the English language, but stuporous. In French and Italian, 'stupidité' and 'stupidità' denoted a mind devoid of thought, or, as French observers put it, 'un foyer sans flamme'. In 1794, Italian psychiatrist Vincenzo Chiarugi reported a pediatric case, a girl of 15 who passed from mania to 'a true stupidity', or apparently catatonic stupor, remaining in this state for some time. She had several further such episodes while in hospital, and then was discharged months later 'very healthy of mind' (14).

Pediatric patients with catatonic stupor exhibited on occasion the same cultural suggestibility that was seen in waxy rigidity. However, stupor could also be profoundly organic. This became evident among children with brain damage in the epidemic of encephalitis lethargica between 1916 and 1926 (15) (see below).

The most penetrating form of organicity is genetic transmission, and in some venues 'endemic catalepsy' was inherited within families. The bearers were known as 'spellbound' (die 'Starren' in one German report from 1863) and subject to periodic attacks of rigid posturing lasting a few minutes. In one village, 'People say they can even recognize it in a new-born child, that rolls up its eyeballs during its first bath' (16).

Modern reports as well indicate an organic basis for the infantile syndrome. In the late 1950s, Maurizio De Negri at the University Psychiatric Clinic in Genoa reported a series of catalepsy-catatonia cases in the age group 2–5, an age not readily susceptible to environmental suggestion. It is true that children learn from those around them. However, there is no indication that these children came from 'catatonic' households. Moreover, the cataleptic symptoms often occurred secondarily to organic diseases, such as encephalitis (17).

Catatonia vanishes during the encephalitis epidemic of the 1920s

The encephalitis epidemic of the 1920s left many pediatric victims with hyperactivity and post-Parkinsonian syndromes. However, accustomed to thinking of catatonia as part of 'schizophrenia', even veteran clinicians missed the catatonic signs in their young patients (18). Impressed with the evidence of 'hyperkinesis', clinicians at such elite institutions as

the pediatric neuropsychiatry service of the Charité Hospital in Berlin, overlooked such obvious symptoms of catatonia as negativism, echolalia, stereotypies, and stupor. A 1932 contribution from that service is sometimes seen as one of the earliest clinical descriptions of attention deficit hyperactivity disorder. However, the children themselves were whirling dervishes of catatonia (19).

In the epidemic, catatonia was sometimes diagnosed under such classifications such as 'hyper-pnoeic breathing' and 'peculiar habits'. An example from 1923: one post-encephalitic child 'had developed a striking peculiarity in his gait... While walking along quite normally he would suddenly stop, bring his feet together sharply, jump into the air, and run a few steps, then resume the normal gait once more'. Another postencephalitic child, aged 3, 'developed the very peculiar habit of stopping suddenly in her play, taking a long forced inspiration, holding her breath, and then, with hands clenched and lips firmly closed, straining forcibly as if trying to defecate' (20). Mentions of catatonia were absent.

The epidemic produced two groups of pediatric cases with psychological sequelae, children with severe personality changes and children in stupor and coma. Swiss psychiatrist, JB Jörger, conducted a survey in 1924 among colleagues of their experience with postencephalitic children. The modal age at the time of exposure to the virus was 7–10. Jörger divided the patients into two groups, the 'active', who manifested aggressive and uncontrollable personality changes, and the 'apathetic', with frankly catatonic symptoms, 'sitting there stiff and immobile, like a catatonic' (21).

The encephalitis lethargica epidemic touched off its own epidemic of symptom-attribution to previous infections, which after all are ubiquitous in childhood. In 1934, Eugen Kahn and Louis H Cohen at Yale University proposed that much hyperactivity, ticcing behavior, and general 'bad boy' comportment were the result of post-encephalitic 'brain-stem' changes; interestingly, a number of these pediatric patients exhibited catatonic symptoms, although the authors did not use the term. Case number one, a boy of 8 with a history of 'a severe attack of measles', 'showed a tic-like movement of his neck in which his head was jerkily thrown back; his shoulders moved rather peculiarly... His face seemed rather immobile and there was a slight internal strabismus of the left eye' (22). 'Brain stem changes' would probably have passed from the scene, had it not become the foundation-stone of the view 20 years later that what characterized these presumably 'post-encephalitic' children was attention-deficit and hyperactivity disorder, treatable with Benzedrine (23). The point here is that, once again in a pediatric population, catatonic symptoms had become subsumed under larger diagnoses, real and imagined.

The epidemic of encephalitis lethargica put pediatric psychiatry on the map, simply because the personality changes in the population of 9–19 year-olds were so striking, and so difficult for physicians, families, and society to deal with (24). However, it did not similarly spotlight pediatric catatonia.

Catatonia and hebephrenia

For the study of disease classification, the problem is keeping catatonia visible as an independent entity, rather than seeing it swamped by competing global psychiatric diagnoses such as schizophrenia, autism, hysteria, or such medical-neurological illnesses as encephalitis lethargica. The history of pediatric catatonia may therefore be understood, in part, as periods of clarity alternating with years of diagnostic confusion and nosological murkiness. In 1881, M. Emanuel Fink, at the Würzburg Psychiatric Clinic, recognized the concept of hebephrenia in a pediatric population. [Hecker, a student of Kahlbaum (25), had coined the term hebephrenia in 1871.] ‘I have adduced the last two cases,’ Fink wrote, ‘to illustrate the similarity which catatonia, often erupting in the period of adolescence, offers to hebephrenia’ (26). (For him, hebephrenia was the core of what would later be called ‘schizophrenia’, psychosis erupting in adolescence.)

Some authorities did clearly differentiate between pediatric catatonia and hebephrenia. In 1909, Julius Raecke at the Kiel University Psychiatric Hospital, published the first series of pediatric catatonia cases: His 10 patients were all adolescents and – before the onset of the great epidemics of influenza and encephalitis lethargica of the First World War – the cause was, according to him, ‘an inborn psychic inferiority’ (27).

Catatonia becomes overpowered by ‘schizophrenia’ and other diagnoses

In 1899, in the sixth edition of his famous textbook, Emil Kraepelin proposed abolishing catatonia as an independent diagnosis and made it part of ‘dementia praecox’ (28); in 1908, Eugen Bleuler baptized dementia praecox as ‘schizophrenia’ (29). As a subtype of schizophrenia, catatonia was thought to share the same general course of deterioration into dementia. This subtyping blinded subsequent generations of researchers to the presence of catatonic symptoms in non-schizophrenic illness, especially in mood disorders. It also ensured, after the introduction of effective psychopharmaceutical agents in the 1950s, that patients with catatonia would be treated inappropriately with antipsychotics –which worsens the disease – rather than with electroconvulsive therapy or, after 1960, benzodiazepines, which are effective treatments for catatonia, but not schizophrenia.

The logic of catatonia as a schizophrenic subtype carried the day in pediatric psychiatry as well. August Homburger’s influential 1926 textbook, the founding text of pediatric psychiatry, made catatonic symptoms part of schizophrenia, and, until the advent of ‘autism’ as a pediatric diagnosis in the 1940s, few challenged this classification.

However, catatonic schizophrenia was mainly diagnosed in adolescents. Of 231 juvenile patients admitted to the Zurich cantonal mental hospital between 1870 and 1920, 51 were diagnosed with schizophrenia; of these, 18 had the subtype of ‘catatonia’. In all but two of these patients, onset was in adolescence. (The childhood ‘catalepsies’ in Zurich canton would have increased the numbers; however, they were apparently not admitted to this institution.) (30)

Other diagnoses envelop catatonia

Schizophrenia was not the only diagnosis that annexed catatonia. There was ‘psychasthenia,’ an anxiety state involving phobias and fears, launched by Parisian psychiatrist, Pierre Janet, in 1903 (31). Psychasthenia flourished as an explanation of myriad symptoms, including what later would be called panic disorder, somatoform disorder, and, last but not least, catatonia. Janet postulated that his patients suffered from ‘aboulia,’ or loss of will, which made them unable to control their motor impulses (32).

Among the sturdiest diagnoses of young women was ‘hysteria’, a term that rings across the ages for various seizures, paralyses, and fainting fits that physicians were otherwise unable to explain. In 1723, for example, Johann Storch, a physician in Eisenach, Germany, attended a girl of 17 who had a long series of paroxysms of various kinds. In a typical attack, ‘she closed her eyes, as if she wanted to sleep, got alternately red and pale in the face, lay still and heard nothing. After a quarter hour, one of her arms began twitching, her expression alternated from smiling to sad; then she began to speak in religious and biblical phrases, as if talking with God, pointing her fingers, then started alternating moderate laughter with brooding. After a quarter hour of this, she began speaking again with persons about her, was however not able to open her eyes, then finally came to herself.’ These are signs of catatonia. Over the months, in 1722 and 1723, this young lady had a number of such attacks, which Storch treated with laxatives and blood-letting (33).

Thus ‘hysteria’ often veiled catatonia. Around 1800, Caleb Hillier Parry, a noted physician in the spa town of Bath, England, treated a ‘young lady’ of 14 who, after a display of fireworks, experienced hysterical ‘spasmodic motions of the hands and fingers, and convulsive actions of various parts.’ Furthermore, ‘As she lay on her back... she threw the left arm and body backwards at measured intervals, exactly keeping time with two or three notes which she sang out with a strong and clear voice; and this scene was repeated every day from 11 to 3 at noon, and from 8 to 10 at night... [She could] assign no reason for the particular song so often repeated, but that ‘it was irresistible’’. After she was bled, leeches, and dosed with purgatives, the attacks ceased (34). The voluminous ‘hysteria’ literature is filled with such reports.

Another contemporary diagnostic term, ‘neurasthenia’, described by New York electrotherapist George Beard in 1869, also contained catatonic syndromes (35). A modern version of neurasthenia is the Pervasive Refusal Syndrome, as defined by Bryan Lask and co-workers at the Hospital for Sick Children in London in 1991. It bears all the symptoms of catatonia, but is thought to have psychological causes, like anorexia nervosa or excessive opposition (36).

Catatonia and child development

In the 1930s, new medical interest was aroused in developmental delays; this fresh curiosity highlighted catatonic phenomena in children. At the Harriet Lane Home for Invalid Children and the Henry Phipps Psychiatric Clinic of Johns Hopkins Hospital, Leo Kanner noted in 1934 (37) that many of the children with catalepsy were developmentally delayed. As

Abraham Blau and Samuel Harris Averbuck, on Israel Strauss's neurological service at the Mount Sinai Hospital and the Hebrew Orphan Asylum in New York, put it in 1936, '... The occurrence of cerea flexibilitalis in a child who is somewhat mentally retarded is comprehensible, for in such a child there has probably been a physiological delay in the postnatal evolution of the cortical organization'. The authors found childhood catatonia associated with such neurological signs as a positive Babinski reflex, the inability to walk, and deep tendon-reflex hyperactivity (38).

In 1943, Kanner introduced the diagnosis of 'autism' in this clinical population, and therewith initiated efforts to subsume catatonia under the larger autism label. Many of the patients he described manifested clear catatonic symptoms in addition to their social isolation and uncommunicativeness (39). This same conflation of social isolation and the classical symptoms of catatonia occurred the following year, in 1944, in Hans Asperger's account of 'autistic psychopathies' in childhood (40) Asperger, who also used Eugen Bleuler's term 'autism', penned his own contribution in ignorance of Kanner's work the previous year. These two classic articles began the period of catatonia's submergence in the pool of autism.

Treatment response

A positive response to a distinctive treatment is a way of making catatonia visible. To be sure, there are treatments, such as sodium amytal, to which both hysteria and catatonia respond. However, catatonia does respond differentially to other therapies, such as electroconvulsive therapy, introduced in 1938, and to benzodiazepines, introduced in 1960 and used for the treatment of catatonia in the last two decades of the 20th century (41). Competing diagnoses such as schizophrenia and autism do not, on the whole, respond to these treatments (although ECT can relieve the positive symptoms of schizophrenia). This is an important clinical point.

However, there is an important historical point as well. In the past, catalepsy was responsive to various therapies that we would consider psychological in nature, and the very success of those treatments doubtlessly entrenched catalepsy as a distinctive disease in the medical thinking of the day. The application of electricity, in a non-convulsive manner, had long been used to relieve cases of catatonic stupor and attendant symptoms. In 1788, George Wilkinson, a surgeon in the English town of Sunderland, was called to visit a young female of 28, subject to 'a rigidity and stiffness of the whole body,' becoming 'totally insensible'. A week later, 'An universal spasm, producing a rigidity of the whole frame, took place in a moment, and deprived her of the power of speech and recollection'. When she proved unresponsive to the drugs then available, Wilkinson began treating her 'catalepsy' with spinal applications of 'medical electricity', to which her symptoms yielded. In the months and years ahead, Wilkinson and a colleague continued to administer electricity, and her attacks subsided after each treatment (42). The therapeutic effects of peripheral electricity were almost certainly because of suggestion; however, the point is that this differential responsiveness kept catalepsy visible as a distinctive disorder.

ECT, of course, involves not just the peripheral applications of electricity, but a cerebral seizure. An early report of the use of ECT in pediatric catatonia dates from 1950. In a psychiatric clinic in St-Sébastien, Spain, clinicians admitted a psychotic 13-year-old girl with the symptoms of 'acute catatonia', including severe motor agitation, negativistic thinking, and 'the somatic symptoms that Stauder reported in his [diagnosis of] 'lethal catatonia'. She recovered over a period of several months following 'shock treatments (insulin and electroshock)'. A year later she was well, 'despite a slight intellectual deficit'. (43). The off-hand manner of the report suggests that at this time, the treatment of psychosis in childhood with ECT must have been routine. Indeed, it is the very responsiveness of catatonia to convulsive therapy and benzodiazepines that prompts the rebirth of the diagnosis today. However, a scientifically unjustified cultural reluctance to use electroconvulsive therapy in children has curtailed its application to pediatric catatonia. Swedish clinicians were willing to use 'insulin therapy' and anticonvulsants on a boy of 7 who suffered from severe 'periodic catatonia'. In contrast, ECT? 'We have not given electro-convulsive therapy, as we do not generally use this method in children' (44).

New and old symptoms in catatonia

After the Second World War, catatonic symptoms in infants began to be noticed. This was no longer the typical teenage girls with catalepsy, or the agitated youths of the 1920s with their behavioral syndromes, but 2-year-olds demonstrating such phenomena as waxy rigidity. An early study of three cases involving children from one to three with infectious illnesses, dated from 1953 (45). Thereafter, occasional reports appear, and Klaus-Jürgen Neumärker offers an able overview of the German-language literature (46).

Stupor and waxy rigidity were the two classical symptoms of pediatric catatonia. However, as time went on, the symptom list lengthened, in exactly the same way that behavior such as stereotypies and posturing were added to the symptoms of the adult form.

Medical attention has recently been directed toward self-injury behavior, whether as a result of an increase in such behavior or a shift in clinical interest. In the older case literature, self-injury was not unknown during attacks of catatonic excitement. However, there, it did not occupy a prominent place on the symptom palette. In a 1926 report of six cases of pediatric catatonia from the Psychoneurological Children's Clinic in Moscow, two of the patients exhibited significant symptoms of self-harm, such as head-banging. However, the author's attention was directed at epileptiform behavior, and he mentioned the self-injury without comment (47). The attention of the profession was riveted by the dramatic case of a 16-year-old girl in New York in the 1930s, who avulsed both her eyeballs and pulled out a number of her teeth, secondary to encephalitis lethargica (48).

The recent literature reports self-injury behavior more commonly. An early account linking self-harm to catatonia comes from Moscow in 1963 of seven children from the same extended family who all fell ill around 13, and over the next 5 years developed the symptoms of Kraepelin's hebephrenia, with strong catatonic features. Of interest here are the self-injury symptoms that some members of this cohort display after they become fully psychotic and are admitted to a Moscow mental hospital: 'Their impulsivity grew, often in

connection with aggressiveness. The patients took on contorted [verschroben] poses, froze briefly into fixed positions, and refused food. They became increasingly inaccessible, negativistic.

In this developmental stage of the illness, diurnal variations in their condition became especially conspicuous: mornings and during the day they were lethargic, inactive, and mostly lay about in bed, had contact with no one; in the evening, however, they became loud, non-inhibited, and silly; it came to linguistic and motor excitement. Typical of many of them was their tendency to self-harm: They beat their heads against the wall or the bed-frame, lay in bed so that their necks rested on the bedrail; they pushed on their eyeballs and choked their necks with their hands or a towel, slammed the door on their fingers and so forth' (49). For these Russian clinicians, catatonia was a subcategory of schizophrenia.

The re-emergence of 'pediatric catatonia' as a diagnosis

The great turning point in liberating catatonia from schizophrenia occurred with the work of Karl Leonhard, the pioneering German nosologist who remains, years after his death in 1988, largely unknown outside Central Europe. In 1960, Leonhard argued that virtually the only kind of 'schizophrenia' that occurred in childhood was catatonia. He distinguished among varieties of pediatric catatonia, and said that the other forms of schizophrenia, such as paranoid and hebephrenic, did not exist in childhood at all. Thus, for Leonhard, catatonia, to all intents and purposes, emerged as a distinct illness in the pediatric population (50). In the seventh edition of his textbook on the 'endogenous psychoses', published posthumously in 1995, Leonhard said that, 'One never can find out what is going on inside a child with catatonia. They always seem foreign to us... One may infer nothing from their facial expressions... their faces are like masks'. Leonhard's thinking deviated somewhat from the nosological scheme of his teacher, Karl Kleist (51), who had considered catatonia part of schizophrenia. Leonhard's disciples showed particular interest in Leonhard's distinction between catatonic children who speak readily and those who speak unwillingly (sprechbereite Katatonie and sprachträge Katatonie) (52).

However, curiosity about pediatric 'schizophrenia' and a possible catatonic subtype lingered. In 2005, David Cohen's group at the Pitié-Salpêtrière Hospital in Paris distinguished between non-schizophrenic and schizophrenic catatonia in children, arguing that the former was 'a clinically relevant subgroup that has been understudied' (53). The subsequent work of Cohen's group no longer recognized schizophrenia and focused on catatonia alone (54, 55).

When the diverse symptoms of pediatric distress became codified as 'autism' in the 1990s, catatonic behavior as well became increasingly ascribed to autism. There is, to be sure, some element of catatonia in many autistic patients, and Lorna Wing, psychiatrist and the mother of an autistic daughter who also had some catatonia, has legitimately propagated this view (56, 57). However, on the whole, autism has tended to smother catatonia, and it is a great irony that, no sooner was catatonia rescued from the embrace of schizophrenia than it threatened to disappear into the pool of autism. In fact, if anything the opposite, conclusion would be indicated, and Dhossche has called autism an early expression of catatonia (58).

Today, as we confront once again the revision of the classification of diseases in psychiatry, an unusual opportunity presents itself: rescuing catatonia from the swamp of competing diagnoses and establishing it as an independent illness entity in adults and children. Max Fink and Donald Klein (59) attempted this rescue in 1995, observing that postencephalitic symptoms in children might be evidence of catatonia rather than ‘schizophrenia’. Since then, efforts have accelerated.

Beginning in 2007, one group of researchers began to attribute evident catatonic symptoms to a paraneoplastic syndrome called ‘anti-NMDA receptor encephalitis’ (60). In 2011, Dhossche et al. (61) took the new diagnosis under the microscope, arguing that it was merely yet another label for catatonia.

In 2010, Dhossche and Wachtel wrote of the necessity of separating catatonia, a motor syndrome, from psychosis: ‘Despite warnings that a complete divorce between catatonia and psychosis would be nasty, [we] believe it should be done...’ for the sake of the children. ‘Separating catatonia from psychosis and schizophrenia would improve early diagnosis and treatment of catatonia in children and adolescents, especially those with autistic or developmental disorders’ (62).

The lesson of history here is that the window available for such a separation is likely to be open only briefly. This is important because catatonia responds readily to defined therapies such as benzodiazepines and electroconvulsive therapy, whereas the larger diagnoses that for many years concealed it are often less responsive to treatment.

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Table 1

Diagnostic labels that have historically obscured catatonia as an independent disease

Stupor	
Catalepsy	All mid-19th century and before
Stupidité	
Death spells	
Neurasthenia	1869
Hysteria	Dissociated from catatonia by the 1920s
Hebephrenia	1871
Catatonia	1874
Dementia praecox	1899
Psychasthenia	1903
Schizophrenia	1908
Encephalitis lethargica	1920s
'Brain stem' changes (as a precursor of ADHD)	1934
Autism	1943
Pervasive refusal syndrome	1991
Anti-NMDA receptor encephalitis	2007