Childhood catatonia, autism and psychosis past and present: is there an ‘iron triangle’?

E. Shorter¹ and L. E. Wachtel²

¹Faculty of Medicine, University of Toronto, Toronto, Canada

²Neurobehavioral Unit, Kennedy Krieger Institute, Baltimore, MD, USA

Abstract

Objective—To explore the possibility that autism, catatonia and psychoses in children are different manifestations of a single underlying form of brain pathology – a kind of ‘Iron Triangle’ of symptomatology – rather than three separate illnesses.

Method—Systematic evaluation of historical case literature on autism to determine if catatonic and psychotic symptoms accompanied the diagnosis, as is found in some challenging present-day cases.

Results—It is clear from the historical literature that by the 1920s all three diagnoses in the Iron Triangle – catatonia, autism and childhood schizophrenia – were being routinely applied to children and adolescents. Furthermore, it is apparent that children diagnosed with one of these conditions often qualified for the other two as well. Although conventional thinking today regards these diagnoses as separate entities, the presence of catatonia in a variety of conditions is being increasingly recognized, and there is also growing evidence of connections between childhood-onset psychoses and autism.

Conclusion—Recognition of a mixed form of catatonia, autism and psychosis has important implications for both diagnosis and treatment. None of the separate diagnoses provides an accurate picture in these complex cases, and when given single diagnoses such as ‘schizophrenia’, the standard treatment options may prove markedly ineffective.

Keywords

autistic disorder; catatonia; child psychiatry; schizophrenia; childhood; nosology

Introduction

The little patient lies perhaps for hours or days seemingly in a sort of mystical contemplation, with limbs more or less rigid, or fixed in strange postures; sometimes there is insensibility to impressions, while in other instances vague answers are given, or there is actual incoherent raving.... The example may serve to illustrate how closely related are disorders of the different nervous centres in children, as well as to show the hybrid character

of the diseases presented, and the artificial character of the divisions usually made between them.

English psychiatrist Henry Maudsley on ‘cataleptoid insanity’ in children, 1867 (1).

Modern child psychiatry focuses upon discrete symptom collection and diagnosis of disease based upon established criteria regarding symptom types and their number, duration and impact. The Diagnostic and Statistical Manual IV-TR is the global reference for such diagnostic decision-making, with further changes expected in the upcoming DSM-5. Yet as Henry Maudsley observed, while many patients fall neatly into established categories, others remain enigmatic, bringing into question the possibility of mixed or novel illness states, with consequent impact on both diagnosis and treatment selection.

We begin with a present-day clinical case:

A 16-year-old Caucasian female with prior diagnoses of autism, severe mental retardation and cerebral palsy was admitted to a rehabilitation unit for bowel clean-out after 1 month of decreased appetite, constipation with inability to pass stool over several days, and episodes of crying and howling with repetitive and self-injurious behaviours, which were initially attributed to rectal faecal impaction found on abdominal X-ray. Psychiatric consultation was requested for agitation and self-injury, as well as concern for psychosis given strong family history of paranoid schizophrenia. The patient had never been verbal, and had been cared for by her maternal grandmother since birth. Grandmother reported decreased ability to perform activities of daily living and previously mastered daily tasks; school staff and multiple therapists commented upon massive skill regression over the months prior to admission. The patient had also demonstrated decreased interest in previously enjoyed activities, amotivation and need for extensive prompting, as well decreased oral intake with 15 pound weight loss in 4 months. Grandmother reported intensification of repetitive rocking and self-injurious behaviours, such that for the 2 months prior to admission, the patient had spent most of the day wildly rocking back and forth in either supine or prone positions, flinging her arms and legs back against the walls, or alternatively repetitively throwing herself face down on the bed. At the time of admission, the patient had developed cellulitis of the chin and required IV antibiotic therapy. She had broken several chairs with the force of her rocking, and also had to wear protective elbow and knee padding to prevent worsening limb injury. There was no behavioural change from baseline that raised concern for response to internal stimuli, although the assessment was limited by lack of functional communication.

Episodes of sudden full body tensing were reported, although attributed to attempts to pass stool. The patient was also reported to intermittently clasp others in sudden ‘death grips’ such that her hands had to be pried off the individual. These clasping episodes had no clear antecedent and did not occur in the context of aggression towards others. Staring episodes were reported as well, in addition to unusual dystonic movements. Neurological evaluation did not find any acute aetiology for them, and video EEG documented a staring episode without corresponding seizure activity. Sleep was disturbed with frequent night waking and engagement in repetitive self-injurious behaviour in the middle of the night.

The patient carried a diagnosis of cerebral palsy from birth, but increasing rigidity was reported, with botulin toxin injections pursued but not beneficial and significant decline in ambulation.

The family history was significant for paranoid schizophrenia in the biological mother, maternal uncle and cousin, as well as twin sister who reportedly had multiple psychiatric hospitalizations before age 6 years when she was started on clozapine with resolution of psychosis, but evidence of significant developmental delay.

The patient presented as a thin, dishevelled Caucasian female lying prone in her net-enclosed bed and wearing knee and elbow padding. She was noted to rhythmically and repeatedly lift her body and fling herself against the mattress several times per minute. This was associated with grunting, and continued for 30 min. The patient postured with her arms and hands when touched, and demonstrated bilateral grasp reflex. Face was notable for sustained upward deviated eye gaze. The patient did not respond to greeting, and did not have any verbalizations or vocalizations other than grunting.

The patient was admitted on lithium and olanzapine therapies, which she had been receiving for years for behavioural dyscontrol and suspected psychosis. Catatonic deterioration was diagnosed in this young lady in the context of underlying autism and possible psychotic illness. Partial reduction of olanzapine and initiation of lorazepam allowed for significant reduction in ongoing repetitive self-injury as well as improved sleep and decreased agitation.

This challenging case, not in the least unusual in the world of developmental disorders, is interesting because the patient actually has three diagnoses in today's terms: autism, catatonia and a suggestion of 'schizophrenia', or at least the inference of some kind of psychotic illness.

We are accustomed to thinking of autism, catatonia and schizophrenia in children as separate illnesses. Yet perhaps they are different manifestations of the same underlying disorder, and the mixed form is the true form of the disease as encountered in nature. Schizophrenia and catatonia have usually been described as adult diseases, although paediatric forms of both illnesses are increasingly recognized (2–5). Autism is typically diagnosed in childhood, although retrospective diagnosis in the adult patient is possible. Yet it is often very difficult in children to determine exactly which adult disease they have, with similar difficulty conferring a paediatric diagnosis on an adult, especially when there is significant symptom overlap such as is seen in these three disease entities. Current genetic research suggests that autism and schizophrenia have common roots, although the genetics of catatonia remain uninvestigated (6). A look at the historical literature may clarify our thinking.

The argument of this article is as follows: From the vast undifferentiated stew of paediatric psychopathology, a kind of 'Iron Triangle' of symptoms had emerged by the First World War. Why this choice of terminology? One might ask. A triangle of iron rather than some other material denotes an intrinsic strength, and as a concept is immediately recognizable. It springs to the lips, in a manner of speaking, and we thought it appropriate. The sides of the triangle represented diagnoses usually reserved for adult and adolescent populations, specifically catatonia, autism and schizophrenia. Catatonia surfaced as a distinctive disorder

with the work of Karl Kahlbaum in 1874 (7); schizophrenia as a well-characterized disorder (called ‘dementia praecox’) with the fourth edition of the famous textbook of Emil Kraepelin in 1893 (8); and autism with the work of Zurich psychiatry professor Eugen Bleuler in 1911 (9). Both Kraepelin and Bleuler admitted the existence of schizophrenia in early childhood, with Bleuler writing in 1911, ‘In childhood, schizophrenia seldom becomes manifest; yet there are cases that may be qualified as primary schizophrenia traceable back to the first year of life’ (9).

In adult populations there is considerable overlap among the three symptom domains. In the World Health Organization’s 1979 follow-up study of patients with psychosis in a number of international centres, among the symptoms portending a poor prognosis were stereotypes, posturing mannerisms, waxy flexibility, autism, social withdrawal, ‘unwillingness to cooperate’ (negativism) and poor rapport (10).

Yet the paediatric population is at issue here. And it is of interest that by the 1920s all three diagnoses, schizophrenia, autism and catatonia, were being routinely applied to children and adolescents. Indeed, in the paediatric population today, the three diagnoses are quite familiar, although considered to be independent disease entities.

Aims of the study

To consider the possibility that paediatric autism, catatonia and psychotic illness are different manifestations of a single underlying form of brain pathology – a kind of ‘Iron Triangle’ of symptomatology – rather than three discrete disease entities.

Material and methods

Systematic evaluation of historical case literature on autism, catatonia and psychosis to determine the concomitances of these three illnesses in children, accompanied by two challenging modern cases representative of the Iron Triangle.

Results

What is striking in the historical literature is that children who qualified for one of the three diagnoses in the Iron Triangle often qualified for the other two as well. This means either that the clinicians of these years were rather sloppy about their work, and failed to make differentiations that are clear to us; or that what we consider independent disease entities were much less mutually exclusive. Children with autism, for instance, were usually psychotic as well (‘childhood schizophrenia’) and also had evidence of catatonia, whereas children who qualified for the diagnosis of catatonia also displayed clear signs of autism and psychosis. This would suggest that all three diagnoses might stem from some underlying common font of brain pathology.

This approach contravenes conventional thinking. In 1972, Michael Rutter at the Institute of Psychiatry in London claimed that schizophrenia in childhood and autism were different diseases (11). (Catatonia was assumed to be a subtype of schizophrenia, so nobody asked about it separately.) And the whole DSM approach has been to keep schizophrenia in

childhood and autism separate. By contrast, paediatric catatonia debuted in the DSM-III in 1980 under the category 'atypical stereotyped movement disorder' yet was said to occur 'almost exclusively in children' (12). Adult catatonia was not mentioned, nor was autism or psychosis. The view that catatonia occurs mainly in children is completely erroneous.

Recently a revisionist trend has developed to integrate these conditions. International scholarship increasingly recognizes a certain confluence of schizophrenia in childhood and autism (13, 14). In 1984, Leonora Petty and colleagues at the University of Minnesota charted 'a subgroup of autistic children in whom schizophrenia develops' (15). Alexandra Sporn et al. at the Child Psychiatry Branch of NIMH wrote in 2004 of the familial co-occurrence between childhood schizophrenia (COS) and autism, 'This fact implies a familial-genetic connection between COS and autism' (16). Even though mainstream scholarship tends to see all three diagnoses as separate entities, this may make no more sense that keeping depression and anxiety separate as disorders, given that the mixed form is the most common presentation (17).

Thus, we propose suggesting a possible fusion. But is this fusion not a revival of Kraepelinian thinking itself, which said that 'dementia praecox' (schizophrenia) existed as a master diagnosis, and that it entailed autistic and catatonic symptoms? No, not really. For Kraepelin, what justified lumping autism, catatonia and psychosis together as a single disease was the uniform downhill course; yet it is clear from these early childhood cases that a number of distinctive clinical courses were possible and that the patients did not, by any means, all deteriorate. Today we are aware of many different outcomes.

The last 30 years have seen a determined effort to separate autism from Kraepelin's schizophrenia. Yet separating autism from a certain, doubtless artefactual construct called 'childhood schizophrenia', and separating it from psychosis are two different things. We find catatonia present in a wide range of childhood and adolescent disorders (18). This has been well known. It is less well known that autistic symptoms too are spread widely over other syndromes and that historically at least, autism did not seem to represent a disease of its own.

This analysis grasps merely one of the elephant's proverbial legs, looking at the diagnosis of autism in the case literature historically to see what else the patients had. One could reverse this procedure, assessing the many cases of 'childhood schizophrenia' historically to determine if autistic and catatonic symptoms accompanied the diagnosis, or examining cases in which catatonia is the chief diagnosis to see what else appears alongside. The cut attempted here is a manageable one, for the other two diagnoses are far more numerous.

On the history of autism

The accepted view is that autism as a term was originally coined by Zurich psychiatry professor Eugen Bleuler in 1911, then slumbered unused until Leo Kanner applied it to a paediatric population in 1943. This view is quite incorrect and awards to Kanner an unjustified prominence in the history of autism. In fact, in paediatric circles, autism had become a standard descriptive phrase by the 1920s for the social withdrawal that small children experienced.

Descriptions of children with a hang to solitude and loveless towards loved ones, and who simultaneously are affected with psychotic illnesses, go way back in the literature of clinical description; traditionally, the phenomenon received no name other than 'hereditary insanity'. Heinrich Schüle, chief physician at the Illenau Mental Hospital in Germany, described in the third, heavily revised, edition of his 1886 textbook children who seemed to have inherited their insanity, 'some of whom have an early tendency to solitude and to phantastical dreaming.... Of the happiness of childhood, of gaiety in the father's lap or at the mother's breast, they show little. Many express indifference, indeed coldness, to the parents and sibs.' But their intelligence is surprising, and Schüle used the phrase 'wise beyond their years.' In their studies, they tend to plod determinedly along 'and become confused and incapable if disturbed in the painfully conscientious conduct of their tasks' (19). This is close enough to later descriptions of high end autism to give pause for reflection.

Thus, the concept of developmentally delayed children with autistic and catatonic symptoms was implanted in the literature by the 1880s. In 1887, the English paediatrician J. Langdon Down, who described the variety of developmental delay named after him, associated autistic behaviour with catatonia in 'developmental idiocy', although he used neither the term autism nor catatonia: 'These children were relentlessly mute. Their language is one of gesture only; living in a world of their own they are regardless of the ordinary circumstances around them, and yield only to the counter-fascination of music'. Down described the 'automatic movements of [the child's] fingers or rhythmical movements of his body.' Such children also loathed to reciprocate affection: 'He runs to you when called, but makes no response in words. He returns your kiss by a bite....' (20). Mutism is a symptom shared in catatonia and autism.

The third arm of the triangle, psychosis, was also added in these years; descriptions of children and adolescents who are simultaneously psychotic, catatonic and show signs of autism are replete in the literature. After Emil Kraepelin popularized the term 'dementia praecox' in 1893 (which he did not coin), such cases were often given this diagnosis, which would later be called 'schizophrenia'. A case circa 1900 from a children's 'colony' in the Vaucluse asylum in a Paris suburb is demonstrative. Jean-Baptiste L. had spent a normal early childhood except that he played alone; at age 6 years he became 'bizarre' and 'changed character'. After receiving his primary-school leaving certificate at 12, he apprenticed but was said to hide away at work. His apathy increased, he refused often to get out of bed, eat or speak, or to 'accomplish the most simple actions'. At age 16 years he was admitted to the facility. Thus far there are suggestions of autism in his social isolation and of catatonia in his mutism and negativism. On the ward, he turned out to be psychotic as well, and was 'always isolated and taciturn'. In 1899, at age 17 years he was transferred to another mental hospital where he demonstrated symptoms of depression and psychosis, was virtually mute, soiled his bed and refused food. This is all catatonic behaviour. It was also possible that he had tuberculosis. Now, at 29, there is nothing much wrong with him, but he is said to be indifferent about discharge from hospital. His clinicians weighed the diagnosis 'dementia praecox' (21). Today, one might have flirted with the diagnosis schizophrenia; his catatonia was flagrant, and there are suggestions of autism. Such cases are present throughout the literature.

Autism, as stated, appears as a diagnosis in 1911. But for Bleuler, 'autism' had nothing to do with the later meaning of social isolation. He described it as a gap between the patient's fantasy world and the unlovely reality the patient inhabited. 'Thus the autistic world of thought remains inaccessible to reality, though perceived by the patient as completely real, while the patient's subjective appreciation of the real world drops to around zero.' Bleuler said that French psychopathologists had noted this gap between fantasy world and understanding of reality for years, although in his own system of schizophrenia he attached to this separation a certain importance (9). Bleuler's theoretical musings had no impact on subsequent discussions of autism, although he receives credit for introducing the term.

The history of autism took a definitive turn in 1913 when, in the eighth edition of his textbook, Emil Kraepelin described what would later be considered typically autistic behaviour as evidence of early-onset dementia praecox. 'We are dealing with children who have always shown a quiet, shy, withdrawn nature, engaged in no friendships and only lived for themselves'. Kraepelin assigned the tendency of dementia praecox patients to shutter themselves off from the outside world as 'negativism'. 'Bleuler', he said, 'has described this important diagnostic sign as autism' (22). Thereafter, autism would be considered definitively as part of dementia praecox, also called schizophrenia.

The term autism came quickly into circulation in understanding childhood schizophrenia. In 1920 F.W. Künkel, an assistant physician at the Eberswalde Asylum of Brandenburg province, referred casually to an 'autistic' group of schizophrenic children – 'quiet, closed off, loners' – as one of four psychological groups (irritable, asocial and pedantic being the other three). Most of the children in all the groups were evidently psychotic, or turned out to be later on when admitted to Eberswalde. 'The autistic children', Künkel said, 'tend more to catatonia, the asocial more to hebephrenia'. The autistic children were also said to be quite 'gifted'. (This remark would actually qualify as the introduction of the concept of high end autism, or 'Asperger's syndrome', had he provided any case histories.) (23).

Yet in an era when nosology was still determined by authoritarian pronouncement, a big professor was needed. And in the 1920s, the guiding light of autism-catatonia-psychosis research was not Kraepelin but Ernst Kretschmer, professor of psychiatry in Tübingen and originator of the hypothesis that body type and personality interacted intimately with disease presentation. In 1921, in his influential book regarding physical type and character structure, Kretschmer presented autism as a character state, not a sign of disease. He described the 'schizoid' personality: 'They live in their own little universes. One cannot know what they feel; sometimes they themselves don't know it.... What they feel, whether a banality, a cranky idea, a nastiness or some fairy tale – that is something no one will find out; they keep it for themselves'. 'Thus are schizoid individuals. Bleuler calls it autism'. Schizoids most commonly were 'unsocial, quiet, reserved, serious (humourless), and bizarre'. We are closing in here on the modern meaning of autism. Kretschmer continued, 'The autistic sealing-off of fellow persons contributes, it goes without saying, to the construction of an inner world of thoughts and favourite predilections' (24). Yet Kretschmer had little interest in paediatric patients.

It was a Russian paediatric neurologist, G. Ewa Ssucharewa, also known as Grunja Jefimowna Sucharewa or Grounia Soukhareva, who in the 1920s gave autism its modern meaning of social isolation. She has remained almost completely unknown to historians. Yet, in concentrating on a paediatric population it was she who for the first time brought autism strongly into association with catatonia. An associate professor at the Moscow University, and staffer at the Moscow Sanatorium-School of the Children's Clinic for Psychoneurology, she was keen to apply Kretschmer's ideas about autism and schizoid personalities to the children who came under her care. [The term clinic here means university hospital.] In 1926, she described six children whose symptoms had all begun before puberty. Case number one, a boy aged 13 years at the time he was admitted to the clinic, was perfectly intelligent, indeed musically gifted, but had a history of bizarre behaviour. 'From early childhood on, the boy made his parents uneasy: he is different from all the other children'. He avoided the company of other children and had peculiar thoughts: 'When he saw a coffin or the deceased were spoken of, he became very agitated and would say, "I am not going to live long"'. From six on he had a deathly fear of being left alone. He was admitted to the special school of the Clinic in 1923, his parents complaining of his obsessive-compulsive tendencies. At the time of the psychiatric assessment, he was described as 'restless'. He displayed a lot of unnecessary movements, sometimes 'tic-like jerkings of his face'. He would answer questions with a torrent of verbiage but get hung up on thoughts. He told the doctors, 'Often it seems to me that a word is turning around and around in my head so that I cannot get rid of it'. In the school he played poorly with the other children, 'chatters endlessly, rhymes words, grimaces, plays the buffoon; periods of greater agitation are also observed in which he leaps around, cuts cloth into rags'. The doctors observe perseverations. Their clinical impression? 'Tendency to automatic behaviour and obsessive-compulsive conditions.... Tendency to autistic reactions'. Diagnosis: 'Psychopathic personality. Schizoid (eccentric)' (25).

Another of Dr. Ssucharewa's six cases was more pronouncedly autistic. Case number 4, a boy of 12 at the time of admission, 'avoided the company of children' in growing up. Off to school, 'he had no contact with classmates'. At the special school, he is filled with obsessive thoughts and compulsive actions and phobias, and 'often expresses the fear that something awful will happen'. (In Moscow in the early 1920s this might not have been entirely unrealistic.) The clinicians found him 'highly intelligent', yet with a strong strain of negativism. Motor skills much delayed and 'always the last to finish a task'. Clinical impression: 'Introverted type; autistic attitudes turned inwards'. Diagnosis: 'Psychopathic personality. Schizoid (eccentric)' (25). One notes the impact of Kretschmer on these Russian clinicians, yet they are describing a quite selected clinical population: children of average or superior intelligence who display autistic behaviour towards other children in particular, meaning social isolation, and who have evidence of catatonia. Perhaps it was merely Dr Ssucharewa's luck of the draw, but none of her boys seems to have been psychotic. The Ssucharewa paper is important in identifying a high-performance group of autistic children who later would be, completely inappropriately, characterized as 'Aspergers'. (The paper was translated into English in 1996 (26))

By the time of Ssucharewa's follow-up paper 8 years later on different forms of 'childhood schizophrenia', her impressions of childhood psychosis had sharpened. By 1932, she had

seen 107 patients ages 7–17 years, and could easily differentiate those of prepubertal-onset (ages 7–12 years) from those of adolescent onset (ages 13–17 years). The latter clearly had a better prognosis. Among the 20 early-onset patients, she deemed ‘typically schizophrenic’ the transformation of the entire personality of the young child: ‘Earliest to suffer is the normal childish activity and the affective contact with the surroundings; the freshness and immediacy of the childhood experience vanished as does the joy at creative activities. The enthusiasm for this or that task and pastime, which characterizes the healthy child, is replaced here with empty chatter, hollow logic-chopping and obsessive brooding, which represent the typical schizophrenic thought disorder of the child. The thought process becomes, empty, formal and unspontaneous, tending to automatic thinking and stereotypes’. Indeed, she was struck at the heavy dose of catatonia in these early-onset patients: ‘In most cases the symptom picture develops on the catatonic side, and such symptoms becomes steadily more evident as all kinds of stereotypes, ritual behaviour, compulsive conditions [appear]; sometimes observed as well are conditions of moderate agitation and catatonic stupors’ (27).

And what distinguished the early onset from the pubertal onset groups? Ssucharewa said the early-onset patients were heavily autistic, whereas the pubertal onset patients were catatonic, even more so than the children with early onset. ‘From early childhood such [prepubertal-onset] children demonstrate a lacking ability to accommodate to life, and a certain autism and incompatibility’. The catatonic colouration of the pubertal group, by contrast, was enormous: ‘In some cases catatonic symptoms predominate, so that one may speak of pronounced conditions of catalepsy or catatonic excitement’ (27).

Ssucharewa was not the first worker to identify childhood schizophrenia, and she cites the landmark 1926 scholarship of August Homburger (28). But she is, as far as we are aware, the first to bring autism, catatonia and psychosis (‘childhood schizophrenia’) together into the Iron Triangle, which of course is our phrase, not hers.

The Children’s Clinic for Psychoneurology in Moscow turned out to be a veritable font of scholarship, one of the few in the prewar Soviet Union. In 1934, Ewa Grebelskaja-Albatz continued this analysis, describing a group of 22 children whose ‘schizophrenia’ had begun early in childhood, at ages 3–8 years, and who were much sicker than the patients of her colleague Ssucharewa. Nelly W, for example, had become ill at two and a half when ‘she underwent a great transformation and ceased answering questions; she began a bizarre kind of cry; she stared for long periods at a certain point, and obviously suffered hallucinations... she lost her toilet training, shed her previously acquired developmental skills, avoided the company of her mother, and suffered from severe autism. Moreover, the child ceased reacting to her toys, often stood for long periods at one and the same place, or walked about the room sucking on her finger’. At seven she underwent ‘a complete disintegration of her personality’. Here we have all three components together: psychosis, autism and catatonia. Many of the 22 patients of Grebelskaja-Albatz conformed to this picture (29). It would represent the less well functioning counterpart to the high-level autism reported by Ssucharewa. Grebelskaja-Albatz distinguished between two groups of ‘childhood schizophrenia’ patients: Kretschmer’s ‘schizoid psychopaths’ or autistic children of normal intelligence vs. a developmentally more disabled and thought-disordered population that was

more numerous. Yet it was the former group that carried the poorer prognosis, and underwent the most rapid decline (30).

It is thus interesting that these Russian scholars, in the German-language medical press, had made fundamental observations about the catatonia-autism-psychosis triangle that would have come as a complete surprise to their Anglo-Saxon colleagues, whose thinking about these matters trailed quite behind.

In accordance with the Kraepelinian doctrine, the clinical course in the pre-Second World War accounts was quite unfavourable. Jakob Lutz, on staff at the Stephansburg Children's Observation Station of the Zurich Psychiatric Clinic, assembled in 1937 a portrait of children who had developed 'schizophrenia' before puberty that was quite shattering. Take for example case number two, Paul, a boy of nine, admitted in 1936: his symptoms began at age 5 years with 'conspicuous changes' occurring in the course of weeks: 'He became anxious, withdrew socially, would contemplate no further interaction, played most contentedly alone in the corner, and spoke vaguely about 'something that might happen'. Upon admission to the Stephansburg he was 'absolutely autistic. No contact with the other children.... Usually he stands at a wall, or in a corner, furrows his brow, turns a toy of some kind in his hand and speaks softly to himself. Occasionally he starts to cry quite without reason and maintains that the other boys had struck him, even though no child was nearby; he also maintains at these times that others are calling to him'. The lad had an especial preoccupation with water, and crayoned burst water pipes and the like. He often grimaced. The staff treated him and others with the insulin coma therapy that was popular in the day. In this seriously affected population, the association among autism, catatonia and psychosis emerges quite vividly. Their future was bleak: 'Often these children have to be admitted as custodial cases in the mental hospitals and live there as psychic ruins' (31). And the earlier in childhood that the symptoms erupted, the worse the prognosis (32). Lutz downplayed the element of catatonia in these patients, yet his vignettes were full of accounts of 'rhythmic movements' and similar symptoms, and it is likely that he had an overly restrictive conception of the subject of catatonia.

Were the Lutz vignettes entirely representative of childhood psychosis? Of the 19 children diagnosed as schizophrenic in Kraepelin's Munich Klinik in the years 1904–22 who could be followed up (and clinically examined later), only four retained the diagnosis of schizophrenia. Many had encephalitis lethargica, a particular menace in child psychiatry in those years, or were psychopathic or mentally retarded. Fully 50 per cent of the presumed 'schizophrenia' patients on whom some kind of follow-up information was available, were later found to have adjusted nicely to the demands of normal living (33, 34). In dealing with 'childhood schizophrenia', it is thus necessary to make a distinction between schizophrenia and psychosis.

In the USA, it is clear from the case literature of the 1930s that many of the children with the diagnosis 'schizophrenia' were also catatonic and autistic. Howard Potter at the New York State Psychiatric Institute used neither of the terms, catatonia or autism, yet the patients he described were filled with such symptoms. Here is case 4, whom Potter reported in 1933, a boy of 11 who, at the age of 3 years had 'suddenly stopped talking, held himself

rigid for periods of several minutes, made faces and showed wide spread choreiform muscular twitches together with incontinence of urine and faeces'. These symptoms dissipated, giving rise to 'lone wolf' behaviour as the lad, now school age, showed uninterest in playing with his classmates. Around the age of 10 years, he then entered the Iron Triangle of symptoms: 'He was found one night sitting up in bed, grimacing and saying that his classmates were outside talking about him. The next day he insisted that the window shades be kept closed. He was sent away to a camp where he kept absolutely to himself, showed no interest in any activities and complained that the boys hated him and talked about him'. Upon his return home, he was sent to the Psychiatric Institute: 'Most of the time he is chewing, with an abstracted air, on his tie or other parts of his clothing'. His answers to questions were 'a perseverated indifferent "I don't know" or "yea"'. He was fully psychotic and believed that his saliva smelled like urine. 'When permitted, he lies in bed with his head covered with the bed clothes, entirely unresponsive and indifferent' (35). This would be a textbook example of the unity of psychosis, catatonia and autism, although Potter, following the Kraepelinian doctrine of the day, saw it as childhood schizophrenia.

Lauretta Bender, the great American authority on childhood schizophrenia, was entirely given over to psychoanalytic formulations when she took charge of the children's ward of the Psychiatric Division of Bellevue Hospital in New York City in 1934; Bender had little interest in catatonia, but considered autism highly common (36–39). Her uninterest in catatonia does not mean, however, that her young patients were without catatonic symptoms, and Bender reports a great deal of catalepsy, grimacing, 'oral mannerisms', and 'rotating and whirling motor play' (39).

In 1938, Juliette Louise Despert, a recent medical graduate of New York University College of Medicine with a clinical appointment in the children's service of the New York State Psychiatric Institute, imported the concept of autism to USA, in the context of 'schizophrenia in children'. Describing 29 children with that diagnosis admitted to the Psychiatric Institute between 1930 and 1937, she found catatonia, autism and psychosis strewn among this clinical population. One of their patients had developed normally until about three and a half when a family trauma swept him up. One day, he had a temper tantrum at a spot in the park where he often played. 'That evening, upon returning from the park it was noted that he paid no attention to the people around him but muttered indistinctly to himself words among which could be recognized 'the little boy in the park'. During the next few days, his loss of contact with reality became more and more marked', his speech inarticulate; he was subject to unmotivated laughing spells. The family doctor made a diagnosis of 'chorea'. Two weeks after admission to the Psychiatric Institute, 'he had a typical catatonic episode; attitudinizing, rigid postures kept during periods of 15–20 min, mutism and incontinence'. Over the 3 years at the Psychiatric Institute, he developed 'rigidity, mutism, active hallucinatory reactions, catatonic excitement and destructiveness and finally complete autism'. He was said to be much 'deteriorated' at the time of the most recent report in June 1937 (40).

It is thus clear, once one escapes the influence of Kretschmer late in the 1920s that the great narrative strand of autism is provided by Kraepelin's schizophrenia: autism and catatonia were part of this master diagnosis, and had a correspondingly poor prognosis.

Kanner and Asperger

In the drama of the autism diagnosis, the work of Leo Kanner and Karl Asperger in the mid-1940s comes more as afterthought than prologue. In 1943, on the basis of eleven case histories, Leo Kanner argued that 'these characteristics form a unique "syndrome", not heretofore reported' (41). The children were filled with the kind of relational difficulties and social isolation now thoroughly familiar in the literature. Kanner argued convincingly that the children did not have 'schizophrenia', yet he did not consider the possibility that these patients displayed large amounts of catatonia.

Kanner's young patients were, however, drenched in catatonic symptoms:

- Case #3: A 3-year-old comes into the hospital with a history of mutism.
- Case #4: A 5-year-old, in Kanner's office, 'got hold of a pair of scissors and patiently and skil-fully cut a sheet of paper into small bits, singing the phrase, "cutting paper", many times'. Kanner notes, 'not only repetitions but [the pattern] recurred day after day with almost photographic sameness'. These are stereotypes (which Kanner acknowledges in general at the end of the article).
- Case #5: 'Repeats phrases, gets stuck on ideas...'; scribbling verbigeration.
- Case #7: 'He sometimes uttered inarticulate sounds in a monotonous singsong manner'.
- Case #8: 'When looking at a picture, he said many times, "Some cows standing in the water." We counted fifty repetitions, then he stopped after several more and then began over and over'.
- Case #10: 'There was endless repetition of sentences'.
- Case 11: 'Her language always has the same quality. Her speech is never accompanied by facial expression or gestures'. This rigidity of the facial muscles is characteristic of catatonia. The case continues, 'She is able to spend hours in some form of daydreaming.... She is inclined to rhythmical movements which always are masturbatory' (37). Thus, a suggestion of stupor and stereotypes. (As for the masturbatory gestures in a girl of seven, it was not for nothing that both Kanner and Bruch had strong backgrounds in psychoanalysis.)

Kanner cites no previous historical literature, either because he wished to stand out the more as a pioneer, or because he was unaware of it. Yet Kanner, of Austrian origin, could not have been entirely ignorant of the substantial German-language literature on this subject reviewed here.

To construct a convincing syndrome, Kanner and his co-worker psychiatrist Hilde Bruch (of anorexia nervosa fame) evidently removed from the mix those children who were psychotic. Otherwise their 'autism' construct would have been criticized as mere schizophrenia. Yet the distinguishing characteristic of schizophrenia was a clinical course that went relentlessly downhill. The distinguishing characteristic of children in the Iron Triangle is that they do not necessarily deteriorate. Kanner's 1943 paper launched autism onto its course as an independent diagnosis. Yet his patients were not so different from those of Ssucharewa in

1926: both had been creamed off the high end of the patients in the catatonia-autism-psychosis complex.

A year later, in 1944, Hans Asperger in Vienna, at the special-education (Heilpädagogisch) division of the Vienna University Children's Hospital, apparently unaware of Kanner's effort, had another cut at the same patient population in a paper entitled 'Autistic Psychopaths in Childhood' (42). Here he presented a clinical population similar to Kanner's. Fritz V, admitted to the unit at age 6 years, had the customary blockages of social relations. Moreover, when asked a question, 'either he doesn't answer at all, or with rejection, rhythmic blows and other stereotypies'. Or he might sing, 'I don't wanna say, I don't wanna say'. Once on the ward, 'Most conspicuous were his motor stereotypies: suddenly he began to strike his thighs rhythmically, or to clap loudly against the table, against the wall, or to strike away at another person, or to hop around the ward'. The catalogue of Fritz V's catatonia continued: verbigeration, echolalia, negativism and automatic behaviour. Did Fritz V have schizophrenia? Asperger did not think so. There was no psychosis or deterioration of the personality.

Asperger's patient Harro L, age 8 years, demonstrated less catatonia, seemed highly intelligent, and spent much of the time on the unit his nose buried in a book (which doubtless gave rise to the later hopeful designation 'Asperger's Syndrome' for high end children). Ernst K, by contrast, seemed slow to the point of being developmentally handicapped, and Asperger ventured a remark about 'autistic intelligence': 'Their achievements are best when the child may spontaneously perform; worst when the child must follow a prescribed path and be tested with learned material'. Asperger described the verbigeration of his autistic students: 'The [educational] deficits appear most crassly in the act of writing: as with almost all autistic children, Ernst has a horrifying scrawl: the pen will not obey, it refuses to move forward and leaks; undisturbed he "improves the text", by writing new letters over the old ones, crossing out, sometimes writing big, sometimes small'. He displayed as well the whole panoply of other catatonic symptoms: the stereotypes, the rhythmic movements of his body, the hopping and jumping about.

Asperger's final case, Hellmuth L, was described as 'an autistic automaton', driven by rituals and other forms of automatic behaviour. It is difficult to see in this clinical population, with one or two exceptions, a pattern of autism that might have given hope to later generations of parents that their child was an 'Aspie', a library rat, rather than deeply disabled with social isolation and catatonic behaviour (42).

In these years, psychosis had drained out of the Iron Triangle because Kraepelin had decreed that 'childhood schizophrenia' was a distinctive illness, and psychotic children were considered to be in a different patient pool than the autistic and catatonic. This hiving off of childhood schizophrenia was harmful because it denied the existence of autism and catatonia in children who were not psychotic (not 'schizophrenic'). It also suggested a horrifying prognosis for psychotic children: relentless progressive deterioration to vegetable status on the backwards of the asylums and institutes for mental retardation. No wonder the parents strenuously resisted the notion that their children with autism might also be psychotic.

In the years after the Second World War autism came increasingly into discussion in child psychiatry, yet usually in the context of schizophrenia. In 1955, for example, Bender described autism in schizophrenic children as secondary symptom formation resulting from primary anxiety (43). Yet in these years the unity of the three sides of the triangle – autism, catatonia and psychosis – began haltingly to re-establish itself independently of schizophrenia. In 1965, GN Pivovarova at the Clinic for Childhood Psychoses of the Academy of Sciences in Moscow reported a group of seven children from the same extended family, five of whom fell ill, either just before puberty or just after. The children had remarkably parallel illness trajectories, beginning with autistic symptoms in which the children became ‘closed off, chose the sidelines, avoided their classmates, became sofa slugs, and only with reluctance left the house’. Then the acute illnesses began that warranted institutionalization: ‘Gradually dominated the hebephrenic-catatonic symptoms.... Impulsivity grew, often in connection with aggressivity. The patients took on eccentric poses, held themselves rigidly for a while in a fixed position, refused food. They became ever less accessible and negativistic.... Typical for many of them was their tendency to self-injury: they beat their heads against the wall and the bedsteads, lay down in bed so that their necks rested on the bed-rail [psychological pillow]; they pressed on their eyeballs [in a suggestion of avulsing them], throttled themselves with their hands or scarves, jammed their fingers in the doors, and so forth’ (44). This is quite an explicit account of self-injurious behaviour (SIB), a form of catatonia, in the context of psychosis and autism, which has recently begun again to have its own literature (45).

The French, who always had been uneasy about the schizophrenia diagnosis, were quick to identify a version of non-deteriorating psychosis in youth (‘heboïdophrenie’) that embraced autism and catatonia as well. Henri Ey and co-authors adumbrated this in 1967, ‘In fact one designates under the term “heboïdes” certain preschizophrenic patients, socially isolated [boudeurs], negativistic, impulsive, and plunged into a kind of morose autism’. The negativism would be suggestive of catatonia; the nonschizophrenic (‘preschizophrenic’) psychosis and autism are clear (46). Within psychopharmacology, the view was suggested that there were two types of autism, one that responded to the phenothiazine medications used to treat schizophrenia, the other that responded to the anticonvulsants used to treat autism in the brain-injured (47).

A combined group of researchers at Oxford and Newcastle made a promising beginning in the 1970s, distinguishing between Infantile Psychosis and Late-Onset Psychosis. In addition to the psychosis, the early-onset group demonstrated a whole range of autistic and catatonic symptoms, ranging from stereotypes and mutism (catatonic) to social isolation (autistic). The late-onset group, by contrast, showed mainly disordered thought, hallucinations, and perplexity, none of which characterize paediatric patients in the Iron Triangle. The early- vs late-onset distinction was, however, not taken up in the literature (48).

What is, in the present authors’ view, the capital contribution to the intersection of psychosis, catatonia, and autism – Karl Leonhard’s *Classification of the endogenous psychoses* (1957) – has been largely overlooked (49). The sixth edition, which appeared in 1986 (50), had a paediatric section. Although Leonhard’s distinction between unipolar and bipolar depression found numerous acolytes and was taken up by DSM-III in 1980, his other

diagnostic refinements have not found traction. But it was Leonhard who, with the authority of an enormous store of paediatric clinical experience gathered in Erfurt and Berlin after the Second World War, called attention in 1986 to catatonia as the primary symptom accompanying paediatric psychosis ('schizophrenia'), and to the presence of 'autoaggressivity' in these children. Autoaggressivity in Leonhardian lingo is SIB, and Leonhard saw it as an important form of childhood autism. (51). (True, Leonhard speaks of 'catatonic schizophrenia', but for him catatonia is the only form of schizophrenia that occurs in these psychotic children and he effectively divorces catatonia from schizophrenia.) It is one of the many unhappy chapters of the history of postwar nosology that the pioneering views of this great psychiatrist theorist have not been adopted outside of a small circle of disciples.

More recently, reports have begun to appear of catatonia superimposed on autism (52, 53). Today, clinical vignettes are accumulating that give currency to the concept of autism, catatonia and psychosis ('schizophrenia') as the sides of a pathological triangle and not necessarily independent disorders. The following contemporary case illustrates the overlap:

A 15-year-old male with past diagnoses of childhood-onset schizophrenia and autism presented for evaluation of motor skill deterioration, including leaning towards one side, falling and lower extremity rigidity, as well as global skill deterioration and decline in speech.

The patient was the product of a full-term gestation requiring Caesarean delivery for failure to progress, and neonatal course was complicated by transient hypoglycaemia and hyperbilirubinaemia. Early motor and verbal milestones were within normal limits, although the patient was described as a cranky, irritable baby who was not social and did not enjoy company of other children. At age 6 years, the patient began to demonstrate hyperactivity, irritability, tearfulness, obsessive interests and ritualistic behaviours including pacing and repeatedly drawing circles. He was initially diagnosed with attention-deficit-hyperactivity disorder, yet developed marked behavioural worsening with dextroamphetamine therapy and no benefit from methylphenidate therapy. Mood swings and behavioural agitation were reported by age 3 years, followed by property destruction, 'fits of rage', fear of fire, and onset of presumed psychotic symptoms, including paranoia with statements 'strangers are behind us and they are coming' and concern for response to acute auditory, visual, olfactory and tactile hallucinations. By the age of 4.5 years, the patient had been treated unsuccessfully with risperidone, quetiapine, olanzapine, ziprasidone, lithium, carbamazepine and oxcarbazepine. Psychiatric evaluation at this age was remarkable for sudden unusual activity including walking in circles and making nonsensical statements. Early-onset psychosis was suspected. The patient had four psychiatric hospitalizations over the next 3 years for behavioural dyscontrol, including aggression, rage and self-injury. He would alternate between demonstrating withdrawn behaviour accompanied by inconsolable sobbing, followed by aggression, agitation and reported racing thoughts and insomnia. He was frequently violent towards his siblings. One hospitalization was precipitated when the patient was found under the family Christmas tree, laughing while chewing the light bulbs and actively bleeding from his mouth. He reported hearing voices emanating from other parts of the home. Another hospitalization was preceded by homicidal threats towards his

mother and self-injurious behaviour. Childhood schizophrenia was diagnosed at age 7 years, and the patient started on clozapine therapy. At age 10 years there was renewed concern for decreased verbal fluency and school performance (including yearly drops in academic achievement and IQ scores), as well as motor deterioration. Motoric concerns included ataxic gait, tendency to lean towards the left, need to touch left side of walls when walking, and sometimes falling when turning around. Dysarthria was reported as well. Medication therapy at this time included lithium, clozapine, aripiprazole, lamotrigine and benztropine. A medication free evaluation at age 10 years led to development of visual hallucinations and report of command auditory hallucinations, with the diagnosis of schizophrenia, paranoid type made at this time and clozapine therapy resumed.

Motor decline was noted first at age 6 years, with clumsy movements and poor handwriting, as well as above issues with leaning towards one side. Further motor decline ensued at age 12 years, with loss of continence, gait disturbance, frequent falling and inability to hold his body straight. Increasing rigidity in his lower limbs led to orthopaedic heel cord release surgery, yet mother reported rigidity was not relieved. Speech deteriorated as well, with word slurring and difficulty in speech comprehension. Physical examination at age 15 years was remarkable for dysmorphic facies including a Cupid's bow mouth, broad flat forehead and low hairline, as well as widely spaced teeth. Up gaze was absent and tongue fasciculations were observed.

Family history was notable. His 16-year-old sister was diagnosed with depression, while 13-year-old brother with Tourette's and 11-year-old brother with bipolar disorder and pervasive developmental delay. Mother was diagnosed with depression, a maternal aunt with bipolar disorder, alcohol abuse and history of learning disorder, and a maternal half-aunt with depression and anxiety. Maternal grandmother diagnosed with depression, anxiety and history of learning disorder. Maternal grandfather had a history of alcohol abuse and anger management issues.

The patient's father carried a diagnosis of attention deficit hyperactivity disorder, and paternal aunt and cousin with bipolar disorder. Paternal grandmother was deceased, but with a history of depression. Paternal great grandmother was diagnosed with depression and died from suicide.

Four brain MRIs at ages 5, 10 and 12 years were all within normal limits. Karyotype, Fragile X DNA testing, FISH for 22q11 deletion syndrome and Smith-Magenis syndrome were all negative.

Similar to the case presented in the introduction, this report is intriguing as another possible illustration of the Iron Triangle. Clearly, none of the three separate diagnoses – schizophrenia, catatonia and autism – alone captures the central features. And the patient was notably unresponsive to the antipsychotic remedies that are customarily prescribed for 'schizophrenia', for which we prefer the term 'chronic psychosis'. As for autism, the patient demonstrated social withdrawal, poor relatedness and unusual patterns of behaviour from an early age. Among his catatonic symptoms were the rigidity, communication loss, various ritualistic behaviours such as walking in a circle, agitation, aggressivity and self-injury. He

also had severe symptoms of chronic psychosis that prompted several hospitalizations. (Some of the symptoms, such as SIB, might be considered in common between autism and catatonia, so frequent is catatonic behaviour in autism.) Such patients not infrequently present as modern-day diagnostic conundrums, and when given single diagnoses such as 'schizophrenia', care is indicated that may be wide of the mark and confer minimal symptom relief.

Discussion

This study raises two questions

First, what is the role of clinical subjectivity in our knowledge of autism, catatonia and schizophrenia? Catatonia has objective verifications and validations; but schizophrenia and autism seem not to be well-circumscribed disease entities, but rather disease pictures that are fluid at the edges, flowing together easily with other diagnoses. The clinical features that various observers singled out in schizophrenia and autism were not the entire picture. Max Fink writes: 'The overlap of autism, schizophrenia and catatonia criteria results from the lack of defining characteristics for each 'measure,' 'symptom,' or 'sign.' How are these terms defined? Each observer had his private image. In essence, the patients were Rorschach images and the observers picked at the signs that they thought they saw as 'objective' criteria. Since the ink-blot had many curves and pieces, some in black-and-white, and some in colour, the observers' answers, as with the Rorschach test, tended to vary' (Fink M, personal communication, 18 June 2012).

Second question: Is the mixed form of catatonia, autism and psychosis the natural form of the disease? If we wish to cut nature at the joints, is the true joint at this intersection? There is in paediatric psychiatry a long tradition of seeing these as separate, discrete disorders. Yet Watkins et al. wrote in 1988, 'The symptoms of schizophrenia and autism each appear to serve as final common pathways for a variety of causal factors and pathophysiologic processes' (16). Thus, at the centre of this Iron Triangle may well lie some deeper font of neuropathology responsible for all three. Consideration and further investigation of this hybrid presentation may offer improved future treatment options for those afflicted.

References

1. Maudsley, H. The physiology and pathology of the mind. New York: Appleton; 1867.
2. Dhossche DM, Ross CA, Stoppelbein L. The role of deprivation, abuse, and trauma in pediatric catatonia without a clear medical cause. *Acta Psychiatr Scand.* 2012; 125:25–32. [PubMed: 22017207]
3. Fink M. Hidden in plain sight: catatonia in pediatrics. *Acta Psychiatr Scand.* 2012; 125:11–12. [PubMed: 22150102]
4. Ghazziuddin N, Dhossche D, Marcotte K. Retrospective chart review of catatonia in child and adolescent psychiatric patients. *Acta Psychiatr Scand.* 2012; 125:33–38. [PubMed: 22040029]
5. Shorter E. Making childhood catatonia visible, separate from competing diagnoses. *Acta Psychiatr Scand.* 2012; 125:3–10. [PubMed: 22150101]
6. Sullivan PF, Magnusson C, Reichenberg A, et al. Family history of schizophrenia and bipolar disorder as risk factors for autism. *Arch Gen Psychiatry.* 2012; 69:1099–1103. [PubMed: 22752149]
7. Fink, M., Taylor, MA. Catatonia: a clinician's guide to diagnosis and treatment. Cambridge, UK: Cambridge University Press; 2003.

8. Shorter, E. A historical dictionary of psychiatry. New York: Oxford; 2005.
9. Bleuler, E. Dementia praecox oder Gruppe der Schizophrenien. Leipzig: Deuticke; 1911.
10. World Health Organization. Schizophrenia: an international follow-up study. Chichester, UK: Wiley; 1979. Tab 10.19
11. Rutter M. Childhood schizophrenia reconsidered. *J Autism Child Schizop.* 1972; 2:315–337.
12. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 3. Washington DC: APA; 1980. DSM-III
13. Dhossche DM. Autism as an early expression of catatonia. *Med Sci Monit.* 2004; 10:RA31–RA39. [PubMed: 14976444]
14. Kakooza-Mwesige, A., Stoppelbein, L., Dhossche, DM. Psychosis in autism. Cambridge, UK: Cambridge University Press; forthcoming
15. Petty LK, Ornitz EM, Michelman JD, Zimmerman EG. Autistic children who become schizophrenic. *Arch Gen Psychiatry.* 1984; 41:129–135. [PubMed: 6696593]
16. Watkins JM, Asarnow RF, Tanguay PE. Symptom development in childhood onset schizophrenia. *J Child Psychol Psychiatry.* 1988; 29:865–878. [PubMed: 3235494]
17. Tyrer P. The case for cothymia: mixed anxiety and depression as a single diagnosis. *Br J Psychiatry.* 2001; 179:191–193. [PubMed: 11532793]
18. Neumärker, K-J. Classification matters for catatonia and autism in children. In: Dhossche, DM, Wing, L, Ohta, M., Neumärker, K-J., editors. Catatonia in autism spectrum disorders. Amsterdam: Elsevier; 2006. p. 3–19.
19. Schüle, H. Klinische Psychiatrie. 3. Leipzig: Vogel; 1886. This passage was not present in the first edition (1878)
20. Down, JL. On some of the mental affections of childhood and youth. London: Churchill; 1887.
21. Vinchon, J. Délires des enfants. Paris: Rousset; 1911.
22. Kraepelin, E. Psychiatrie: ein Lehrbuch für Studierende und Aerzte. 8. Leipzig: Barth; 1913.
23. Kunkel FW. Die Kindheitsentwicklung der Schizophrenen. *Monat Psychiatr Neurol.* 1920; 48:254–272.
24. Kretschmer, E. Körperbau und Charakter: Untersuchungen zum Konstitutionsproblem und zur Lehre von den Temperamenten 1921. 7. Berlin: Springer; 1929.
25. Ssuharewa GE. Die schizoiden Psychopathien im Kindesalter. *Monat Psychiatr Neurol.* 1926; 60:235–261.
26. Wolff S. The first account of the syndrome Asperger described? Translation of a paper entitled 'Die schizoiden Psychopathien im Kindesalter'. *Eur Child Adolesc Psychiatry.* 1994; 5:119–132.
27. Ssuharewa G. Ueber den Verlauf der Schizophrenen im Kindesalter. *Zeits ges Neurol Psychiatr.* 1932; 142:309–321.
28. Homburger, A. Vorlesungen über die Psychopathologie des Kindesalters. Berlin: Springer; 1926.
29. Grebelskaja-Albatz E. Zur Klinik der Schizophrenie des frühen Kindesalters. *Schweiz Archiv Neurol Psychiatr.* 1934; 34:244–253.
30. Grebelskaja-Albatz E. Zur Klinik der Schizophrenie des frühen Kindesalters. *Schweiz Archiv Neurol Psychiatr.* 1935; 35:30–40.
31. Lutz J. Ueber die Schizophrenie im Kindesalter. *Schweiz Archiv Neurol Psychiatr.* 1937; 39:335–372.
32. Lutz J. Ueber die Schizophrenie im Kindesalter. *Schweiz Archiv Neurol Psychiatr.* 1937; 40:141–163.
33. Seelig P. Zur Frage der Schizophrenie im Kindesalter. *Zentralbl ges Neurol Psychiatr.* 1930; 57:845–846.
34. Corberi G. Dementia praecocissima, Dementia infantilis, Phrenasthenia aparetico-aphasica tardiva und vorübergehende psychopathische Präpubertätszustände. *Zeits Kinderforsch.* 1931; 38:268–274.
35. Potter HW. Schizophrenia in children. *Am J Psychiatry.* 1933; 89:1253–1270.
36. Bender L. Schizophrenia in childhood. *Nerv Child.* 1942; 1:138–140.
37. Bender L. Childhood schizophrenia. *Psychiatric Q.* 1953; 27:663–681.

38. Bender L. Childhood schizophrenia. *Am J Orthopsychiatry*. 1954; 26:499–506.
39. Bender L. Childhood schizophrenia: clinical study of one hundred schizophrenic children. *Am J Orthopsychiatry*. 1947; 17:40–56. [PubMed: 20282040]
40. Despert JL. Schizophrenia in children. *Psychiatric Q*. 1938; 12:366–371.
41. Kanner L. Autistic disturbances of affective contact. *Nerv Child*. 1943; 2:217–250.
42. Asperger H. Die ‘Autistischen Psychopathen’ im Kindesalter. *Arch Psychiatr Nervenkr*. 1944; 117:76–136.
43. Bender, L. Twenty years of clinical research on schizophrenic children, with special reference to those under six years of age. In: Caplan, G., editor. *Emotional problems of early childhood*. New York: Basic Books; 1955. p. 503–515.
44. Pivovarova GN. Die Besonderheiten des Verlaufs der Hebephrenie im Jugendalter. *Psychiatr Neurol Mediz Psychol*. 1965; 17:185–191.
45. Wachtel LE, Kahng S, Dhossche D, et al. ECT for catatonia in an autistic girl. *Am J Psychiatry*. 2008; 165:329–333. [PubMed: 18316431]
46. Ey, H., Bernard, P., Brisset, C. *Manuel de psychiatrie*. Paris: Masson; 1967.
47. Goldman D. Discussion comment. *Ann NY Ac Sci*. 1962; 96:374.
48. Kolvin I, Ounsted C, Humphrey M, et al. The phenomenology of childhood psychoses. *Br J Psychiatry*. 1971; 118:385–395. [PubMed: 5576636]
49. Leonhard, K. *Die Aufteilung der endogenen Psychosen*. Berlin: Akademie-Verlag; 1957.
50. Leonhard, K. *Die Aufteilung der endogenen Psychosen und ihre differenzierte Ätiologie*. 6. Berlin: Akademie-Verlag; 1986.
51. Leonhard, K. *Die Aufteilung der endogenen Psychosen und ihre differenzierte Ätiologie*. 7. Stuttgart: Thieme; 1995.
52. Dhossche D. Brief report: catatonia in autistic disorders. *J Autism Dev Disord*. 1998; 28:329–331. [PubMed: 9711489]
53. Dhossche, DM. Is autism early-onset catatonia?. In: Ryaskin, OT., editor. *Trends in autism research*. Waltham, MA: Nova Biomedical; 2005. p. 11–14.

Clinical recommendation

- There is considerable evidence to suggest that autism, catatonia and childhood psychosis may represent three different manifestations of the same underlying brain disorder; indeed, the Iron Triangle may even be the natural form of the disease. Yet this mixed form has been largely overlooked because its clinical features are conventionally seen as separate disorders.

Additional comment

- Further investigation of this hybrid presentation is necessary to confirm the Iron Triangle hypothesis.