Catatonia

A Clinician's Guide to Diagnosis and Treatment

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1

Catatonia: A history

The nineteenth century concept of 'disorder of motility' is one of the most difficult to grasp from the perspective of today. This tells much about the role of ideology and metaphor in descriptive psychopathology. For what might have in common clinical states as diverse as stupor, akinesia, catalepsy, psychomotor retardation, agitation, impulsions, bradyphrenia, parkinsonism, dyskinesias, akathisia, grimacing, mannerisms, posturing, stereotypes, soft neurological signs, tremors and tics except, perhaps, the fact that they all refer in a general way to human movement? Confronted with such a list, the neurologist of today might respond as he would to a medieval bestiary, i.e. with amused disbelief.

Berrios, 1996a: 378.

While we have no doubt that catatonia has long been a feature of human behavior, the first description in our literature is that of a patient in stupor by the English physician Philip Barrough in 1583, under the title *Of Congelation or Taking.*[1]

Catoche or Catalepsis in Greeke . . . The newe wryters in phisick do call it Congelatio, in English it maie be called Congelation or taking. It is a sodaine detention & taking both of mind and body, both sense and moving being lost, the sicke remaining in the same figure of bodie wherin he was taken, whither he sit or lye, or stand, or whither his eyes be open or shut. This disease is a meane betwene the lethargic and the frenesy, for it cometh of a melancholy humour for the most parte, as shalbe declared afterward . . . This evill differeth from Carus (as Galen saith) for that in it the eye liddes are ever shut, but in this disease they sometime remaine open. (Hunter and Macalpine, 1982: 26.)

A century later, in 1663, under the caption "catalepsy," Robert Bayfield wrote:

A congelation, is a sudden surprizal of all the senses, the motion, and the minde, with the which those that are seized upon, and invaded, remain and abide stiff, in the very same state and posture in which they were taken and surprized, with their eyes open and immovable . . . Galen mentioneth a story of a school-fellow of his, who when he had

wearied himself with long studie, fell into a Catalepsis or Congelation; he lay (saith he) like a log all along, not to be bent, stiffe, and stretched out, and seemed to behold us with his eyes, but spake not a word: And he said, that he heard us what we said at the time, although not evidently and plainly, and told us some things that he remembered, and said, all that stood by him were seen of him, and could remember, and declare some of their gestures at that time, but could not then speak, or move one part of his body . . .

Another I saw like a dead man, lying along, with neither seeing, hearing, nor feeling when he was pinched; but he breathed freely, and whatsoever was put into his mouth he presently swallowed; if he was taken out of his bed, he did stand alone, but being thrust would fall down; and which way soever his arm, hand, or leg was set, there it stood fixed, and firm; you would have taken him for a Ghost, or some rare Statue. (Hunter and Macalpine, 1982: 170.)

Individuals, once "taken", remained ill for months or years, occasionally recovering spontaneously as after a febrile illness or an epileptic seizure, but more often persisting in their condition until infection or starvation ended their lives.

A patient with negativism is eloquently described in an inquiry into the state of "madhouses" for the British House of Commons in 1815 by Thomas Bakewell.

Female, single, Age, 25. Had been six months under the care of another Keeper, when brought to me. I often said, that if ever the devil was in woman, he was surely in this one. Good heavens! When I look back upon the trouble and anxiety I underwent with this creature, I wonder how ever I got through it; her filth, her fury, disgusting language, and her almost constant nakedness for nearly two months, it being totally impossible to keep any clothes upon her, and it was scarcely possible to keep her from tearing her own flesh to pieces, as well as others; these altogether left almost without the appearance of a human being; till I had her, I thought I could manage any with the straight waistcoat; but her teeth bid defiance to every attempt to keep even that upon her. But all our extraordinary trouble arose from our not making the discovery sooner, that her particular hallucination was, had only to express the opposite of our wishes, and it was immediately done; as, Miss, you must not eat that food, it is for another person; and it was immediately taken and eaten up. Miss, you must not take that medicine, it is for such a lady, this is your's; and it was gone in an instant. Miss, you must lie still today; you must not get up, and wash you, and dress you very neatly; and up she got, and did all we bid her not to do. We therefore took care to bid her to be sure to tear her clothes all to pieces, and she remained dressed. This was certainly a departure from my usual plan of treating my patients as rational beings; but it was a case of necessity. Purgatives, tonics . . . the warm bath, cold effusion, and embrocations to the head, were put in requisition: industry and perseverance may do wonders; she got quite well, and became the well-dressed, well-bred

lady . . . After this case, I shall never think any too bad for recovery; she was under my care six months. (Hunter and Macalpine, 1982: 706.)

The image of "cataleptoid insanity" by the British physician Henry Monro in 1850 captures a catatonic patient's travail.

Cataleptoid insanity. I have been induced to apply the word cataleptoid to a certain class of insane patients who have evinced symptoms bearing a striking resemblance in some points of view to catalepsy. I will now describe this class as briefly as I can. In a large collection of insane patients we cannot help marking a few who stand in apparently profound sopor; their eyes are glued down or else staring open in a fixed manner, so immovable that you do not observe the least twinkle of the eyelid; the skin is cold and clammy; you speak to them, they will not answer; you offer them food, they will not eat. They indeed are most unwilling to move from the spot which they have taken up. You would say of them at first sight that they are in a perfectly apathetic and probably unconscious state until you try to cross their will, and then you often find a most resolute resistance. The state of the intellect in these cases is often hard to arrive at; for the mind is a prisoner; all the ordinary avenues of expression by which the caged spirit may take flight are sealed up by an influence of a numbing character, which in many points of view seems to resemble simple drowsiness. Sometimes when you lay hold suddenly of such a patient, you may shake him out of the stupor, and you find that his mind is by no means lost; that he has a clear perception of all that has been going on even during the trance; and he will argue about it as about an incubus which he could fully appreciate but could not control. I have heard the term acute dementia applied to this class of cases, but I repudiate the word on many accounts. First, it is a contradiction in terms to speak of acute dementia; the word dementia should indicate a state of fatuity which is generally the result of acute disease (the second stage as it were of madness), but is always to be applied to a passive rather than an active state; and to add the word acute to it is about equivalent to speaking of vigorous imbecility. But letting this pass, the state of mind of many of these cases is anything but demented. (Hunter and Macalpine, 1982: 988–9.)

Karl Ludwig Kahlbaum (1828–1899)

The concept of catatonia was formulated and the illness named by the German clinician Karl Ludwig Kahlbaum.

Catatonia is a brain disease with a cyclic, alternating course, in which the mental symptoms are, consecutively, melancholy, mania, stupor, confusion, and eventually dementia. One or more of these symptoms may be absent from the complete series of psychic 'symptom-complexes'. In addition to the mental symptoms, locomotor neural processes with the general character of convulsions occur as typical symptoms.[2]

Kahlbaum's clustering of the signs of catatonia into a single disease entity is his major contribution to psychiatry.[3] Early in his career, Kahlbaum developed a psychiatric nosology based on the course of illness rather than the cross-sectional picture alone. When his work received little interest from the German academic establishment, he took a position in a private sanitarium at Görlitz in 1866, became its director in 1867, and remained until his death in 1899. Numerous authors have described his life, acknowledging his contributions to the role of education in child and adolescent psychiatry and his delineation of catatonia. The most detailed biography is that of Katzenstein (1963), and his childhood and adolescence is described by Steinberg (1999). Shorter biographies by Neisser (1924), Morrison (1974a), Lanczik (1992), Bräunig and Krüger (1999, 2000), and Ahuja (2000c) praise his work. Kraepelin's assessment is described in his *History of Psychiatry:*

> He [Kahlbaum] was the first to stress the necessity of juxtaposing the condition of the patient, his transitory symptoms and the basic pattern underlying his disease. The condition of one and the same patient may change often and in diverse ways, with the result that in the absence of other clues any attempt to rescue him from his plight is doomed to failure. Moreover, identical or remarkably similar symptoms can accompany wholly dissimilar diseases while their inner nature can be revealed only through their progress and termination and, in some instances, through an autopsy.
>
> On the basis of such considerations Kahlbaum sought to delineate a second pattern of illness similar to that of paralysis and, like it, embracing both mental disorders and physical concomitants: catatonia in which muscular tension provided a basis for comparison with paralysis. Although his interpretation is faulty, Kahlbaum deserves credit for having suggested the right approach: careful attention to the progress and termination of mental disorders, information gleaned in some instances from autopsies and insight into underlying causes have made possible the juxtaposition of a vast array of evidence and often diagnosis on the basis of symptom pattern. (Kraepelin, 1919: 116–17).

Another evaluation described Kahlbaum as "*the first German psychiatrist to systematically elaborate forms of mental diseases from the pure clinical viewpoint.*"[4]

Karl Jaspers (1963, quoted in Magrinat et al., 1983) praised his work:

> Kahlbaum formulated two fundamental requirements: firstly, the entire course of the mental illness must be taken as basically the most important thing for any formulation of disease-entities and, secondly, one must base oneself on the total picture of the psychosis as obtained by comprehensive clinical observation. In emphasizing the course of the illness, he added a new viewpoint.

Kahlbaum presented a systematic psychopathology in *Die Gruppirung der psychischen Krankheiten und die Einteilung der Seelenstörungen* ("The divisions and classification of mental disorders") in 1863.[5] He created the terms *vesania*, *vecordia*, *dysphrenia*, and *neophrenia*, which have since been ignored, and the terms *paraphrenia*, *cyclothymia*, *hebephrenia*, and *catatonia* that are now in our nomenclature.[6] (Credit for the description of hebephrenia, another syndrome delineated by the course of illness, went to his co-worker Ewald Hecker.[7])

In a small book of 104 pages titled *Die Katatonie oder das Spannungsirresein*, Kahlbaum (1874) described his experience with 26 patients in both the stuporous and excited forms of the illness. He defined 17 signs of the syndrome following the diagnostic rules set down by Sydenham more than a hundred years earlier.[8] Rigid posture, mutism, negativism, and catalepsy initiated an illness that was soon followed by the hyperkinetic phenomena of stereotypes, verbigeration, and excitement. The illness had a progressive course:

> The typical signs of the condition termed atonic melancholia may be described as a state in which the patient remains entirely motionless, without speaking, and with a rigid, masklike facies; the eyes focused at a distance; he seems devoid of any will to move or react to any stimuli; there may be fully developed "waxen" flexibility, as in cataleptic states, or only indications, distinct, nevertheless, of this striking phenomenon. The general impression conveyed by such patients is one of profound mental anguish, or an immobility induced by severe mental shock; it has been classified either among the states of depression (which explains the term atonic melancholia) or among the conditions of feeble-mindedness (stupor or dementia stupida); others have regarded it as a combination of the two.[9]

Catatonia was "a temporary stage or a part of a complex picture of various disease forms."[10] He compared catatonia to dementia paralytica, a disorder that dominated the psychiatric practice at the time:

> In this newly defined group of disorders, similar to general paralysis of the insane (GPI) – with or without delusions of grandeur – clinical changes in the locomotor apparatus form the main and typical features of the disease; in addition, each disease (GPI and this disease) exhibits manifold patterns of symptoms. In GPI the paralytic components are of many varying grades of severity and type; one or other may be absent in any particular case. . . . In the same way as in GPI, the spastic signs in the newly described clinical form of the disease are also manifold and varied . . . These muscular symptoms . . . display alterations in muscular tone . . . and I would like to name this disease entity the tonic-mental disorder (Spannungs-Irresein) or vesania katatonica (catatonia) . . .[11]

He criticized the philosophy that held abnormal behaviors to result from brain lesions.

> "This anatomicopathological work produced much valuable material but contributed nothing to the basic views on the origin of mental illness or on the anatomical locus of their diverse and significant manifestations; the view is now spreading that only comprehensive clinical observation of cases can bring order and clarity into the material by using the method of clinical pathology. . . . It is futile to search for an anatomy of melancholy or mania, etc. because each of these forms occurs under the most varied relationships and combinations with other states, and they are just as little the expressions of an inner pathological process as the complex of symptoms called fever . . . How wrong it inevitably was to expect pathological anatomy alone to reform the obsolete psychiatric framework."[12]

His patients meet DSM-IV criteria for bipolar disorder, depressive mood disorder, schizophrenia, and delirium. At least seven patients were systemically ill with deliria associated with peritonitis, tuberculosis, and general paralysis of the insane.[13] The range of his patients' illnesses serves well our present image of catatonia as a syndrome in diverse psychiatric and general medical conditions.

The novelty of his presentation was quickly acknowledged. Within three years, Kiernan (1877) recognized catatonia in four patients with mania and depression. A decade later Spitzka (1883), writing from New York City's Ward's Island, described two forms of catatonia and the cyclic course of illness beginning with an initial stage of melancholia. In the same year, Neisser in Germany recognized the syndrome as a feature of mania (Neisser, 1887: 84–5).

Acknowledging catatonia as a new entity, von Schüle (1898) described six subtypes and criticized Kraepelin's adoption of catatonia as a feature of dementia praecox. In the same journal, Aschaffenberg (1898) reported an experience with 227 psychiatric patients, finding different distribution ratios among men and women for those with catatonia (men to women, 2:3) from those with dementia paralytica (3:1). An active academic industry commenting on Kahlbaum's concept quickly followed among German, French and other European as well as American authors.[14] Each effort, in samples of one to twelve patients each, confirmed Kahlbaum's descriptions and discussed the "somatic" and "psychologic" explanations for the disorder.

Catatonia purloined: Emil Kraepelin[15]

The most profound commentary on Kahlbaum's work was that of Emil Kraepelin. This leader of the German academic establishment folded Kahlbaum's descriptions of catatonia and hebephrenia into his concept of dementia praecox.[16] In successive editions of his textbook, dementia praecox became a progressive disorder that began in adolescence, deteriorating in affect and thought, ending in a state of malignant dementia. Catatonia was a phase of this unfolding pattern. Kraepelin's voice has dominated psychopathologic thought to the present day and is the basis for the classification of catatonia in DSM and ICD systems.[17]

In a distinct departure for an author writing in an age that looked to brain pathology for explanations of psychopathology, Kraepelin endorsed a psychological explanation for catatonia. He interpreted catatonic signs as *mental blocking* without a structural basis. Bleuler, using the same concepts, described mutism, negativism, and rigidity as "generalised and persistent blocking – an exaggeration of the phenomenon seen in healthy individuals when they are overwhelmed by emotional disturbance."[18] He envisioned the catatonic patient as suppressing unpleasant memories by silence (mutism), tenseness and rigidity (holds back acts that are compelled by memories), refusal to obey commands, and displacing rising emotions and tension into motor acts that shut out reality (posturing, grimacing, staring, stereotypes). Lethal catatonia was an expression of the death wish.[19]

As a corollary to these interpretations, Bleuler considered sodium amobarbital to "release" blocking, an effective therapeutic use described by Bleckwenn in 1930. Such psychodynamic thoughts are no longer fashionable today, but a recent paean to Frieda Fromm-Reichmann describes her prolonged and fruitless interactions with mute patients by "waiting them out" in months of silence.[20]

Other commentators

In a monograph on catatonia, Urstein (1912) related an experience with 30 patients. He faulted Kraepelin's adoption of catatonia as a subtype of dementia praecox, finding catatonia in patients with syphilis and other infectious diseases, toxic states, depression, mania, and delirium. The prognoses varied,

being good when episodes were few, and worsening as the episodes increased in number. Thirty-eight years after Kahlbaum's book, Urstein again regretted the lack of an effective treatment.

In follow-up studies covering more than 10 years of illness, Lange (1922) reported an experience with 200 patients meeting Kraepelin's constructs for manic-depressive illness and for dementia praecox. He found catatonia to be more prominent among the manic-depressive patients than among those with dementia praecox.

In 1940, Kleist, Leonhard and Schwab re-examined a cohort of patients identified separately in two samples examined between 1921 and 1926, one initially diagnosed by Kleist and the second by Leonhard. They classified their patients into seven groups, each subdivided into typical and atypical varieties. Stupor, rigidity, akinesia, negativism, and stereotypy were identified as forms of catatonia. At the time of the follow-up, 61 patients were living and 43 had died. A progressive course was seen in 63% and a recurrent course in 37%. They claimed the diagnoses to have been stable, concluding that the course of the illness confirmed their initial diagnoses. Again, treatment was not discussed. These many forms of catatonic schizophrenia have recently been the focus of study by Beckmann and his co-workers at the University of Würzburg in their search for a genetic basis for schizophrenia.[21]

Kahlbaum described catatonia before an audience at the University of Königsberg in 1868. On the centenary of this lecture, Pauleikhoff (1969) described his 35-year-experience with 552 hospitalized psychiatric patients. He identified 353 patients as suffering from one of five forms of catatonia: Kahlbaum's catatonia (159), stupor (100), excitement (51), malignant catatonia (26), and exogenous (systemic) catatonia (17). Twelve patient histories illustrate the catatonia variants and these descriptions are consistent with the patients that we recognize today. Pauleikhoff presented his data in five-year increments, and reported a drop in the frequency of the diagnoses after 1953. He ascribed the reduced numbers of identifiable catatonic patients as the result of a change in clinic administration and in diagnostic styles, and not as a change in the incidence of the syndrome. He called attention to the deliria that were present in his patients and concluded that catatonia was a syndrome of many forms, most with favorable outcome, and not a phase of a progressive disorder with a dementia outcome.

A neurologic image of catatonia was widely discussed by French and other European authors.[22] Catatonia was one among many motor syndromes, similar to dystonia, parkinsonism, and dyskinesia. The neurologic connection was also central to the studies of epidemic encephalitis by Von Economo who described catatonia in many patients in the acute and chronic phases of the illness (Von Economo, 1931). A comment on this era stated:

> The fact that an undoubted neurological disease, encephalitis lethargica, could produce a wide spectrum of psychiatric illness challenged the central idea of the so-called functional psychoses in an era that had come to be dominated by the teachings of psychoanalysis.[23]

The lesson that catatonia is a syndrome in post-encephalitic states is still ignored. In the successful book and film *Awakenings*, the patients experienced prolonged and severe motor inhibition, posturing, rigidity, and mutism. They were diagnosed as suffering from parkinsonism and their treatment with *l*-dopa was given prominence.[24] In 1996, the author Oliver Sacks was asked whether he considered the patients to have exhibited catatonic features. He averred that they did not have catatonia and that he had not considered using the treatments for catatonia for these patients.[25]

Conflicts in interpretation of catatonia between the somatic and psychologic views also marked the American experience. George Kirby (1913) pictured catatonia as typically occurring among patients with manic-depressive illness, and argued that Kraepelin had drawn the boundaries of schizophrenia much too broadly. In a monograph titled *Benign Stupors*, August Hoch (1921) described 25 psychiatric patients in stupor. Thirteen with manic-depressive illness had a favorable prognosis and 12 with general medical illnesses or schizophrenia had a poor prognosis.[26] Kirby and Hoch wrote at the time when a psychodynamic image of schizophrenia and catatonia dominated American psychiatry. Adolf Meyer, Smith Eli Jellife, and William Alanson White, who led American psychiatry, viewed schizophrenia and especially its catatonic form as evidence of the psychological basis for the psychoses.[27] Such views became the basis for the 1952 DSM classification that described abnormal behaviors as reactions to psychological and physical stressors, and not as defined syndromes.

A periodic form of catatonia with hormonal connections was described by Gjessing.[28] In the absence of effective treatment, he observed his patients for long periods, describing their spontaneous relapses and remissions.[29] Carefully examining thyroid metabolism, he reported direct associations with shifts in behavior, and an occasional treatment success with thyroid extracts. He concluded that periodic catatonia was a metabolic disorder. Similar reports of a periodic form of catatonia with a relationship to thyroid metabolism dot the literature.[30]

Another form of catatonia, with an acute onset and a malignant outcome, was described by Stauder in 27 patients. He labeled the disorder *Die tödliche Katatonie*, a term that is best translated as *lethal catatonia* (Stauder, 1934). Young adults between 18 and 26 years of age suddenly became mute, rigid, and either stuporous or severely excited. Fever and autonomic dysfunction were severe and the outcome was quickly fatal. The syndrome has been described by many authors, and is best known today as *malignant catatonia* (MC).[31] A subtype of the syndrome, associated with the use of antipsychotic drugs is widely recognized today as the neuroleptic malignant syndrome (see Chapter 3).

Catatonia disappears

The widespread acceptance, after 1930, of sodium amobarbital to relieve catatonia was the first sign of the coming era of psychopharmacology.[32] With the introduction of modern psychoactive drugs, clinical interest shifted from the detailed descriptions of behavior that had been the staple of psychiatric practice to the simpler goal of labeling the most prominent signs in order to select a medicine for its relief. Illnesses were described as depression or psychosis, and the treatments labeled antidepressant or antipsychotic. In the absence of a newly identified treatment for catatonia among the introduced patented medicines, interest in its descriptive psychopathology waned. Failing to undertake detailed examinations that identified the motor signs of catatonia led clinicians to conclude that the syndrome had disappeared. Mahendra's question in *Psychological Medicine* in 1981 "Where have all the catatonics gone?" argued that the syndrome no longer existed, and ascribed its disappearance to the efficacy of antipsychotic drugs.[33]

The new drugs were classified as *antipsychotic*, *antidepressant*, *antimanic*, *anxiolytic*, and *anticonvulsant*. If a patient was psychotic, one pill was prescribed; if depressed, another; if anxious, a third. Polypharmacy became widespread. Mania virtually disappeared from consideration until lithium became available, assuring a reason to identify patients with the syndrome.[34] As no medicine was labeled "anti-catatonic," catatonia was ignored. To accommodate assumed and sought differences among the medicines, and in the search for uses that could be described as "specific" for any one compound, the number of classified diagnoses increased with the publication of each new DSM classification scheme.

Another factor in the poor recognition of catatonia is the troubling awareness that the new drugs were often accompanied by disabling motor effects. The motor side-effects masked endogenous catatonic features. But an appreciation of motor side-effects for the new medicines was out of line with the enthusiasm expressed by researchers and industry leaders for their benefits. Awareness and acknowledgement of adverse motor signs was repressed.[35] Clinicians observed rigidities, tremors, dystonias, akathisias, and occasionally even the fatal *syndrom malin* in their use of the antipsychotic drugs, yet these observations were pushed out of awareness and denied. It took more than two decades for these effects to be publicly acknowledged.[36] The processes that suppressed recognition of tardive dyskinesia, tardive dystonia, and drug-induced parkinsonism served also to suppress the recognition of catatonia.

The segregation of severe psychiatrically ill patients to long-stay facilities, out of the experience of academic and research psychiatrists, also contributed to the failure to recognize catatonia. Among chronically ill patients, persistent signs of catatonia are commonplace.[37]

Interest revives

Interest in catatonia was aroused in the 1970s by studies by Morrison, Abrams and Taylor, and Gelenberg. Morrison reported that 10% of the Iowa (USA) 500 patient series had met criteria for the retarded or the excited forms of catatonia. He described catatonia as occurring more often among patients with mood disorders than among those with schizophrenia.[38]

Among patients otherwise diagnosed as suffering from mania, depression, and toxic brain states, Abrams and Taylor reported a high prevalence of catatonia.[39] They surveyed hospital admissions, reporting a prevalence of catatonia as twice as likely among patients with manic-depressive disorders as in those with schizophrenia.

Gelenberg identified catatonia in patients with neurotoxic syndromes secondary to the use of antipsychotic drugs.[40] His observations anticipated descriptions of the neuroleptic malignant syndrome and drug-induced parkinsonism.

A *syndrom malin* secondary to the use of antipsychotic drugs was described by Delay and associates (1960) and individual clinical reports followed slowly.[41] Meltzer (1973) identified a severe neurotoxic reaction to depot fluphenazine, and Weinberger and Kelly (1977) reported a syndrome of malignant catatonia secondary to neuroleptic administration. Caroff's summary of published reports captured clinical interest and his name of a *neuroleptic malignant syndrome* (NMS) was accepted (Caroff, 1980). He hypothesized that NMS resulted from excessive dopamine blockade, recommending dopamine agonists for its relief. Some patients exhibited fever and muscle weakness, suggesting a similarity to malignant hyperthermia (MH), the toxic genetic response to inhalational anesthetic agents.[42] MH was treated with the muscle relaxant dantrolene, and dantrolene and dopamine agonists were discussed as treatment for NMS (see Chapter 7).

Catatonic mutism had been relieved with barbiturates since 1930. When benzodiazepines were introduced as safer and as effective alternatives, Fricchione et al. (1983) reported lorazepam to be as effective as amobarbital in relieving catatonia. Diazepam, zolpidem and the anesthetic etomidate, for example, were soon shown to be effective.[43] The similarity of the syndrome of NMS with that of malignant catatonia was quickly recognized.[44]

That the lethal form of catatonia recognized by Bell (1849) and Stauder (1934) could be effectively treated by electroconvulsive therapy (ECT) pointed to another therapeutic direction. The report by Arnold and Stepan (1952) was confirmed by Häfner and Kasper (1982), Mann et al. (1990), and Philbrick and Rummans (1994), establishing ECT as a life-saving option for this condition. As NMS was recognized with increasing frequency, comparisons were made with malignant catatonia. Most authors could not

distinguish patients with malignant catatonia from those with the neuroleptic malignant syndrome (see Chapters 3 and 7). The view of NMS as a unique dopaminergic fault on the one hand, and as a syndrome indistinguishable from malignant catatonia on the other, spurred debates as to the proper classification of NMS and malignant catatonia.[45] The reports at the end of the 20th Century find the two syndromes indistinguishable.[46]

Soon after the introduction of monamine oxidase inhibitors, a toxic serotonin syndrome (TSS) was described. The name appeared in the psychiatric literature in 1982.[47] By 1991, the syndrome was reported to have clinical hallmarks very similar to those found in NMS, except that gastrointestinal symptoms are more prominent.[48] TSS responded to the same treatments as NMS, and some investigators conclude that it is a variant of catatonia.[49]

A formal catatonia rating scale was presented by Lohr and Wisniewski in 1987, and other scales quickly followed.[50] Although none have been widely accepted or used clinically, rating scales help researchers define the incidence of the syndrome and quantify treatment progress.

The classifications of psychiatric disorders of the 20th Century took the writings of Kraepelin and Bleuler as their guides.[51] In each diagnostic system, catatonia was defined only as a subtype of schizophrenia. When DSM-IV was proposed, the writings of the German and American authors that reported catatonia in conditions other than schizophrenia and that disagreed with Kraepelin's formulation were brought to the attention of the drafters.[52] They considered the experience of limited merit, however, and continued to recognize catatonia as a subtype of schizophrenia. But they did offer a separate class for catatonia when secondary to general medical conditions, giving it a call number (293.89). They also offered catatonia as a modifier for mania and depression.[53]

An animal model for catatonia, based on the administration of bulbocapnine, was developed by de Jong and Baruk.[54] Loss of motor initiative, catalepsy, and resistance to change in position was described in mice, cats, monkeys, pigeons, and frogs. Similar effects were observed with hallucinogens. At a time when catatonia was viewed as a psychologic reaction to stress, these experiments spurred images of brain mechanisms as the basis for a common final pathway in behavior.

The neurologic aspects of catatonia were again discussed in the 1990s by Rogers in his *Motor Disorders in Psychiatry*.[55] The postures of patients suffering from encephalitis lethargica overlapped those of patients with schizophrenia. He criticized the psychodynamic paradigm and argued for a neurologic one. To identify the specific abnormalities in his populations he developed a rating scale for catatonia.

Some authors, seeing the variety of forms that catatonia took in their patients, reverted to the term *Kahlbaum syndrome* for the patients with the retarded form of catatonia.[56]

A strong interest in catatonia re-appeared among German investigators.[57] Finding Kraepelin's descriptions unhelpful in describing patients in clinical drug trials, Beckmann, Stöber, Pfuhlmann, and Franzek turned to the earlier descriptions by Wernicke, Kleist, and Leonhard for guidance. They identified family pedigrees in which catatonia was prominent, and cited an identifiable gene associated with a subtype of the syndrome.[58] They formed a society that encouraged recognition of catatonia as a syndrome among severe psychiatric illnesses.[59] Their view of catatonia as a specific form of schizophrenia sparked a debate with American authors seeing catatonia as a syndrome of diverse manifestations. Reports from the ongoing debate were recently published.[60]

Catatonia is increasingly recognized among adolescents and children, including those with mental retardation and autism. It has not always been so. Whether catatonia is to be seen in patients with mental retardation and other developmental disorders is an ongoing debate.[61] Yet, when catatonia is recognized among patients with mental retardation, it is as responsive to catatonia treatments as it is among adults.[62]

Autism, a disorder that interferes with socialization, has an onset in early childhood. Mutism, echolalia, echopraxia, odd hand postures, freezing of ongoing movements, rigidities, forced vocalizations, stereotypy, and insensitivity to pain are its hallmarks. These signs have generally been interpreted as evidence of schizophrenia, leading to the confusion between autism and schizophrenia.[63] Clinical reports now find the treatments for catatonia to be useful for the cardinal signs of autism.[64]

Adolescents exhibiting catatonia signs are described as suffering from pervasive refusal syndrome, idiopathic recurring stupor, myalgic encephalopathy, and chronic fatigue syndrome.[65] The relation of these syndromes to catatonia is a subject of recent inquiry.[66]

Present status

Kahlbaum extracted a syndrome of catatonia from his clinical experience. His syndrome was quickly incorporated into the concept of dementia praecox, a disorder formulated by Kraepelin and Bleuler. Many authors endorsed Kahlbaum's position and criticized Kraepelin's limited view of the syndrome. Despite the evidence to support the concept of catatonia as a syndrome not tied to schizophrenia, most 20th Century classifiers of psychiatric disorders follow Kraepelin and Bleuler. The recognition of catatonia among patients with diverse disorders challenges the present classifications and it is timely to develop a separate class for catatonia. Clarification is ongoing, however, in the debate on the similarities between NMS and MC, the role of catatonia in autism and other childhood disorders, and the relationship of catatonia to stupor, delirium, and diverse neurologic disorders. The search for the genetic basis for one form of catatonic schizophrenia is another example.

In the first half of the 20th Century, catatonia was interpreted in psychologic terms. Once the syndrome could be relieved by amobarbital, however, and an animal model described, a biological model took precedence and treatment algorithms were quickly developed that are remarkably effective. The efficacy of the benzodiazepines and electroconvulsive therapy (ECT) is now well defined, and this experience would be the envy of the clinicians who described the syndrome but whose knowledge provided no effective relief for their patients. The effective interventions are anti-epileptic in their action, raising seizure thresholds and stimulating the brain's GABA-ergic systems. These commonalities offer a special focus in the search for an understanding of the pathophysiology of catatonia.

But while we have made much progress in identifying the patients who have the disorder and in our ability to help them, the position of catatonic patients in our society is fraught with unnecessary peril. Despite our social commitment that the state should protect the interests of minors and the psychiatrically ill, special regulations inhibit the use of effective treatments in patients with catatonia. Patients who are mute, negativistic, in stupor, or excited are unable to give verbal or written consent for their care. In many venues the application of intravenous sedatives, and more so, the application of ECT is severely restricted, even proscribed, by demands that the patients must personally give written, signed consent to the treatments. A 22-year-old

woman, without any prior psychiatric illness, developed malignant catatonia following a Cæsarean delivery.[67] The legal hurdles in place in California severely impeded her treatment for 33 days, risking her life and causing her and society great expense. Within 36 hours of treatment she no longer required special nursing care. The legislated obstacles in place in our states for the proper treatment of patients with catatonia serve our patients poorly.

ENDNOTES

1 Diethelm 1971; Hunter and Macalpine, 1982.
2 Kahlbaum, 1874; translated edition, 1973: 85. In the original: "*Die Katatonie ist eine Gehirnkrankheit mit cyklisch wechselndem Verlaufe, bei der die psychischen Symptome der Reihe nach das Bild der Melancholie, der Manie, der Stupescenz, der Verwirrtheit und schliesslich des Blödsinns darbieten, von welchen psychischen Gesammtbildern aber eins, oder mehrere fehlen können, und bei der neben den psychischen Symptomen Vorgänge in dem motorischen Nervensystem mit dem allgemeinen Charakter des Krampfes als wesentliche Symptome erscheinen.*" Kahlbaum, 1874: 87.
3 Mora, 1973: xiii. Also, Kraepelin, 1919; 1962.
4 Katzenstein, 1963.
5 Berrios, 1996b.
6 Kahlbaum, 1874.
7 Sedler, 1985.
8 Robins and Guze, 1970.
9 Kahlbaum, 1874, translated edition 1973: 8–9.
10 Kahlbaum, 1874, translated edition 1973: 26.
11 Kahlbaum, 1874, translated edition 1973: 27.
12 Kahlbaum, 1874, translated edition 1973: 2.
13 Berrios, 1996a: 382–3.
14 German authors: Neisser, 1887; von Schüle, 1898; Aschaffenburg, 1898; Urstein, 1912; Lange, 1922; Arnold and Stepan, 1952; and Pauleikhoff, 1969; and doctoral theses by Behr, 1891; Rauch, 1906; Hansen, 1908; Siroth, 1914; Winkel, 1925 (published 1929); and Maisel, 1936.
French authors: Dide, Guiraud and LaFage, 1921 and Guiraud, 1924; Czech author: Haskovec, 1925; Danish author: Reiter, 1926; and Swiss author: Steck, 1926, 1927, 1931; are discussed by Rogers, 1990.
American authors: Spitzka, 1883, Kiernan, 1877, Kirby, 1913, and Hoch, 1921.
15 Johnson (1993) described Kraepelin as "hijacking" these concepts.
16 Kraepelin, 1896, 1903, 1913, 1919.

17 DSM: American Psychiatric Association, 1952, 1980, 1994; ICD: World Health Organization, 1992.
18 Bleuler 1924; 1950: 211.
19 Rogers, 1991; Blacker, 1966.
20 Hornstein, 2000.
21 Beckmann et al., 1996; Stöber 2001; Stöber et al. 1995, 2000a,b,c, 2001. The genetic studies are discussed in Chapter 3.
22 Dide, Guiraud and LaFage, 1921; Guiraud, 1924; Haskovec, 1925; Reiter, 1926; Steck, 1926, 1927, 1931.
23 Johnson, 1993: 737.
24 Sacks, 1974.
25 Comment to MF, May 1996.
26 *Patient 3.15* in Chapter 3 is abstracted from his report.
27 Jelliffe and White, 1917; Jelliffe, 1940.
28 Gjessing, 1932, 1936, 1938, 1939, 1976. Also Chapter 3.
29 *Patient 3.12*, Chapter 3 is one of his patients.
30 Lindsay, 1948; Minde, 1966; Komori et al., 1997; Kinrys and Logan, 2001.
31 Chapter 3. Bell, 1849; Scheidegger, 1929; Scheid, 1937; Billig and Freeman, 1943; Arnold, 1949; Arnold and Stepan, 1952; Geller and Mappes, 1952; Huber, 1954; Pauleikhoff, 1969; Gabris and Muller, 1983.
32 Bleckwenn, 1930.
33 Also Silva et al., 1989.
34 Baldessarini, 1970.
35 Repression is the active psychologic process, an ego defense mechanism, that keeps unpleasant thoughts or observations out of awareness. (Hinsie and Campbell, 1970: 660.)
36 Gelman, 1999; Healy, 2002.
37 Turner, 1989; Bush et al., 1997.
38 Morrison 1973, 1975.
39 Abrams and Taylor, 1976; Abrams et al., 1979; Taylor and Abrams, 1973, 1977; Taylor, 1992.
40 Gelenberg 1976, 1977; Gelenberg and Mandel, 1977.
41 Lazarus et al., 1989; Gelman, 1999.
42 Lazarus, et al., 1989.
43 McEvoy and Lohr, 1983; White and Robins, 1991; White, 1992; Takeuchi, 1996; Zaw and Bates, 1997; Thomas et al., 1997.
44 Fricchione, 1985; Rosebush et al., 1990; White and Robins, 1991; White, 1992.
45 Fink, 1996a,b,c; Caroff et al., 1998a,b; Fricchione et al., 2000.
46 Davis et al., 2000; Fricchione et al., 2000; Mann et al., 2001; Fink and Taylor, 2001.
47 Insel et al., 1982.

48 Sternbach, 1991.
49 Fink, 1996b; Keck and Arnold, 2000.
50 Lohr and Wisniewski, 1987; Taylor, 1990, 1999; Rosebush et al., 1990; Rogers, 1992; Bush et al., 1996a,b; Fink, 1997a; Northoff et al., 1999b; Bräunig et al., 2000. Chapter 5.
51 Freedman, 1991.
52 Fink and Taylor, 1991.
53 American Psychiatric Association, 1994.
54 de Jong and Baruk, 1930; de Jong, 1945.
55 Rogers 1991, 1992; Rogers et al., 1991; Lund et al., 1991.
At the MacKeith Conference in 2001, Rogers showed the same overlaps in videotapes of patients.
56 Pauleikhoff, 1969; Magrinat et al. 1983; Barnes et al., 1986; Goldar and Starkstein, 1995; Peralta et al., 1997.
57 Kindt, 1980; Hippius et al., 1987; Northoff et al., 1997; Bräunig et al., 1995a, 1998, 1999, 2000; Stöber and Ungvari, 2001; and Stöber, 2001.
58 Stöber et al. 1995, 2000a,b; Beckmann et al., 1996; Stöber et al., 2000c.
59 The WKL (Wernicke–Kleist–Leonhard) Society has met every other year for a decade.
60 Stöber and Ungvari, 2001; Riederer, 2001.
61 Earl, 1934; Turner, 1989.
62 Bates and Smeltzer, 1982; Cutajar and Wilson, 1999; Thuppal and Fink, 1999; Fink, 1999a; van Waarde et al., 2001; Friedlander and Solomons, 2002.
63 Kanner, 1943; Wing and Atwood, 1987; Mullen, 1986; Realmuto and August, 1991; Dhossche and Bouman, 1997a,b; Dhossche, 1998; Zaw et al., 1999; Wing and Shah, 2000a,b; Chaplin, 2000.
64 O'Gorman, 1970; Dhossche and Bouman, 1997a; Dhossche, 1998; Zaw et al., 1999.
65 Lask et al., 1991; Tinuper et al., 1992, 1994; Palmieri 1999.
66 A Mac Keith Conference on *Catatonia in Childhood* was held in London, September 26–27, 2001 organized by Drs. M. Prendergast and G. O'Brien of Northumberland, UK.
67 Bach-Y-Rita and De Ranieri, 1992.