Chapter 1

Historical Origins of Catatonia

Catatonia before Karl Kahlbaum

Well before Karl Ludwig Kahlbaum's contributions, numerous historical authors had observed and described catatonic signs and symptoms. Writers from German- and English-speaking regions documented phenomena such as melancholia attonita, catalepsy, stereotypies, mannerisms, stupidité-stupor, negativism, madness, dementia, and alternating states. However, these manifestations were not conceptualized as a distinct nosological entity at the time (for a comprehensive historical analysis of catatonia, see references [1–5]). At the turn of the nineteenth century, the majority of authors described catalepsy as a disturbance of the brain and nerves, which would correspond with a purely neurological–mechanistic theory. With an emphasis on nerve trunks, Wilhelm Andreas Haase (1784–1837) provided an excellent description of catalepsy in 1817 [6]. He stated:

> Purely hypothetical are the assumptions that its [catalepsy's] proximate cause is due to a spasm of the roots of the sensory nerves, whereby the course of the vital spirits is hindered, or in an obstacle to the entrance of the fluidi nervei into the afflicted nerve trunks, or in an excess of blood in the head, in which an excessive quantity of nerve juice separates, as it were putting the nerves into a state of overfilling, and disturbing their function. [6, pp. 487–488]

Furthermore, catalepsy has been linked to structural changes in the brain in addition to nerve trunks. An English physician named George Man Burrows (1771–1846) [7] reported many brain disorders worsened by "insanity" in 1828, including vertigo, epilepsy, paralysis, convulsion, apoplexy, hysteria, and catalepsy. He emphasized structural brain changes as the main cause of catalepsy in his writings on the origins and treatments of insanity:

> The original seat is doubtless in the brain and nerves; but morbid anatomy throws a little light on the subject. The heads of those who are said to have died cataleptic have been opened; and according to Haller, Boerhaave, Lieutaud, and Tissot, there have been discovered the same morbid appearances, such as turgid blood-vessels, effusions of serum, polypi, concretions, tumours, etc., as have been seen in the crania of persons who have died of other and quite dissimilar diseases. [7, p. 181]

But as more cataleptic patients were observed, it became clear that the condition is linked to intense emotional states; as a result, symptoms like rigidity or catalepsy were frequently regarded as belonging to the concept of "hysteria," which primarily focused on the emotional experience of psychiatric patients. Particularly, it has been claimed by a number of historical documents and case studies that those who are thought to be more susceptible to

adverse stimuli are more likely to develop catalepsy and stiffness. In particular, this concept was outlined by the German physician Johann Gaspar Spurzheim (1776–1832) [8–10] who is known for the development and dissemination of phrenology, a field suggesting that the brain's faculties could be discerned by studying the skull's shape. Spurzheim did an impressive job of describing affective catalepsy causes in his work:

> The causes of catalepsy seem to be seldom local, but mostly general. There have been examples where plethora has produced this singular disorder and where it has been cured by a spontaneous hemorrhage. This may be the case in suppressed catamenia and catalepsy . . . Mostly, however, the causes are of a debilitating nature, and painful emotions of the mind, as unfortunate love, terror, grief, anger, etc. These affections certainly will produce a greater determination of blood to the head, while the bodily strength is diminished. The plan of cure must be modified accordingly. [9, pp. 24–25]

Similar to that, August Friedrich Hecker (1763–1811) [11], German physician and psychiatrist, primarily known for his classification of psychiatric disorders, emphasized the notion that the catatonic symptomatology had a significant affective component. He developed this concept in his posthumously published book, *Lexicon medicum theoretico-practicum reale*, which was released in 1818. He stated:

> Catalepsy or rigidity is the name given to a disease which, in certain attacks, suppresses voluntary movement as well as consciousness and sensations . . . The causes of this disease are the same as those of all nervous diseases, namely violent passions such as terror, anger, love, hatred, sadness, persistent and deep thought, extraordinary exertions of mind and body . . . Especially weak, sensitive, hypochondriacal and hysterical persons are predisposed to this disease, the female sex more than the male. [11, pp. 130–136]

Building on the ideas of Hecker, Gabriel Andral (1797–1876), a distinguished French pathologist, proposed that unstable mental states could serve as a potential cause of catalepsy. In his 1838 work, *Lectures on the Diseases of Nervous Centers* [12], Andral elaborated on the neuromechanistic perspectives of catalepsy introduced by earlier scholars. He posited that disruptions in brain function lead to a triad of impairments – sensory, cognitive, and motor – all of which are deeply interconnected and mutually dependent:

> The course and outcome of catalepsy are highly variable . . . In general, the number and frequency of the seizures and the duration of the whole disease are highly indeterminate. Sometimes it changes after a longer duration into hysteria, melancholy or epilepsy . . . According to theory, one must assume that the coincidences constituting catalepsy originate in the brain and consist in a triple injury of sensibility, intelligence and motility. Where the catalepsy has an intermittent character, the lesion of the brain must also disappear and return with the seizures. [12, p. 479]

In 1853, German physician Hermann Eberhard Richter (1808–1876) [13] provided early support for the psychomotor underpinnings of catalepsy by elucidating the intricate relationship between motor, affective, and cognitive processes in the condition. His observations reinforced the psychomotor foundation that would later become central to Kahlbaum's conceptualization of catatonia:

> Catalepsy as an independent form of the disease is rare; it occurs most frequently in females of nervous constitution and pale blood mixture . . . The attack itself is brought on by violent

> movements of the mind or mental exertion (as a similar torpor is known to occur with fright or deep thought), sometimes also by psychic infection, especially in hospitals; or it follows suppression of menstruation, haemorrhoids, perspiration, etc. Incidentally, the causes are those of nervous diseases in general. [13, p. 571]

The adoption of psychomotor concepts such as the idea that mental processes are closely linked to motor and cognitive processes based on disorders of brain structure and function proved difficult at this time. This difficulty stemmed from the concurrent emergence of Wilhelm Griesinger's (1817–1868) attempts to explain psychiatric disorders strictly in physiological and pathological terms. Griesinger advocated for recognizing all mental diseases as diseases of the brain, asserting that understanding each symptom necessitated this acknowledgment.

Richard von Krafft-Ebing (1840–1902)

What is interesting here is that although Kahlbaum was the first who postulated the idea of catatonia being a disorder characterized by motor, affective, and behavioral signs in his monograph *Catatonia or Tension Insanity*, an approach that we nowadays call "psychomotor," Kahlbaum himself did not use the term "psychomotor" at all. We searched his book using the PDF search engine but did not find the term psychomotor. This raises the question of where the term "psychomotor" originated and which author was the first to use it in a psychiatric context. After consulting with various experts in the field of sensori- and psychomotor neuroscience and inspired by our own research in historical literature, we came across the name Richard von Krafft-Ebing (1840–1902) [1, 14, 15].

Richard Fridolin Joseph Freiherr Krafft von Festenberg auf Frohnberg, called von Ebing, also known as Richard (Freiherr) von Krafft-Ebing for short (born August 14, 1840 in Mannheim; died December 22, 1902 in Graz) was a psychiatrist and sex researcher who is believed to have written some extraordinarily influential works on melancholy [16] and theoretical conceptualizations of sexuality and perversion [17, 18]. Krafft-Ebing studied medicine in Heidelberg from 1858 to 1863. In 1863, he received permission to practice medicine, heard the lectures of the influential psychiatric reformer Griesinger in Zurich, and received his doctorate in Heidelberg with a thesis on "Sensory Deliriums," which was published in 1864.

From a scientific point of view, Krafft-Ebing is best remembered for his research in sexual pathology. In 1877, he published his first sexual pathological essay, which contains 12 case vignettes and can be regarded as the forerunner of the famous "Psychopathia sexualis" that was published in 1886 [17, 18]. This work is one of the earliest clinical studies of human sexuality, and it played a significant role in the development of sexology as a scientific discipline. In this work, he described various sexual deviations, including homosexuality, which he classified as "functional signs of degeneration." Krafft-Ebing played a key role in shifting the discourse from moral condemnation to the medical treatment of sexual deviations. However, Krafft-Ebing's contributions to psychiatry were not limited to the study of sexuality. While he's most widely known for "Psychopathia sexualis," his interests and research spanned various areas of psychiatry and neurology. Krafft-Ebing had a particular interest in organic explanations for psychiatric disorders, which led him to investigate the relationships between brain lesions, abnormalities, or diseases and their manifestations as mental disorders or behavioral abnormalities. This approach included looking into psychomotor abnormalities and their potential neurological underpinnings. One of his key beliefs

was that many psychiatric disorders had an organic basis, meaning they were the result of some physical alteration or damage to the brain. This was a departure from some prevailing views of the time, which often attributed mental disorders solely to moral failings or other nonphysical causes.

To understand Krafft-Ebing's great relevance to the study of psychiatric disorders in general and psychomotor abnormalities in particular, it is important to look at his clinical and scientific work from the perspective of his family members, friends, and colleagues. Volkmar Sigusch (1940–2023; psychiatrist and pioneer of sexual medicine in Germany) has done an excellent job of reviewing and analyzing the papers of Krafft-Ebing's granddaughter Marion Josefine Georgine and great-grandson Rainer Franz Constantin Krafft-Ebing in Graz [19–21]. These are medical records from the last three decades of the nineteenth century, mostly written by hand in old German. According to the great-grandson's information about Krafft-Ebing, there are 1,386 case histories with a total volume of about 7,600 sheets from the years 1871 to 1902. About 10% of the case histories contain letters from the patients or third persons such as family members, friends, or doctors. Further, interesting aspects of his work can be seen in the references of Krafft-Ebing's assistant Alfred Fuchs (1870–1927). Fuchs studied at the universities of Prague and Vienna (MD in 1894), practiced in the Purkersdorf nerve sanatorium, and became assistant to Krafft-Ebing in 1900 and to Julius Wagner-Jauregg (1883–1940) in 1902. Fuchs described the following in his obituaries:

> Von Krafft-Ebing's way of working was mainly based on clinical observation and he always deduced his conclusions only from a large number of analogous cases. When he left Vienna (in 1902 for Graz), he took with him a collection of more than 20,000 clinical case histories. [22, p. 167]

And elsewhere Fuchs also writes:

> "He had the habit of sketching all the clinical histories himself. Many thousands of such individual sheets with his characteristic, not easily legible handwriting still exist in Mariagrün [the sanatorium he founded near Graz; V. S.]" [23, p. 183].

But when did Krafft-Ebing first speak of psychomotor disorders in the context of mental illness? In his monograph *Melancholia: A Clinical Study* [24], which was published in 1874, Krafft-Ebing used the term "psychomotor" a total of three times. On page 41, in Krafft-Ebing's remarks on *Melancholia passiva*, he impressively described different psychopathological phenomena that we can now call catatonic:

> In the highest degrees of this condition, the psychomotor rigidity may go so far that the patient outwardly presents the appearance of a stupid nonsense, and older observers (Georget, Esquirol, Ellis) have repeatedly lumped together this *melancholia stupida* as *stupidité* with primary states of stupor. The distinguishing features are that in melancholia cum stupore the disease is often acute and primary, especially in females or after epileptic seizures, that behind the mask of stuporous astonishment there is a painful facial expression instead of the meaningless one of stupor, sleep is absent, the resistance to passive movements is great, or the muscles are affected by that peculiar *flexibilitas cerea*, food is refused, the patient rapidly loses weight, and finally the condition remissions or changes momentarily into excitement, in which rapturous suicides are possible. Not infrequently, however, this melancholy stupor passes gradually and unnoticed into real insanity, in that the affect is extinguished and a deep disintegration of the mental life develops. [24, p. 41]

Interestingly, Krafft-Ebing also described nonmedication and medication therapies for *Melancholia passiva*:

> Until the end of the month the treatment consisted of balmy baths and subcutaneous injections of "extract. *opii aquos*." (up to 0.12 twice a day), the panphobia and agonising psychomotor inhibition were somewhat reduced, the patient became somewhat intelligible. [24, p. 48]

Krafft-Ebing went even further and described his clinical impression that the psychomotor abnormalities can alternate between *Melancholia activa* and *Melancholia passiva*:

> Thus, *melancholia activa* appears only as a certain psychomotor form of reaction to certain processes of consciousness in melancholic people, which in turn must be thought of as partly dependent on individuality, temperament, etc. In any case, *melancholia activa* is a form of reaction to certain processes of consciousness. In any case, it is not a special form of illness, otherwise it would be incomprehensible how such states can suddenly change into completely opposite clinical pictures, e.g. into passive melancholia with or without cataleptic rigidity. [24, p. 50]

Further, Krafft-Ebing also used the term "psychomotor" in his work *Textbook of Psychiatry on a Clinical Basis for General Practitioners and Students* [25], which was first published in 1879. This book was one of the prominent psychiatric textbooks of the time and went through multiple editions. It provided a comprehensive overview of psychiatric knowledge as understood in the late nineteenth century. In this textbook, Krafft-Ebing laid out his understanding of psychiatric disorders and particularly engaged with the concept of "psychomotor" symptoms. He used the term to describe observable physical manifestations that were believed to be connected to mental or psychiatric conditions. This could encompass a wide range of symptoms, from motor tics and twitches to more broad behavioral manifestations. In the 1883 version of his textbook [26], he described the psychomotor disorders as follows:

> These are acts of movement which have the character of intentional acts, are at any rate triggered in psychomotor brain centres, but come about without the influence of the will, on the basis of inner organ-related stimulus processes. [26, p. 89]

In particular, Krafft-Ebing also described stupor in rich detail and in a similar way as we do presently:

> Stupor. All psychic functions are inhibited here, but without being entirely suspended. Consciousness is clouded, in as much as the ideas do not rise to the clarity of normal life; apperception is clouded, slowed down, the course of ideas impeded, the associations sluggish. In particular, however, the inhibition manifests itself in the psychomotor sphere. The patient lacks all spontaneity, stands for hours on one spot, the expression of the face is one of indifference or dumbfounded amazement. Voluntary movements are rare, with obvious effort and great slowness. [26, p. 103]

Further, Krafft-Ebing impressively mentioned how motor functions are connected with psychic functions:

> In the first place, and following on from the disturbances of the psychomotor sphere, the fact must be remembered here that the entire voluntary muscular system is continually brought

> into co-excitation by the psychic precursors, on which excitation not only physiognomic expression, but also posture, intonation, timbre of voice, etc., depend. This psychic-motor innervation is diminished by the pathological psychic precursors and reflects these in the patient's external appearance. On the other hand, it is again perceived and utilized as altered muscle tone 1) by the diseased consciousness. It can be asserted that every psychopathic state, as is also the case with the effects of physiological life, has its own facies, a particular physiognomic expression 2) and overall mode of movement, which allows the experienced observer to make an approximate diagnosis even at a fleeting encounter. [26, p. 129]

At this point, Krafft-Ebing emphasized that the psychological functions have an influence on the motor symptoms and the external appearance of the patient. What is even more interesting is that Krafft-Ebing also mentioned catatonia-associated states of his patients. Krafft-Ebing wrote that following on from the mental states that move in a cyclical alternation between melancholy and mania, a type of insanity must be mentioned that is characterized by a typical alternation between mania-like states of excitement and stupor [26, p. 266]. Some of these cases formed the basis for Kahlbaum's description of his "catatonia." Although he cited Kahlbaum, he coined the disorder not catatonia but "cyclical insanity":

> The stuporous phase was characterized by intercurrent psychomotor agitation lasting for hours in the form of obsessive positions, obsessive movements, verbigeration, urge to speak with stilted diction and religiously pathetic content. The manic pictures appear clinically balanced in comparison to the usual raving madness by comic pathos in behaviour and diction, a tendency to verbigeration, by compulsively repeated, truly automatically impulsive acts of movement (spinning in circles, somersaults etc.), which, probably arising from degenerative masturbatory conditions, joined the manic phenomena of the genuine urge to move. In the majority of my cases, recovery occurred from a prolonged stupor with increasingly rare episodic states of excitement, which concluded the series of symptoms. [26, p. 267]

What the aforementioned historical authors have in common is that catatonia was not described as a distinct diagnostic entity; instead, only individual signs and symptoms of catatonia were mentioned (see Figure 1.1 for an overview).

Karl Kahlbaum

Upon joining the Görlitz private mental hospital Reimer in 1866, Kahlbaum found a fertile environment to refine his ideas on clinical psychiatry and its nosological foundations. His contributions to mental disorders nosology, documented in several seminal works, remain pivotal in the history of psychiatry. In his 1863 monograph *Grouping of Psychiatric Diseases and the Classification of Mental Disorders* [27], Kahlbaum advocated the clinical method over the anatomical approach, emphasizing the importance of objective behavioral observation in the study and classification of mental disorders [28]. This work can be seen as a critique of Heinrich Neumann's (1814–1884) theory of unitary psychosis. Kahlbaum regarded mental diseases as natural experimental phenomena, and his rigorous clinical methodology enabled the delineation of more precise diagnostic categories, including hebephrenia and catatonia. He sought to integrate clinical observations with neuropathological and anatomical research, aiming to illuminate the neurobiological underpinnings of mental illness. These ideas are further elaborated in the fourth chapter of his monograph, particularly in his discussions on catatonia [29]. In 1871, Ewald Hecker (1843–1909) [30],

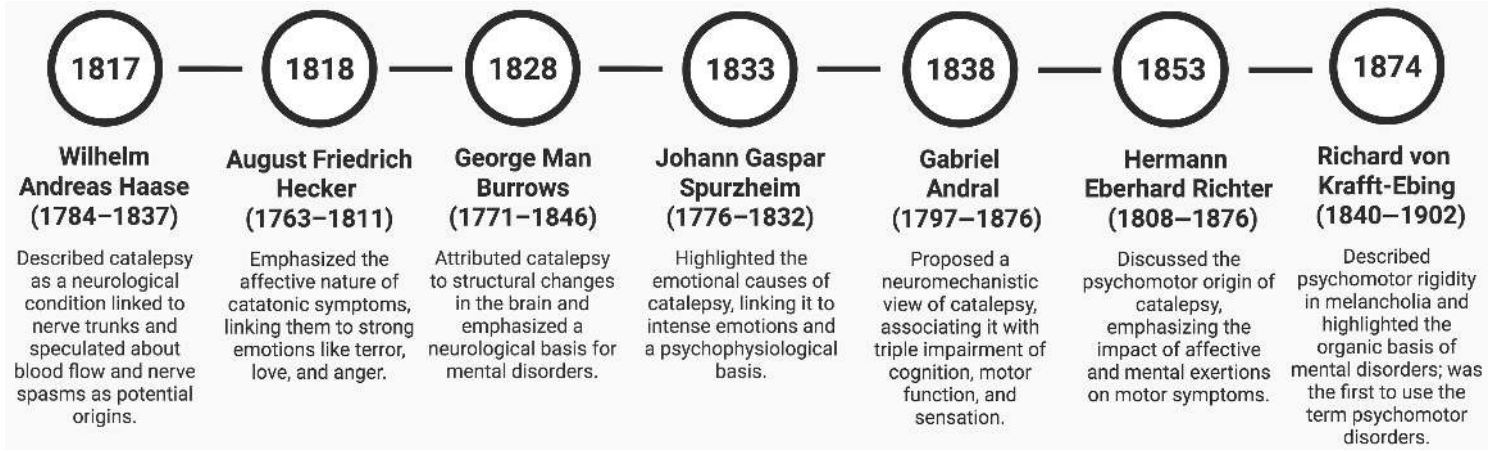

Figure 1.1 Overview of early descriptions of catatonia: Common to these authors is that catatonia was not identified as a distinct diagnostic entity. Instead, they only referenced individual signs and symptoms associated with catatonia.

a student of Kahlbaum from Görlitz, expanded upon these principles in his monograph *Hebephrenia: On the Justification of the Clinical Standpoint in Psychiatry* [31]. E. Hecker introduced a novel diagnostic framework that emphasized the importance of extended symptom monitoring, advancing Kahlbaum's clinical approach and solidifying it as a cornerstone of modern psychiatric methodology:

> This problem is particularly evident in the fact, well known to every psychiatrist that the names generally used to designate mental illnesses: Melancholia, mania, madness, confusion and dementia are quite unsuitable and inadequate, because these names do not denote real illnesses, but rather only temporary conditions. Applied to somatic medicine, they would correspond to a classification of diseases into headache, chest pain and abdominal pain, etc. [31, p. 204]

E. Hecker distinguished hebephrenia from catatonia, particularly with regard to its progression and prognosis. He observed that individuals with hebephrenia often experience a rapid decline into a state of impairment characterized by diminished emotional expression and lack of motivation, described in German as "*Verblödung*." Hecker noted the potential variability in the severity of this impairment but emphasized the persistent risk of recurring episodes of acute madness, as reflected in his statement:

> Under certain circumstances this defect may be minor, but a recurrence of intercurrent raving madness should always be feared. Perhaps this work will stimulate the publication of further cases of this kind and more precise information on the prognosis of hebephrenia can be obtained later on the basis of more comprehensive statistics. For the time being I must state that it is – if not absolute – at least very unfavorable. [30, p. 426]

However, it is important to acknowledge that the outcomes for Kahlbaum's catatonia patients were not uniformly favorable [32]. Of the 26 individuals he studied, seven fully recovered from catatonic symptoms and were discharged, seven remained in a chronic state within the hospital, and 12 died because of somatic complications. Kahlbaum recognized the progression of catatonia into a chronic or deficit condition in many cases. In the years that followed, Kahlbaum, along with E. Hecker, introduced numerous diagnostic categories that remain integral to psychiatry today, including hebephrenia, catatonia, paraphrenia, and cyclothymia. They also coined a range of terms to describe phenomena associated with mental illness – such as verbigeration, confabulation, pareidolia, reflex hallucinations, thought withdrawal, and negativism – underscoring the critical role of language in capturing the nuances of psychiatric conditions. In particular, the term catatonia, describing an independent diagnostic entity, was first introduced by Kahlbaum in 1874 in his monograph *Die Katatonie oder das Spannungsirresein, eine klinische Form psychischer Krankheit* (Figure 1.2) [33].

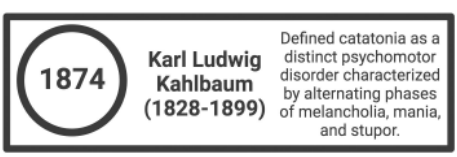

Figure 1.2 Kahlbaum considered catatonia to be a distinct diagnostic entity.

Kahlbaum referred to the observation of symptoms throughout the course of the illness in 26 patients. Based on the knowledge gained through this "clinical method," Kahlbaum defined catatonia as a separate disease entity with a specific symptom constellations, a specific course, and a specific prognosis. Catatonia was thus presented as an idiopathic, progressively advancing disease with symptoms that simultaneously affect mental and motor functions:

> Catatonia is a mental disease with a cyclically changing course, in which the mental symptoms present in sequence the picture of melancholia, mania, stuporescence, confusion and finally stupor, of which, however, one or more of the overall mental pictures may be absent and in which, in addition to mental symptoms, precursors appear in the motor nervous system with the general character of convulsions as the essential cause. [33, p. 87]

The term "catatonia" comes from the Greek and means "through-and-through tension," which was soon interpreted as "very tense" [34].

Kahlbaum spoke of the so-called neuromotor stimulation phenomena, which lead to "spasmodic states" of motor (and also psychological) functions. In the area of the speech organs, these "cramps" lead to the "verbigerations" that Kahlbaum considered pathognomic for catatonia "in which the patient pronounces meaningless or incoherent words and sentences in repetition in the apparent character of a speech ... sometimes they (the words) are completely arbitrary, but formed according to the character of a speech and even such are repeated stubbornly" [33, p. 39]. According to Kahlbaum, these verbal migrations are caused by "clonic convulsions," whereas he compared mutism, the complete "silence of the organs of speech," rather with "tonic spasms." "Convulsive states" also dominated in the area of movement and volition, leading to symptoms such as spasms, mannerisms, catalepsy, stereotypies, and negativisms. Kahlbaum thus characterized the diverse symptoms of catatonia – by the common character of "spasm" and "tension" – and contrasted them with the clinical picture of progressive paralysis, which was considered primarily a paralysis symptom.

While catatonia is characterized by an "excess" or "too much" tone, sensation, and movement, progressive paralysis is characterized by a lack or "too little." In addition to the "neuromotor stimulation symptoms," Kahlbaum also described affective and cognitive changes in connection with catatonia. After initial hypo- and hyperkinesis, melancholia with stupor can occur in a second stage of the disease, which can then develop into mania with agitation and confusion. Finally, catatonia can lead to a state of "Blödsinn." Depending on the sequential course of the various symptoms, Kahlbaum differentiated between three groups of catatonia, each with different degrees of severity: "Catatonia mitis" was characterized by "melancholia attonita" or "melancholia cum stupor," while "catatonia gravis" could be characterized by a high degree of mania following previous melancholia. Kahlbaum described the third group of catatonia as a "protracted form of catatonia," in which the neuromotor symptoms only appear during the course of the affective and cognitive symptoms, and can be accompanied by remissions and intermissions. These different forms of catatonia are thus characterized by different and, in individual cases, variable constellations of "neuromotor stimulation symptoms," affective abnormalities and cognitive defects.

Kahlbaum did not regard catatonia as a primarily hereditary disease. Instead, he emphasized predisposing trigger situations such as intellectual overexertion, sexual overexcitement, religious emotions, and the tendency to loneliness. Anatomically and pathologically, Kahlbaum

postulated a "cerebral degeneration process" with localized tribulations of the basal arachnoid membrane characteristic of catatonia (for details see Hirjak et al. [29]). In contrast to progressive paralysis, atrophy of the brain in catatonia would only occur in the late stage, the state of "obliteration."

Interestingly, Kahlbaum and E. Hecker challenged the existing symptom-based nosology in psychiatry, which categorized mental illnesses by clusters of symptoms without considering disease etiology or progression. Influenced by contemporary advancements in general medicine, particularly the understanding of bacterial diseases, they proposed that psychiatric illnesses could similarly be understood as distinct disease entities with specific courses and outcomes [35]. While Kahlbaum and E. Hecker were initially overlooked by psychiatric research, their contributions were crucial in shaping the broader field of psychiatry. Despite their skepticism toward neuropathological methods, their focus on empirical observation and clinical course significantly advanced psychiatric nosology. This paradigm shift, from symptom complexes to empirical disease forms, continues to influence contemporary psychiatric diagnosis and research [35].

Catatonia and Schizophrenia: How It All Began?

Emil Kraepelin's (1856–1926) methodology of recording mental illnesses was based on the "clinical method" introduced by Kahlbaum and pursued a synthesis of clinical description with somatic and psychological aspects. With regard to the nosological classification of catatonia, a development from Kahlbaum's concept of its own disease entity to the subordination of catatonia to schizophrenia can be observed in Kraepelin's work. In the first edition of his textbook [36], catatonia itself was not yet mentioned, while states of tension and stupor, flexibilitas cerea, and catalepsy are dealt with under the "Dämmerzustand." In the following edition, catatonia was regarded as a subtype of melancholia [37]. However, the inner states of tension occurring in the context of melancholia were distinguished from "catatonic madness" with its cognitive disturbances [37, p. 337], so that the separation between affective and cognitive symptoms of catatonia with their different categorizations (to manic-depressive insanity and dementia praecox) were already prepared here. A corresponding classification was already made in the third edition [38], where catatonia was cited as a fourth subtype of delusional disorder. In the fourth edition, catatonia was finally subordinated to dementia praecox, whereby Kraepelin postulated "numerous overlapping links" between the various forms of dementia praecox (dementia praecox, dementia paranoides, catatonia) [39, p. 435]. In the fifth edition of his textbook [40], catatonia was then regarded as a "metabolic disease with a fading process," while the affective impairments associated with catatonia are pushed into the background. In the subsequent editions of Kraepelin's textbooks, catatonia was firmly established as a subgroup of dementia praecox, where it was characterized not only by motor symptoms but above all by cognitive symptoms with blinding (=dementia) and an uncertain prognosis. Of note, Kraepelin's conceptualization of catatonia remains a subject of ongoing discussion, particularly in the context of its association with schizophrenia. Kraepelin, a pivotal figure in psychiatric nosology, recognized that catatonia signs were not exclusive to schizophrenia but could be observed in a variety of other psychiatric and medical conditions. He acknowledged that individual catatonic features, such as rigidity, mutism, or stereotypies, appeared across a broad spectrum of nosological categories, including affective disorders and neurological conditions. Despite this recognition, Kraepelin ultimately categorized patients who exhibited enduring catatonic signs accompanied by global cognitive and