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The origins of catatonia – Systematic review of historical texts between 1800 and 1900

Dusan Hirjak [a,*], Jack R. Foucher [b], Miriam Ams [a], Ludovic C. Jeanjean [b], Katharina M. Kubera [c], Robert Christian Wolf [c], Georg Northoff [d]

[a] Department of Psychiatry and Psychotherapy, Central Institute of Mental Health, Medical Faculty Mannheim, University of Heidelberg, Mannheim, Germany
[b] ICube - CNRS UMR 7357, Neurophysiology, FMTS, University of Strasbourg, CEMNIS (UF 4768) Non-invasive Neuromodulation Center, University Hospital Strasbourg, BP 426, 67 091 Strasbourg, France
[c] Center for Psychosocial Medicine, Department of General Psychiatry, University of Heidelberg, Heidelberg, Germany
[d] Mind, Brain Imaging and Neuroethics Research Unit, The Royal's Institute of Mental Health Research, University of Ottawa, Ottawa, ON, Canada

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ABSTRACT

Since January 1st 2022, catatonia is (again) recognized as an independent diagnostic entity in the 11th revision of the International Classification of Diseases (ICD-11). This is a relevant time to systematically review how the concept of catatonia has evolved within the 19th century and how this concept changed under the influence of a wide variety of events in the history of psychiatry. Here, we systematically reviewed historical and modern German and English texts focusing on catatonic phenomena, published from 1800 to 1900. We searched five different electronical databases (https://archive.org, www.hathitrust.org, www.books.google.de, https://link.springer.com and PubMed) and closely reviewed 60 historical texts on catatonic symptoms. Three main findings emerged: First, catatonic phenomena and their underlying mechanisms were studied decades before Karl Ludwig Kahlbaum's catatonia concept of 1874. Second, Kahlbaum not only introduced catatonia, but, more generally, also called for a new classification of psychiatric disorders based on a comprehensive analysis of the entire clinical picture, including the dynamic course and cross-sectional symptomatology. Third, the literature review shows that between 1800 and 1900 catatonic phenomena were viewed to be 'located' right at the interface of motor and psychological symptoms with the respective pathophysiological mechanisms being discussed. In conclusion, catatonia can truly be considered one of the most exciting and controversial entity in both past and present psychiatry and neurology, as it occupies a unique position in the border territory between organic, psychotic and psychogenic illnesses.

1. Introduction

Catatonia (from Greek *kata* = down + *tonos* = tension) is characterized by motor phenomena (e.g. stupor, posturing, catalepsy, waxy flexibility, stereotypies, akinesia), affective signs (e.g. fear, aggression, anxiety, flat affect, affect incontinence, impulsivity), and cognitive-behavioral disturbances (e.g. mutism, autism, negativism, echolalia, echopraxia, grimacing, mannerism, rituals, automatic obedience). Catatonic symptoms are clinically relevant and verifiable, neurobiologically still poorly understood, but if detected in time, then also well treatable (Fink, 2013; Fink et al., 2010; Fink and Taylor, 2003). Catatonia is a clinical feature with a prevalence of about 5 %–18 % on inpatient psychiatric units and about 3.3 % on neurology/neuropsychiatric tertiary care inpatient units (Llesuy et al., 2018; Solmi et al., 2018; Stuivenga and Morrens, 2014). A recent study by Rogers et al. utilizing electronic health records showed an increasing incidence of catatonia (Rogers et al., 2021). Despite its high prevalence, catatonia is often widely underdiagnosed among psychiatrists (Wortzel et al., 2022).

From a historical point of view, the concept of catatonia as a distinguished psychomotor entity began with Karl Ludwig Kahlbaum (1828–1899), a German psychiatrist and probably best remembered for his 1874 book on *"Catatonia or Tension Insanity"* (Kahlbaum, 1874). Although Kahlbaum was the first to describe catatonia as a distinct nosological entity, numerous catatonic symptoms have been reported previously without being brought together. A whole list of great names

* Corresponding author at: Department of Psychiatry and Psychotherapy, Central Institute of Mental Health, D-68159 Mannheim, Germany.
E-mail address: dusan.hirjak@zi-mannheim.de (D. Hirjak).

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have made contributions to better re understanding of neurobiological origin and therapeutic approaches of catatonic phenomena before and after Kahlbaum (for overview see (Shorter and Fink, 2018)). More than 220 years of scientific enthusiasm for catatonic symptoms is also reflected in the continuously increasing number of studies on phenomenology, neurobiology, and therapy of catatonia in the last three decades (for systematic reviews see (Hirjak et al., 2020; Walther et al., 2019)). More recently, catatonia as a distinct disorder has also entered our current classification systems. In particular, there is a recognition of catatonia as an independent diagnostic entity (at the same hierarchical level as mood disorders, anxiety and fear-related disorders, etc.) in the latest 11th revision of the International Classification of Diseases (ICD-11; begin on January 1st, 2022). This development was also accelerated by the DSM-5 where catatonia can be classified as *"catatonia not otherwise specified"* i.e. as residual category (Tandon et al., 2013) as well as linked to other mental disorders or specific medical conditions. Yet another example is the Research Domain classification (RDoC). Here, a sixth domain, namely *"sensorimotor domain"* devoted to sensorimotor system deficits and psychomotor abnormalities in mental disorders (Sanislow et al., 2019) has been introduced into the RDoC matrix. These milestones can be seen as a recent result of fruitful scientific discourse on sensorimotor, psychomotor and behavioral abnormalities in psychiatric disorders. These more recent developments can historically be traced to the discussion about catatonia at the era of Kahlbaum. Despite – or may be even because – of its (historical and clinical) complexity, catatonia is still not on the radar of most clinically practicing psychiatrists (Shorter and Fink, 2018). Therefore, to provide important lessons for both basic research and clinical applications, we should look into the beginnings of a more than 220-year history of catatonic phenomena and what lessons it can teach us for psychiatry in general including both diagnosis and therapy.

Several articles and books on catatonia have been published in recent years and the idea of outlining the history of catatonia is not new. In this context, the extensive studies of Max Fink (Fink, 2009, 2013; Fink and Taylor, 2009), Eduard Shorter (Fink et al., 2010; Shorter, 2006; Shorter and Wachtel, 2013) and Kenneth S. Kendler (Kendler, 2020b; Kendler and Engstrom, 2017) are particularly noteworthy when examining the history of catatonia. However, the history of catatonic phenomena is much less investigated outside Kahlbaum including the discussion prior to and during his time. Therefore, the main purpose of this paper is to provide a comprehensive and systematic review of historical texts in the period before and 25 years after Kahlbaum's first introduction of catatonia in 1874 (Kahlbaum, 2007; Kahlbaum, 1874) to flesh out the lessons this carries for catatonia in particular and Psychiatry in general. This study has three major goals: First, we will examine whether authors considered catatonic phenomena relevant in psychiatric disorders before 1874 and how catatonic phenomena were described in the time before Kahlbaum. We will focus on the initial situation which Kahlbaum found, and against this background we recall his contribution to psychiatric symptomatology and his presentation of the nosological entity, which he called catatonia. In particular, we will systematically review the key historical texts that played a role in the evolution of catatonia concept during the 19th century. In doing so, this review will contribute to the existing literature by exploring how different authors of Kahlbaum's epoch have influenced the understanding and definitional construct of catatonia. Second, this study will bring together the history and actuality of the clinical and neurobiological concepts of catatonia. We will suggest and discuss practical implications of our findings for future neurobiological and clinical studies on catatonia by combining the scientific paradigm of sensorimotor neuroscience (Hirjak et al., 2021a; Hirjak et al., 2021b) and the humanistic methodology of psychiatric historians (Heckers, 2020; Kendler, 2020b; Kendler and Engstrom, 2017). Finally, we will examine which insights can be transferred from historical concepts and current approaches into the daily clinical practice.

2. Material and methods

The search strategy and study selection followed PRISMA guidelines (Fig. 1 – PRISMA flow chart). The literature was searched using the following five electronic databases (a) https://archive.org, (b) www.hathitrust.org, (c) www.books.google.de, (d) https://link.springer.com, und (e) PubMed. The databases https://archive.org, www.hathitrust.org, www.books.google.de, and https://link.springer.com contain scanned historical journal articles and textbooks. We focused on journal articles and textbooks sections authored by physicians published from 1800 to December 31st, 1900. The main goal was to identify historical texts on catatonia in the time before and 25 years after Kahlbaum. The following English (first term) and German (second term) search terms or their combinations were used: *"catalepsy", "Katalepsie", "stereotypies", "stereotype Bewegungen", "negativism", "Negativismus", "stupor", "stupor", "catatonia", "Katatonie"*. Although there were no restrictions with respect to country of origin, we only included journal articles and textbooks sections in German or English. Articles and textbooks in French between 1800 and 1900 were not systematically reviewed. We have screened all available PDF files of the identified historical records (journal articles or textbook sections) according to the above-mentioned search terms. For further analysis and evaluation, three authors (DH, MA and GN) have selected only those reports that have dealt in greater detail with the above-mentioned search terms in patients with mental disorders and had relevance for catatonia research. DH and MA translated and GN cross-checked the selected quotations from German-language books into English. The identified historical and current sources were then critically discussed in the discussion section of this paper. Additional hand searches through reference lists identified by previous authors (Kendler, 2020a, 2020b, 2020c, 2021) and specialist psychiatric and neuroscience textbooks and journals were performed by two authors (DH and MA).

3. Results

We conducted a systematic literature search using the above-mentioned search criteria and identified a total of 3721 references (Fig. 1 – PRISMA flow chart). After reviewing titles of journal articles and textbook sections, a total of 3030 references were excluded from the analysis (e.g. no information regarding catatonia or catatonic symptoms and no human studies). 691 historical reports were assessed for eligibility. After exclusion of duplicates ($n = 217$) and not significant texts ($n = 414$), 60 documents and 72 text passages were included in the final analysis. Although we are aware of the fact that this study will turn to some more or less well-known historical texts on catatonic phenomena, the results section contains only a small selection of text passages from historical authors highlighting the evolution of catatonia concept before and 25 years after Kahlbaum (Table 1). Finally, the results are structured so that each section corresponds to a specific period, which makes it easier to follow the evolution of catatonia concept (Fig. 2).

3.1. Catatonia before Karl Ludwig Kahlbaum (1800–1874)

The history of catatonic symptoms began long before Kahlbaum. Numerous authors from German- and English-speaking countries have described typical catatonic symptoms such as melancholia attonita, catalepsy, stereotypies, mannerisms, *stupidité-stupor* or *dementia*, negativism, madness and alternation, but have not considered them as a distinct and unique nosologic entity. At the beginning of the 19th century, the majority of authors have understood catalepsy as a disturbance of brain and nerves suggesting a solely neurological-mechanistic approach. This was exemplarily described by Wilhelm Andreas Haase (1784–1837) in 1817 (Haase, 1817) who focused on nerve trunks (see quote no. 1 in Table 1). In addition to nerve trunks, structural brain alterations have been discussed as a possible cause of catalepsy. In 1828, Georg Man Burrows (Burrows, 1828) (1771–1846), an English

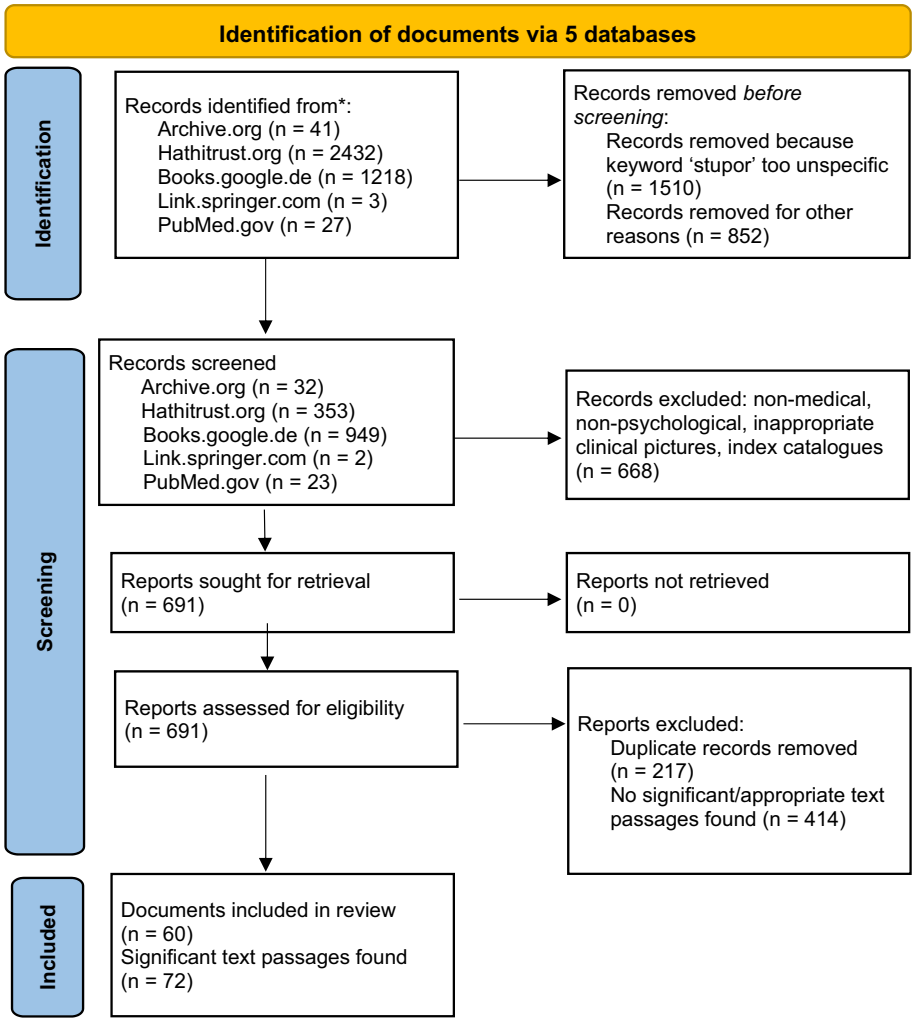

Fig. 1. PRISMA flow-chart.

physician, described different brain diseases complicated with "insanity" such as vertigo, epilepsy, paralysis, convulsion, apoplexy, hysteria and catalepsy. In his commentaries on the causes and treatment of insanity, he highlighted structural brain alterations as the primary cause of catalepsy (see quote no. 2 in Table 1). However, through further observation of cataleptic patients, it was recognized that catalepsy is associated with strong emotional states and hence, symptoms such as catalepsy or rigidity were often considered to be part of the concept of "hysteria" focusing mainly on the emotional experience of psychiatric patients. In particular, a number of historical texts and case series suggested that individuals considered to be more sensitive to negative stimuli are at higher risk of developing catalepsy and rigidity. This idea was elaborated especially by Johann Gaspar Spurzheim (1776–1832) in 1817 (Spurzheim, 1817). In his work, he impressively described affective causes of catalepsy (see quote no. 3 in Table 1). Another author that highlighted the idea of a strong affective component in the pathogenesis of catatonic symptomatology was August Friedrich Hecker (Hecker, 1818) (1763–1811). He has developed this idea in his work named *"Lexicon medicum theoretico-practicum reale"*, which was published after his death in 1818 (see quote no. 4 in Table 1). Hecker's description of the personality traits that make a person vulnerable to the development of catatonic symptomatology is in line with the concept of aberrant orbitofrontal activity, which might lead to disturbed processing of negative stimuli, higher sensitivity to everyday stress and negative emotions such as tension, anxiety, stress, and fear (Northoff et al., 2004; Northoff et al., 1999c; Taylor et al., 2019). Later on, Gabriel Andral (1797–1876), French pathologist, described unstable mental states as a possible origin of catalepsy. In his *"Lectures on the diseases of the nerve host"* (Andral, 1838) from 1838, Andral has extended the neuromechanistic approach to catalepsy suggested by earlier authors and postulated that brain changes lead to a combined, triple lesion of sensibility, cognition and motor function (see quote no. 5 in Table 1). In particular, Andral suggested that affective, cognitive and motor functions are closely dependent and thereby aligned to each other, similar to Wilhelm Griesinger's work and current studies highlighting this interrelationship in psychomotor abnormalities (Northoff et al., 2021a; Wolf et al., 2021).

In support of the above mentioned key points highlighting the psychomotor origin of catalepsy, in 1853, Hermann Eberhard Richter (Richter, 1853) (1808–1876), a German physician, described the connection between motor, affective, and cognitive processes underlying catalepsy, in line with the psychomotor origin of Kahlbaum's catatonia concept (Northoff et al., 2021a, 2021b) (see quote no. 6 in Table 1). However, it was not easy to embrace psychomotor or neuropsychiatric concepts (i.e. mental processes were brain processes) in this period, because it was also during this time that Wilhelm Griesinger (1817–1868) attempted to explain psychiatric disorders in strictly physiological and pathological terms. Griesinger suggested that it is necessary for the understanding of every disease symptom to recognize all mental diseases as diseases of the brain.

When Kahlbaum came to the Görlitz private mental hospital Reimer (named after the owner Hermann Andreas Reimer; 1825–1906) in 1866, he was given the chance to further develop his thoughts on clinical

Table 1
Selected quotes from historical authors to the development of the catatonia concept.

Quote No.	Author	Text
1	Wilhelm Andreas Haase (1784–1837)	*"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."* (pp. 487–488)
2	Georg Man Burrows (1771–1846)	*"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."* (p. 181)
3	Johann Gaspar Spurzheim (1776–1832)	*"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."* (pp. 24–25)
4	August Friedrich Hecker (1763–1811)	*"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."* (pp. 130–136)
5	Gabriel Andral (1797–1876)	*"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."*
6	Hermann Eberhard Richter (1808–1876)	*"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."* (p. 571)
7	Ewald Hecker from Görlitz (1843–1909)	*"This defect is expressed in particular in the fact that every psychiatrist is sufficiently aware of the fact that the names generally used to designate mental illnesses are: melancholy, mania, insanity, confusion and idiocy are quite 'unsuitable' and inadequate, because these names do not refer to the actual forms of disease, but only temporary forms of psychic states."*
8	Ewald Hecker from Görlitz (1843–1909)	*"In all the cases [of hebephrenia] that I have seen, feeble-mindedness usually sets in within the first three months and the latest (very rarely) a year."*
9	Karl Ludwig Kahlbaum (1874–1899)	*"In very rare cases, the entire course of the disease seems to begin immediately with the clinical picture of stupor (melancholia attonita) which has then usually been the case after very strong mental and physical insults, as after a very great fright or, as in a case taken from the literature, after an attempted hanging."* (p. 27)
10	Kahlbaum	*"More peculiar are the characters that compose the elements of the exaltation or maniacal stage. Considered as a whole, the symptoms of this stage represent either the clinical picture of agitated melancholy, or that of severe excitement, or that of more fixed madness."* (p. 30)
11	Kahlbaum	*"Mostly it is grief and worry and generally depressive moods and affections turned against oneself that give rise to catatonia. A particularly large number are love-grams and self-reproaches because of secret sexual sins, which will be given special consideration in the aetiology. Next in importance are worries about property and injured honor (shame), which form the content of the initial symptoms. Not infrequently, hypochondria and moods directed against the outside world, anger, sensitivity, irritability are also observed, and all the other melancholic symptoms, fear of poisoning, delusions of persecution, religious delusions of* sin, etc. *are often present as well."* (p. 30)
12	Kahlbaum	*"If we first look at the general mental picture which catatonia presents, we have already noticed that in its course the main types of the various mental states can all occur, namely the forms of melancholy, mania, stupor (melancholia attonita), confusion and stupidity (pseudo-dementia). The duration of the presence of the individual states can be very different, and it is not uncommon to observe multiple alternations between states of depression and exaltation."* (p. 24)
13	Kahlbaum	*"The so-called melancholia attonita (stupor), as is well known, represents that state in which the sick person sits silently, or completely mute and motionless, with a rigid expression, an immobile gaze fixed in the distance, motionless and apparently completely without will, without reaction to sensory impressions, sometimes with the fully developed symptom of flexibilitas cerea, as in catalepsy, sometimes only with a very slight, but clearly recognizable degree of this conspicuous appearance. The overall condition of such a patient gives the impression of being frozen in the deepest pain of the soul, or in the greatest fright, and as a type of disease has been placed sometimes among the states of depression (whence the name melancholia attonita), sometimes among the states of weakness (stupor or dementia stupida – cognitive deficit), sometimes also regarded as a combination of both (Baillarger's melancholie avec stupeur)."* (p. 5)
14	Kahlbaum	*"Let us now move on to the somatic symptoms. Already in the case of the last mentioned phenomena of disturbed volitional activity, it is obvious to think of a pathological innervation of the motor nerves, and this assumption becomes an extremely probable one when we see that decided convulsive states are essential symptoms of this form of disease [catatonia]. […] A part of these motor abnormalities might be regarded as something psychical, even dependent on volition, like the phenomena which were mentioned before as disturbances in volitional movement and activity. Some of them must be regarded as cerebro-spinal, like the condition of the flexibilitas cerea and the contracture-like bent postures of the limbs."* (pp. 50–51)
15	Kahlbaum	*"A further characteristic difference of catatonia is the limitation of the location or direction of the exudate deposits in the cerebral membranes, especially in the arachnoid, in that, as shown above, the base shows relatively denser and more frequent opacities, and the free leaf of the arachnoid, overlapping from the pons to the chiasm and to the frontal lobe, together with the strip of the arachnoid extending along the fossa Sylvii, is preferably the seat of this exudation. […] For this extraordinarily close relationship of tuberculosis to catatonia, it is necessary to look for an explanation in the special conditions of the catatonic disease process and it is obvious to find this in the general muscle inertia and muscle rigidity, […]."* (pp. 84–85)
16	Kahlbaum	*"Catatonia is a cerebral disease with a cyclically changing course, in which the mental symptoms present in turn the picture of melancholy, mania, stupor (melancholia attonita), confusion and finally stupidity (cognitive deficit; German: "Blödsinn"), of which overall mental pictures one or more may be absent, and in which, in addition to the mental symptoms, processes in the motor nervous system with the general character of convulsions (spasm) appear as essential symptoms."* (p. 87)
17	Kahlbaum	

(*continued on next page*)

Table 1 (*continued*)

Quote No.	Author	Text
		"Ferrum and quinine in combination with a good diet and appropriate regulation of the way of life, if necessary even against the will of the patient, seem to have contributed in no small measure to the achievement of the favorable goal in some cases of recovery." (p.102)
18	Ewald Hecker from Görlitz (1843–1909)	*"With the raving madness we see those peculiar states of tension alternating in the psychic as well as in the muscular sphere, as is uniquely characteristic of catatonia. […] The tension is noticeable in the psychic sphere through the reserved, suspiciously shy nature and culminates in the symptom of mutism, which is so extremely important for catatonia, and which differs essentially from the taciturnity that occurs, for example, in idiots, through the tightly clenched teeth and its intended partiality. At the same time, a second symptom peculiar to catatonia is expressed: the unwillingness to eat, the negativism, which is most blatantly expressed in the persistent refusal to eat. About half of all food refusers are catatonics and this symptom is therefore one of the most important."* (pp. 610–613)
19	Caspar Brosius (1825–1920)	*"Under the above designation [catatonia] Kahlbaum has described a clinical picture which, although repeatedly contested, nevertheless retains its justification, since the symptomatological whole is specific, – specific not only through the constant involvement of the motor center, which manifests itself in the various stages of the disease process partly through real cramps of various kinds, partly through striking, stereotypical forms of movement and posture, – but also specifically through the special form of the psychic symptom complexes, the states of excitement and depression, which differ in catatonia from mania and melancholia, although they appear as such and are also described in the literature. According to Kahlbaum's approach, catatonic insanity can be contrasted with paralytic insanity as a clinical group, which he has described in such detail and faithfully."* (pp. 770–786)
20	Heinrich Schüle (1840–1916)	*"In catatonic madness, psychomotor spasmodic symptoms (compulsive acts) alternate with motor spasms and contractions. […] It is this clinical connection of the two series of symptoms which anatomically-physiologically allows us to link their affinity and clinically their common origin to the deeper (*i.e. *lower structures of the brain) damaging progress of cerebral affection."* (p. 512)
21	Clemens Neisser (1861–1940)	*"But Kahlbaum rightly says that in all stages of this form of the disease (catatonia) the characteristic symptoms are so striking that confusion with other forms of the disease is not well possible once one has recognized and become acquainted with the form of the disease of catatonia as such. (…) I would now like to believe that anyone who has observed a large number of cases of catatonia with attention will agree with me when I say that not only a few prominent symptoms, but the totality of the clinical pictures presents itself as a decidedly peculiar one. With the recognition of this proposition, however, the right to exist of catatonia as an independent form of disease is pronounced anew."* (pp. 90–91)
22	Jakab Salgó (1849–1918)	*"I am more and more inclined to agree with the view of catatonia as a psychosis sui generis, even if I am by no means able to share all the details of Kahlbaum's doctrine. In any case, proper catatonia is not madness, and catatonic attonia can almost always be distinguished in itself, […], from melancholic attonia; also, indeed, I would often be at a loss to say where I would like to classify a particular disorder if I rejected catatonia as a separate species of disease. Catatonia may begin as it likes, with a hallucinatory confusion or whatever, it may present itself in its course with various forms of condition […]: one always encounters the characteristic phenomena emphasized by Kahlbaum […]."* (pp. 90–91)
23	Jules Seglas (1856–1939) and Philippe Chaslin (1857–1923)	*"We may complete this study by saying, that Kahlbaum's attempt does not seem to us so far sufficiently justified. We might repeat in substance with regard to catatonia what M. J. Falret said before concerning catalepsy, namely, that in the description of this affection, some authors have coupled together facts which, from different points of view, are dissimilar; and that they have rather recorded the history of a symptom (or better, of a "syndrome "), than of a veritable disease. If we consider further that from the physical point of view the prominent symptom is the presence of disturbance of the neuro-motor functions, whilst the principal psychical feature is a more or less acute condition of melancholia (the other symptoms, progress,* etc., *presenting nothing special), we are certainly of opinion that for the present catatonia must be classed under the general group of stupors – simple or symptomatic – of which it may only be a variety more closely connected with a degenerative and more particularly hysteric ground. We must add, that this conclusion is not an explanation, but we think it to be the only opinion which can be formulated in the present state of science. We will leave to other writers, more competent and bold, the chance of venturing upon the path, still very imperfectly known, which leads to the elucidation of the various forms assumed by the hysterical psychoses, and to define, if possible, the domain, so extensive and so vague, of mental degenerations."(pp. 230–231)*
24	Emil Kraepelin (1856–1926)	*"Under the name of catatonia, Kahlbaum has described a clinical picture which presents in turn the symptoms of melancholia, mania, stupor, and in the case of an unfavourable course also confusion and stupor, and which is also characterized by the occurrence of certain motor spasms and inhibitions, precisely the "catatonic" symptoms. (…) Even if I must consider this summary of etiologically, clinically and prognostically often quite divergent conditions to be a schematized overestimation of similar individual traits, I nevertheless see myself prompted by certain experiences to single out a certain group of cases from the field of "catatonia" as a peculiar form of disease. It is essentially a matter of the acute or subacute occurrence of peculiar states of excitement which pass into stupor and later into imbecility, with confused delusions, individual sensory illusions and the phenomena of stereotypy and suggestibility in expressive movements and actions."* (Psychiatrie: Ein kurzes Lehrbuch für Studierende und Ärzte / (Leipzig: A. Abel, 1893), 4th edition, pp. 445–446)
25	Kraepelin	*"The outcome of catatonia in the sense defined here is regularly a considerable degree of mental weakness, which not infrequently progresses to profound apathetic stupidity. (…) Kahlbaum did not regard the prognosis of catatonia as so unfavorable and reported quite a number of cures. I would like to doubt, however, that his cases were really of the same kind; apart from some, as I believe, periodic forms, he also included in catatonia observations of dementia acuta, which, in my opinion, are to be assessed essentially differently. All cases of pronounced catatonia which I have seen in the course of many years have, without exception, developed unfavorably in the manner described above. (…) Catatonia must therefore be closely related to dementia praecox, a view which is further supported by the frequency of catatonic manifestations in the latter psychosis, as well as by the occurrence of certain transitional cases between the two forms."* (Psychiatrie: Ein kurzes Lehrbuch für Studierende und Ärzte / (Leipzig: A. Abel, 1893), 4th edition, p. 454)
26	Kraepelin	*"This first stage of the disease, which in all its main features resembles that of certain hebephrenic forms, is followed in more or less marked form by those states which are peculiar to catatonia in particular, the catatonic stupor and the catatonic agitation"* (Psychiatrie: Ein kurzes Lehrbuch für Studierende und Ärzte / (Leipzig: A. Abel, 1889), 6th edition, 2nd book, p. 163)
27	Kraepelin	*"Some thirty years ago a disease was described by Kahlbaum as catatonia, or ‚insanity of rigidity', of which the most prominent symptom is a stiffness in the muscles, which would only be increased by outward interference. The disease should run through a series of different evolutions, and end at last in recovery or dementia. In the main, Kahlbaum's long-contested description has proved to be right, although I have to assume that the descriptions of disease summed up by him as catatonia are only special forms of dementia praecox. At all events, in catatonia also, disturbances of the emotional province and of action control the condition, while comprehension and memory suffer little in proportion. But then we meet with the catatonic symptoms – negativism, stereotypes, more especially the automatic obedience already described, the strange behavior, and the sudden onset of senseless impulses – in all gradations in the different forms of dementia praecox."* (Lectures on Clinical Psychiatry, 1904, p. 32)
28	Carl Wernicke (1848–1905)	*"Clinical observation confirms our view in so far as we shall find the symptom with preference in the severe states of the disease, quite apart from the uncommonly frequent occurrence of it in the agitated forms of the paralytic psychoses. Among the non-*

(*continued on next page*)

Table 1 (*continued*)

Quote No.	Author	Text
		paralytic psychoses it is especially the two clinical pictures of confused mania and hyperkinetic motility psychosis where the symptom is almost never missed and forms a very essential part of the clinical picture. Gentlemen! It should not surprise you that our AZ scheme also permits the derivation of certain borderline cases in which the principle of secondary identification disorders is violated and a transition between primary and secondary disorders of identification seems to take place." (Grundriss der Psychiatrie in klinischen Vorlesungen, p. 214)

psychiatry and its nosological entities. Kahlbaum left behind several works on psychiatric nosology, which are important for the history of psychiatry. In his monograph on the classification of psychiatric diseases (Kahlbaum, 1863) (German: *"Die Gruppierung der psychischen Krankheiten und die Einteilung der Seelenstörungen"*; English: *"Grouping of psychiatric diseases and the classification of mental disorders"*), Kahlbaum used the clinical (not the anatomical) method and suggested that the unprejudiced behavioral observation of clinical cases (patients) is of utmost relevance for research and delineation of psychiatric disorders (Brune, 2000). Kahlbaum's monograph can be considered as a reaction to Heinrich Neumann's (1814–1884) theses on unitary psychosis. Kahlbaum considered psychiatric disorders to be *"experimental states provided by nature"* and his (clinical) method of delineation has led to more independent diagnostic entities being described (e.g. hebephrenia and catatonia). Furthermore, Kahlbaum tried to link his clinical material with neuropathological/−anatomical findings to better describe the neurobiological origin of psychiatric disorders. Similarly to Kahlbaum, in 1871, Ewald Hecker from Görlitz (1843–1909; mentee of Kahlbaum) wrote a monograph on hebephrenia called *"Hebephrenia: On the justification of the clinical standpoint in Psychiatry"* (Hecker, 1871) and proposed a novel diagnostic system based on the longitudinal observation of disease symptoms as well (see quote no. 7 in Table 1). Furthermore, E. Hecker recognized that there was a difference between hebephrenia and catatonia, namely in the outcome and prognosis. Patients with hebephrenia often exhibited a rapid transition to a deficit state characterized by a blunted affect and drive deficit (German: "*Verblödung*") (see quote no. 8 in Table 1). In the following years, Kahlbaum and E. Hecker defined a number of new diagnostic categories, which we still use today: Hebephrenia, Catatonia, Paraphrenia, and Cyclothymia. They also defined new psychopathological terms, such as verbigeration, confabulation, pareidolia, reflex hallucinations, thought withdrawal, and negativism (see next epoch), which once again highlight the importance they attached to language in psychiatric disorders.

To summarize the epoch before Kahlbaum, two points are particularly noteworthy. First, catatonic symptoms were described long before Kahlbaum. However, these were sole descriptions of single signs and not of catatonia as an independent entity. Second, during this time period, E. Hecker and Kahlbaum attempted to delineate more and more independent disease entities (e.g. paraphrenia, cyclothymia, etc.) and psychopathological symptoms (e.g. verbigeration, negativism, confabulation, etc.). It is therefore no coincidence that hebephrenia (Hecker, 1871) and catatonia (Kahlbaum, 1874) were described at about the same time. The disentanglement of hebephrenia and catatonia was probably the result of a long maturation, scientific discussion, and familial connection between Hecker and Kahlbaum. It can be speculated that it was the close family connection (Hecker married Kahlbaum's daughter in 1871) that freed Kahlbaum from his hesitation (after bad experiences from 1868) to finally coin catatonia as a distinct disease in 1874.

3.2. Introduction of Karl Ludwig Kahlbaum's catatonia (1874)

Kahlbaum's famous monograph of 1874 was the first book of a planned series of clinical treatises on mental illnesses, in which the aim was to describe (and develop) novel nosological entities according to clinical methods (Berrios, 2007). Kahlbaum described a total of 26 patients presenting with flamboyant psychomotor abnormalities and coined this clinical disorder "catatonia" (Kahlbaum, 2007; Kahlbaum, 1874). He defined 17 catatonic signs including posture, mutism, negativism, and catalepsy, respectively (Kahlbaum, 2007; Kahlbaum, 1874). In Kahlbaum's description of catatonia, he differentiated between (i) psychological and (ii) physical/bodily symptoms. In line with the sensualist account of mental processes endorsed by most German psychiatrists, the psychological/mental symptoms/phenomena might be also divided into disorders of (a) feelings (pp. 30–36 of *"Die Katatonie oder das Spannungsirresein"* (Kahlbaum, 1874)), (b) willing or will and (c) thinking. In line with this, Kahlbaum defined two seemingly opposing patterns or catatonia: (a) the agitated, hyperkinetic form, and (b) the negative, hypokinetic form associated with tension or tonic spasm. Kahlbaum proceeded systematically and described the most important domains (affective, motor and behavioral) of catatonia in a very detailed manner. Additionally, Kahlbaum used emotional and affective symptoms almost synonymously. At the beginning of his monograph (p. 24), Kahlbaum suggested that the fluctuating course of strong affective symptoms such as depression, mania and exaltation might result in stupor (melancholia attonita) or raving madness (German: "*Tobsucht*") (see quotes no. 9 and 10 in Table 1). More specifically, he assumed that the onset of catatonic symptoms follows a continuum, the emotional/affective symptoms such as depressed mood, psychic impulses, anger, sensitivity, and irritability are often leading phenomena at the beginning of a catatonic state (see quote no. 11 in Table 1). Kahlbaum understood catatonia as a phenotype, which implicitly supposes that it should account for all the clinical pictures observed during the whole life of a same patient. Hence he defined catatonia in longitudinal terms and distinguished three different courses characterized by a distinct sequence of symptoms and different duration and intensity: (1) *catatonia mitis (*beginning often with *"melancholia attonita"* or *"melancholia cum stupore")*, (2) *catatonia gravis* (often accompanied by mania) and (3) *catatonia protracta* (often accompanied with chronic-progressive symptom development) (Starkstein et al., 1995). What stands out is how Kahlbaum understood *melancholia attonita* (see quotes no. 12 and 13 in Table 1) and to what extent its correlate with the concepts and findings of current clinical and neuroimaging studies on catatonia. Kahlbaum emphasized locomotor phenomena up to name his "disease candidate" by them. These were essential to him as their co-occurrence with mental symptoms were very suggestive of the organic nature of the whole phenotype (pp. 50–51 and 62–86). He suggested that like the general progressive paralysis, catatonia is also a psychic form of disease, which in itself often leads to death without competing with other diseases, and therefore it will perhaps be possible to *"find the anatomical genesis"* (p. 62). Kahlbaum's ideas regarding the neurobiology of catatonia were by no mean developed randomly, but were a result of a large number of autopsies on catatonic patients at the East Prussian Provincial Hospital in Allenberg. In the 4th chapter of his monograph devoted to pathological anatomy, Kahlbaum described macroscopic examinations of the brain during autopsy of 11 patients (cases #15-#25). He found structural alterations within pons and medulla oblongata (brainstem), thalamus, striatum and the frontal regions that could lead to catatonia (similar to recent fMRI results (de Crespin de Billy et al., 2021)). Furthermore, Kahlbaum suggested that *"negativism"* is rooted in the psychic-brain and assumed that catatonic motor symptoms such as *"flexibilitas cerea"* or *"cramp-like contorted postures of the limbs"* are caused by a mixture of mental and neurological determinants, i.e.

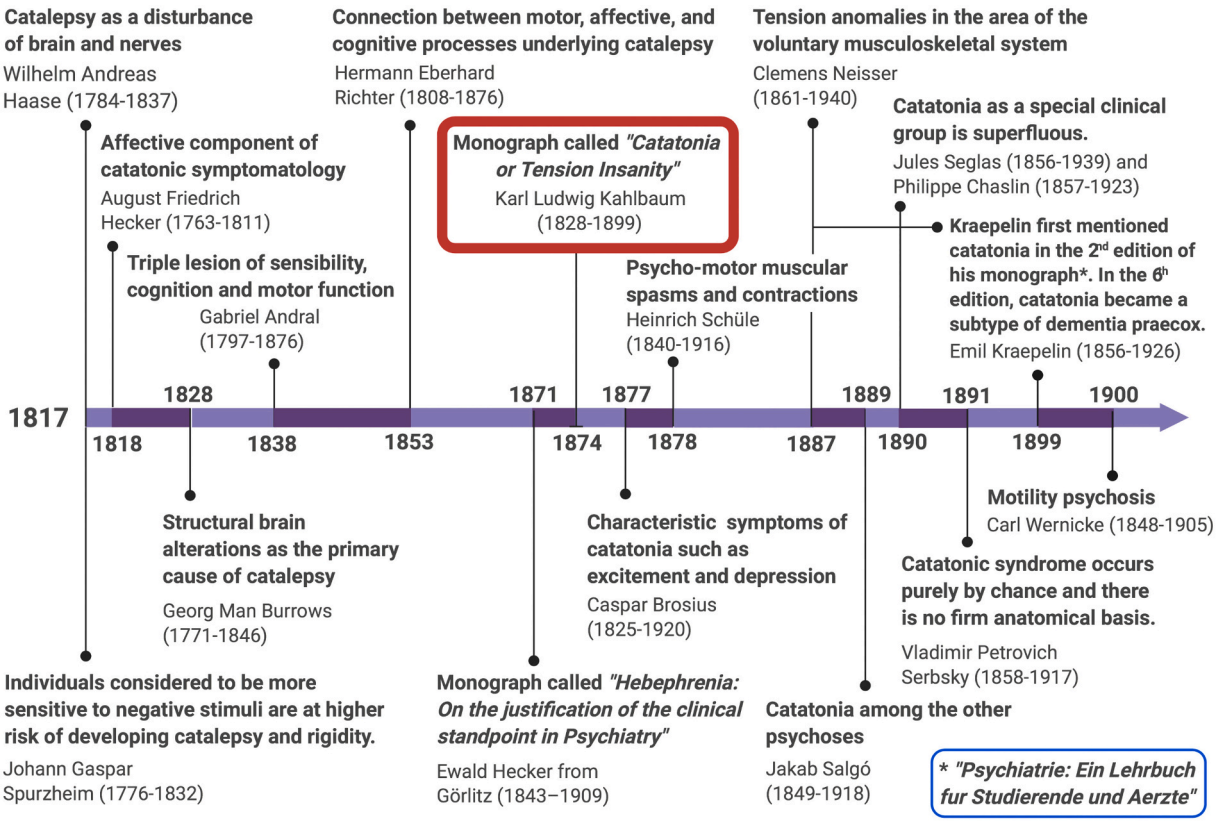

Fig. 2. History timeline showing the chronological order of authors and their concepts of catatonic symptoms before and after Karl Ludwig Kahlbaum.

reduced volition/drive and impaired cerebro-spinal functioning, respectively (see quote no. 14 in Table 1). Kahlbaum also saw a possible connection between tuberculosis-associated structural brain changes and catatonia (pp. 84–85) (see quote no. 15 in Table 1). In contrast to E. Hecker, Kahlbaum not only described the fluctuating character of affective and motor symptoms, but also observed partial decline of cognitive functions (probably due to a brain disorder) leading to a deficit and chronic state in his patients (see quote no. 16 in Table 1). This said, Kahlbaum's catatonia patients did not only have a favorable prognosis (Ungvari et al., 2010). Of his 26 patients, 7 achieved a full remission of catatonic symptoms and could be discharged home, 7 developed a chronic state and continued to stay in the hospital and 12 have died of somatic complications. What also made Kahlbaum progressive at the time was that he has not only described the clinic and neurobiology, but also recommended the therapy of catatonia with leeches, bloodletting, ferrum and quinine (p. 102; see quote no. 17 in Table 1). Although Kahlbaum did not use the term "psychomotor" (coincidently appearing the same year as Kahlbaum's monography, i.e. 1874, in both German and French literature: in Krafft-Ebing's book on melancholia and Berger's review on motor systems), he has repeatedly highlighted the close relationship between the affective, cognitive and motor domain. This is exactly what we might see in both neurologic and psychiatric movement disorders as well as sensorimotor abnormalities presented by psychiatric (especially catatonic) patients.

3.2.1. Reception of Karl Ludwig Kahlbaum's catatonia (1875–1900)

The question was less about the reality of the clinical picture than about its nature as "disease candidate". Kahlbaum and his followers (E. Hecker, Caspar Brosius, J. W. H. Jensen, W. A. Hammond, E. C. Spitzka, C. Neisser, A. Staelin and others) summarized under the uniform name of catatonia all psychotic disorders which in their course were accompanied by any locomotor manifestations. Furthermore, E. Hecker (Hecker, 1877) described a very diverse clinical picture of catatonia as well, but have dealt with the various individual symptoms in some more detail and attempted to offer an explanations for the appearance of these symptoms. In particular, E. Hecker (Hecker, 1877) focused not only on catatonic motor symptoms, but also on negativism and fear of food and drink, symptoms which to this day can lead to significant (especially somatic) complications (see quote no. 18 in Table 1). Like Kahlbaum and E. Hecker, Caspar Brosius (1825–1920), a German psychiatrist, has also emphasized that motor phenomena are characteristic symptoms of catatonia. In his publication from 1877 *"Catatonia: A psychiatric sketch"* (Brosius, 1877), Brosius agreed with Kahlbaum 's description of catatonia and hence, he further described, similar to Kahlbaum, analogous reasoning between catatonia and paralytic insanity being rooted in the quality of muscular tension (spasm/cramps vs. paralysis). However, in comparison to paralytic insanity, Brosius integrated excitement and depression of catatonic patients in a totally different ensemble. Brosius called it "symptom-complex", i.e. a coherent ensemble of and interacting manifestations that was supposed to be related to specific brain systems (≈ neurological syndrome), and was different from isolated manic and melancholic symptoms (see quote no. 19 in Table 1). A similar view of catatonia as a flamboyant disease characterized (mainly) by muscular spasms and contractions was suggested by Heinrich Schüle (1840–1916), a German psychiatrist and long-time director of the sanatorium and nursing home Illenau (see quote no. 20 in Table 1). However, in his monograph (Schüle, 1878), Schüle was skeptical about Kahlbaum's concept of catatonia and distinguished "psychomotricity" as an independent layer from the motor and the psychic one. Another proponent of Kahlbaum was Clemens Neisser (Neisser, 1887) (1861–1940), a German psychiatrist, who suggested that regular concomitant and essential partial symptoms of catatonia consists in tension anomalies in the area of the voluntary musculoskeletal system, mainly characterized by spasm (Neisser, 1887). In addition to motor symptoms, similar to E. Hecker (Hecker, 1877), Neisser also described motor and behavioral catatonic symptoms such as negativistic strivings,

sitophobia (rejection/refusal of food), mutism, verbigeration, movement and posture stereotypes, religious ecstasy and pathetically colored exaltation. In his doctoral dissertation *"Regarding Catatonia—A Contribution to Clinical Psychiatry"* (Neisser, 1887), Neisser (Neisser, 1887) emphasized the idea of catatonia as an independent disease entity, because of its unique pattern of key symptoms and signs (see quote no. 21 in Table 1).

However, Kahlbaum era might also be characterized as a turbulent era because there were both advocates and skeptics regarding the new nosologic entity of catatonia. In particular, there were numerous critical voices, which did not agree with the Kahlbaum's concept of catatonia, especially with the idea that catatonia is an independent disease entity. One of the first authors who tried to include catatonia among the other psychoses was Jakab Salgó (Salgó, 1889) (1849–1918), a Hungarian psychiatrist, in 1889 (see quote no. 22 in Table 1). Jules Seglas (1856–1939) and Philippe Chaslin (1857–1923), French psychiatrists and representatives of French descriptive psychopathology (for details see Berrios and Fuentenebro (Berrios et al., 1995)), went in 1890 (Seglas and Chaslin, 1889) even further and considered Kahlbaum's attempt to list catatonia as a special clinical group not sufficiently justified (see quote no. 23 in Table 1). Both authors also acknowledged that pathetic posturing, uniformity of movements, "spasm" and verbigerations with the fine distinction with mania are of clinical interest, but without pathognomonic character for a specific disease. Interestingly, these authors also suggested that "catatonia" (as a whole clinical picture) had for a long time been diagnosed and described under the name of "stupor" in France (i.e. melancholia attonita) and preferred Antoine Ritti's (1844–1920; French psychiatrist) opinion that Kahlbaum's catatonia would be referred to as *"Folie à double forme"* (a form of bipolarity without free intervals described in Ritti's book *"Traité clinique de la folie à double forme"* published in 1883).

Towards the end of the 19th century, Vladimir Petrovich Serbsky (1858–1917), a Russian psychiatrist and author of several books on forensic psychopathology, referred to Kahlbaum's description of catatonia as based on the symptom complex which was long referred to as *melancholia attonita* (shortened to attonity) or stupor (*stupidité*). Serbsky suggested that none of the catatonic symptoms can be described as a characteristic feature of a particular form of the disease. In his view, the composition of catatonic symptoms occurs purely by chance and there is no inner organic relationship or a firm anatomical basis (Serbsky, 1891). In 1887, in the 2nd edition of his textbook of psychiatry *(p. 339; "Psychiatrie: Ein Lehrbuch fur Studierende und Aerzte"* published in 1887), Emil Kraepelin (1856–1926) first mentioned catatonia ((see quotes no. 24 and 25 in Table 1). In subsequent editions of his textbook of psychiatry, the term *"Katatonie"* was used more often (3rd edition of 1889: 5 times; 4th edition of 1893: 15 times, 5th edition of 1896: 35 times; 6th edition of 1899: 17 times). Interestingly, in the 6th edition of his textbook, Kraepelin has described catatonia as a peculiar disease similar to hebephrenia (see quote no. 26 in Table 1) and suggested that approximately 59 % of all patients develop a severe deficit state (German "*Blödsinn*"). In line with this, in Kraepelin's later view, catatonia became a subtype of dementia praecox (see quote no. 27 in Table 1). Finally, in 1900, Carl Wernicke (1848–1905), a German physician, anatomist, psychiatrist and neuropathologist, published his book "Grundriss der Psychiatrie in klinischen Vorlesungen" (Wernicke, 1900). In this book, Wernicke has made the most comprehensive attempt to elucidate the complexities of psychomotor disturbances associated with major psychoses (see for example Foucher et al. (Foucher et al., 2020)) and introduced his concept of motility psychosis (German "Motilitätspsychose") (see quote no. 28 in Table 1). According to Wernicke's description, motility psychosis presents with akinetic and hyperkinetic poles both characterized by episodic course, good prognosis and motor symptoms which, although excessive, differ only quantitatively from normal movements, i.e., lack odd and bizarre qualities (Gazdag et al., 2017; Ungvari, 2014). Last but not least, Wernicke's and Karl Leonhard's (founders of the Wernicke-Kleist-Leonhard school of psychiatry) motility psychosis largely correspond to Kahlbaum's catatonia (Caroff et al., 2015; Peralta et al., 1997; Ungvari, 1993).

To summarize this epoch, three points are particularly noteworthy. First, Kahlbaum considered affective, motor and behavioral symptoms central to his concept of catatonia. Second, all three symptom-domains of the disorder might fluctuate over time and are presumed to have a neurobiological origin. Furthermore, Kahlbaum's catatonia refers to one of the first attempts to conceptualize a psychiatric disease entity according to empirically defined criteria. Kahlbaum's concept was rooted in precisely documented longitudinal observations of his patients, which distinguished it from all other contemporary attempts of disease classification. Third, there were both proponents and opponents of Kahlbaum's catatonia concept, but it was not until 25 years later that his thoughts were integrated into scientific practice.

4. Discussion

From the large number of historical and current materials we have reviewed here we can extract three major lessons about the history of catatonia: (1) Catatonic phenomena were described and studied decades before Kahlbaum's catatonia concept introduced in 1874 and hence, psychic-motor rather than motor causes of these symptoms were already postulated (cf. Griesinger 1845 – in our article). (2) Kahlbaum not only introduced catatonia as new psychiatric disease accounted for by an organic dysfunction but also called for revising the view of psychiatric disorders in general by highlighting the need for a comprehensive analysis of the entire clinical picture, including the dynamic course of psychiatric illness. (3) Historical texts in Kahlbaum's era provided important insights into the clinical courses, neurobiological origins and therapy outcomes of catatonia, even more precisely than current studies. In the next sections, we will draw out and discuss parallels with the Kahlbaum era and lessons from historical texts for future studies of catatonia.

4.1. Parallels between the Kahlbaum era and modern sensorimotor neuroscience

Although there is rapid progress of our understanding of catatonia, the road to this point was not straight but marked by great skepticism (Shorter and Fink, 2018). Therefore, several aspects of early pioneers and their parallels with modern sensorimotor neuroscience should be emphasized: First, several clinical scales have been established to capture catatonic symptoms (for overview see Sienaert et al. (Sienaert et al., 2011)). However, it was not until the introduction of the Northoff Catatonia Rating Scale (NCRS) (Northoff et al., 1999a) in 1999 that the affective symptoms described by Kahlbaum and his predecessors as a part of catatonic symptomatology were revived and brought into the focus of clinicians. This is crucial for the understanding of the complexity of catatonia because not only motor but also affective, social, cognitive and behavioral symptoms are characteristic features of catatonia. From a clinical perspective, this is similar to other psychiatric disorders. For instance, MDD consists not only of mood disorders, but also perceptual-sensory and sensorimotor phenomena as well as various cognitive and social symptoms. Further, dissociative disorders, post-traumatic stress disorders (PTSD), and psychotic disorders also show symptomatic overlap and are characterized by a variety of common symptoms such as social, affective, cognitive, and sensorimotor, respectively. Therefore, catatonia covering a variety of different symptom domains is paradigmatic for all other psychiatric disorders. However, this modern approach is different than the one postulated by Kahlbaum, because Kahlbaum wasn't considering catatonia as something that existed across diagnoses but rather a discrete clinical entity or a distinct disease state. Second, Kahlbaum suggested that severe affective symptoms are seen in patients with catatonia. From the above-mentioned historical quotes, it is obvious that Kahlbaum postulated a close relationship between motor and psychological processes. This

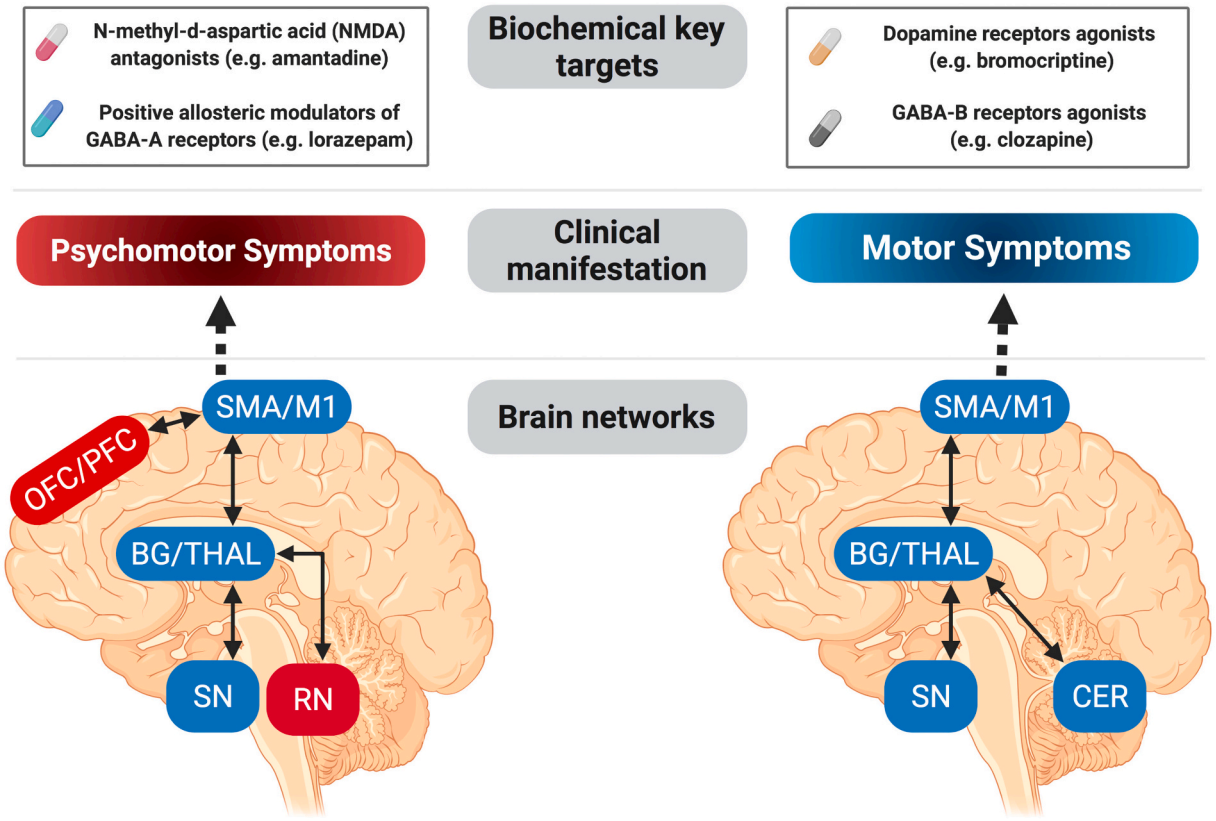

Fig. 3. Biochemical targets, clinical manifestation and associated brain networks of psychomotor and motor symptoms. OFC: orbitofrontal cortex, PFC: prefrontal cortex, SMA: supplementary motor area, M1: primary motor area, BG: basal ganglia, THAL: thalamus, SN: substantia nigra, RN: raphe nucleus, CER: cerebellum.

means that catatonia is neither a purely motor nor a purely psychological disease (Rogers, 1985). The distinction advocated by many historical (e.g. Emil Kraepelin and Eugen Bleuer (Kendler, 2020b; Kendler and Engstrom, 2017)) and current authors (Heckers et al., 2010; Tandon et al., 2013) as well as previous classification systems should be reconsidered because the evidence point towards more complex and dynamic pathophysiological mechanisms. Especially the very flamboyant motor phenomena (caused by disturbances of the sensorimotor networks) are accompanied by a number of psychological symptoms, which are caused by dynamic changes of different motor and non-motor networks and neurotransmitter systems (e.g. GABA, dopamine and serotonin) (Conio et al., 2020; Magioncalda et al., 2020; Northoff et al., 2021). Such truly psychomotor rather than simple motor mechanisms can also be observed in other disorders beyond catatonia like psychomotor excitation and agitation in bipolar disorders (Hirjak et al., 2020; Magioncalda et al., 2020; Northoff et al., 2021). This points again to the paradigmatic character of catatonia for psychiatric disorders in general.

A third important parallel to Kahlbaum's historical texts is the fact that even then the risk factors or vulnerability factors for catatonia were described. This is in accordance with the current findings from recent neuroimaging studies (Northoff et al., 2004; Northoff et al., 1999c; Northoff et al., 2000), which could show that the processing of negative emotions and distressing environmental stimuli lies in the GABAergic dysfunction. Catatonia is (among others) based on aberrant GABAergic and glutamatergic higher-order cortical and dopaminergic motor subcortical-cortical functioning (Northoff et al., 1997; Northoff et al., 2004; Northoff et al., 1999c; Northoff et al., 2000) (Fig. 3). In everyday clinical practice, there is an outstanding and almost immediate effect of GABAergic agents, e.g. lorazepam or zolpidem (Richter et al., 2010) that rapidly relieves affective catatonic symptoms (Northoff et al., 1995). Further, catatonic patients also benefit from clozapine (complex neuropharmacological properties) and electroconvulsive therapy [ECT; increase of GABA concentration in the medial prefrontal cortex (Xia et al., 2018)].

Finally, Kahlbaum has also postulated and applied a scientific method that combined the clinical (focus on variable symptoms), etiological (focus on pathological anatomy), prognostic (focus on temporal aspect and outcome), and therapeutic approaches. This approach is also seen in his monograph (Kahlbaum, 1874), which is divided into seven chapters (1: Definitions of clinical boundaries and patients' medical histories; 2: Symptomatology; 3: Etiology; 4: Pathological Anatomy; 5: Diagnosis; 6: Prognosis; and 7: Therapy). Therefore, the Kahlbaum's method approaching catatonia and the flamboyant clinical picture illustrates the need for a new approach to the diagnosis and study of psychiatric disorders and phenomena, which dissolves the boundaries of psychologic vs. organic disorders and between the different symptoms/functions/domains (affective, cognitive, motor, etc.).

One may then raise the question how such novel approach can look like in our times. The often tacitly presupposed affective or cognitive psychopathology is not sufficient as it limits our view to affective or cognitive symptoms and their neural correlates (Northoff, 2016a, 2016b). Similarly so for Phenomenological Psychopathology where the focus is on experience while leaving out more or less the brain (Stanghellini et al., 2019). What could provide a unifying framework that allows to intrinsically connect (i) psychological and motor levels, (ii) the different symptom domains, and (iii) brain and experience? One recent approach in this direction is "Spatiotemporal Psychopathology" (Northoff, 2016a, 2016b). In a nutshell, *Spatiotemporal Psychopathology* postulates that cognitive, motor, affective, social symptoms in psychiatric disorders are shaped by changes in their underlying spatial and temporal organization. For instance, neural activity may be too slow in depression and too fast in mania which, in turn, leads to the co-occurrence of their respective affective, motor, and cognitive symptoms all being characterized by abnormal slowness or fastness (Northoff

et al., 2018). Importantly, spatiotemporal features may also be shared by both brain and experience as their "common currency" (Kolvoort et al., 2020; Northoff et al., 2020). Such spatiotemporal view of psychopathological symptoms is already visible in the discussions of catatonia by Kahlbaum as well as his predecessors and contemporaries. That further underlines the actuality of those discussion for our time and their enormous potential for a need to develop a more comprehensive psychopathological framework.

4.2. Lessons learned for clinical and scientific practice

What are the take home lessons for the future of catatonia? First, we very welcome the inclusion of catatonia in the ICD-11 classification and hope that it will inspire future researchers to perform even more basic and clinical studies on this renewed entity. By introducing catatonia as a distinct diagnostic category in ICD-11, it will be easier for scientists to explore and publish the underlying pathophysiology. At this point, it's nice to see that catatonia has freed itself from schizophrenia. Now clinicians can treat and explore the clinical picture, pathophysiology, and prognosis of catatonia independently of other mental illnesses. In particular, first-person reports or citations of patients' statements will be examined in order to better understand the structure of patients' subjective (a priori) experience (e.g. following a phenomenological approach) (Sass and Parnas, 2003) before and after development of catatonia (Northoff et al., 1999b). Second, it will be easier to communicate the disease to patients. In particular, the planning of further therapies (e.g. benzodiazepines and ECT) and the process of destigmatizing of catatonic symptoms will hereby be set in motion. In addition, clinicians should also consider and recognize the diversity of catatonic symptoms and individual outcomes (rapid remission and good prognosis vs. chronic state and poor prognosis) (Ungvari et al., 2005). Nonetheless, clinicians should not equate catatonia exclusively with acutely emerging mostly stuporous catatonic state and disregard other catatonic phenomena and outcomes (Ungvari et al., 2010). Third, with the introduction of ICD-11, clinicians' (and not just experts') awareness of catatonia will be heightened as a result. Patients can benefit because they will receive guideline-based treatment faster than before. Finally, it is important to study the history of psychiatric disorders carefully to take the right clinical and research steps. It is obvious from the Kahlbaum works and citations which thoughts the authors already had 150 years ago, and which research work they carried out and how we can learn form that for our current frameworks.

4.3. Limitations

Although we identified very promising findings on the evolution of the catatonia concept across historical texts from the 19th century, several limitations apply to the present review. Considering the feasibility of the literature search, we focused only on a small number of catatonic symptoms (e.g. catalepsy, stereotypies, negativism and stupor). A further major problem remains to be solved, namely that the systematic search was to a large extent limited to journals and textbooks which were available via the 5 electronical databases. Therefore, we may not have covered all available sources on catatonia with this automated search. Another limitation is that we did not systematically reviewed articles and textbooks in French between 1800 and 1900. We have found only a small number of French authors who have published in English or German (e.g. English publication of Jules Seglas and Philippe Chaslin). However, before 1874, French authors used a very different framework whose account would have taken us out of the scope of this article. Hence, they made poor use of the term "catatonia" before the turn of the 20th century. Nevertheless, to better understand the development of the catatonia concept, a systematic research of French and other European literature would be recommended.

5. Conclusion

The theoretical contribution of this study to the literature on catatonia begins by providing a systematic review of historical texts between 1800 and 1900. From this review it is possible to see how the understanding of catatonia (motor vs. psychomotor and catatonia as an independent disease entity vs. catatonia as a subtype of dementia praecox) has developed during the 19th century. Given that history provides the context in which we operate in the present, the present review sketches the current lines of discussion and shows that they are not as new as we think they are. Instead, they can be traced to the different lines and development of discussion in the past, the history of the concept of catatonia. One may nevertheless say that such historical view is merely relevant historically but not for our present and especially neuroscience. However, that is not true. We see nowadays two dominating streams of neuroscientific research on catatonia, one more motor-based (Walther et al., 2019) and one more psycho-motor based (Hirjak et al., 2020; Northoff et al., 2021a, 2021b). Hence, history and its myriad lines clearly shape the neuroscience of our days. This is also clinically relevant as different rating scales, more motor (Bush et al., 1996a, 1996b) and more psychomotor (Northoff et al., 1999a) have been developed and are used in both research and clinical practice (Hirjak et al., 2020). Rather than considering motor and psychomotor views as mutually exclusive, one may opt for their reconciliation as the motor may be a specific subset of and embedded within the more comprehensive psychomotor framework.

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Declaration of competing interest

The authors have declared that there are no conflicts of interest in relation to the subject of this study.

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DH and MA: design of the study. DH and MA: data collection and literature search. DH and KMK: data analysis. DH and GN: first draft of the manuscript. DH, JRF, MA, LCJ, KMK, RCW and GN: interpretation of the results, discussion of the topic, writing and manuscript revision.

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