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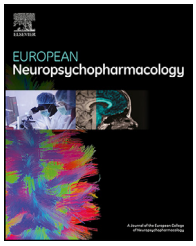

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The polysemous concepts of psychomotricity and catatonia: A European multi-consensus perspective

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Abstract
Current classification systems use the terms "catatonia" and "psychomotor phenomena" as mere a-theoretical descriptors, forgetting about their theoretical embedment. This was the source of misunderstandings among clinicians and researchers of the European collaboration on movement and sensorimotor/psychomotor functioning in schizophrenia and other psychoses or ECSP. Here, we review the different perspectives, their historical roots and highlight discrepancies.
In 1844, Wilhelm Griesinger coined the term "psychic-motor" to name the physiological process accounting for volition. While deriving from this idea, the term "psychomotor" actually refers to systems that receive miscellaneous intrapsychic inputs, convert them into coherent behavioral outputs send to the motor systems. More recently, the sensorimotor approach has drawn on neuroscience to redefine the motor signs and symptoms observed in psychoses.
In 1874, Karl Kahlbaum conceived catatonia as a brain disease emphasizing its somatic - particularly motor - features. In conceptualizing *dementia praecox* Emil Kraepelin rephrased catatonic phenomena in purely mental terms, putting aside motor signs which could not be explained in this way. Conversely, the Wernicke-Kleist-Leonhard school pursued Kahlbaum's neuropsychiatric approach and described many new psychomotor signs, e.g. parakinesias, *Gegenhalten*. They distinguished 8 psychomotor phenotypes of which only 7 are catatonias. These barely overlap with consensus classifications, raising the risk of misunderstanding.
Although coming from different traditions, the authors agreed that their differences could be a source of mutual enrichment, but that an important effort of conceptual clarification remained to be made. This narrative review is a first step in this direction.

1. Introduction

Psychomotor and catatonic signs and symptoms are back in the spotlight (Foucher et al., 2018; Hirjak et al., 2019; Walther et al., 2017). Both phenomena are even considered to be independent from psychotic and mood disorders in the ICD-11 (Reed et al., 2019), which is viewed as a return to Kahlbaum's original concept (Fink et al., 2010; Peralta et al., 2001). Recent conceptual developments and the increasing number of publications on sensorimotor and psychomotor phenomena in psychiatric disorders motivated the gathering of European collaboration on movement and sensorimotor & psychomotor functioning in schizophrenia and other psychoses (ECSP), under the auspices of the European Scientific Association on Schizophrenia and other Psychoses. In the writing of our first consensus paper (Walther et al., 2020), terminological and conceptual differences emerged in the understanding of "catatonia/catatonic" and "psychomotricity/psychomotor" vs "motor/sensorimotor". These terms are polysemous as they relate to different concepts and phenomena depending on their reference framework. This diversity of viewpoints is a richness, but only if we remain able to understand the ones of others, otherwise it will confront us with the problem of incommensurability (Kuhn, 1996). As we shall see, the opposition between Kahlbaum and Kraepelin might well have been caused by the use of the term "catatonia" to actually refer to different patients.

Hopefully, incommensurability is not a fatality (Sousa, 2010). But the cure should not be worse than

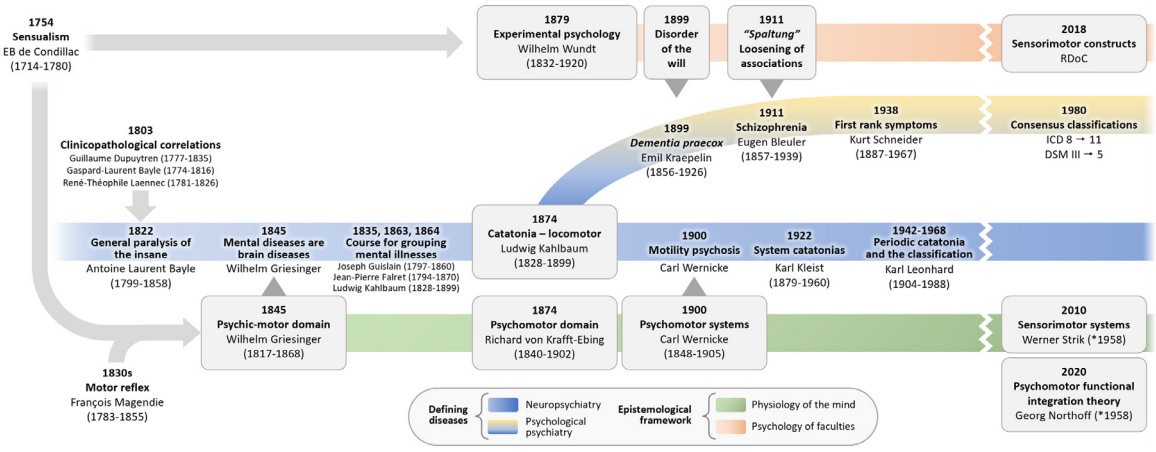

Fig. 1 Timeline. Mental diseases are defined in two ways. ***Blue***. The neuropsychiatric pathway which started with the general paralysis of the insane (Antoine-Laurent Bayle was the nephew of Gaspard Laurent Bayle). ***Yellow***. The psychological pathway according to which mental illnesses are defined by the impairment of psychological constructs. ***Green***. Physiological account of the mind. The RDoC sensorimotor constructs are more in line with psychological approach than physiological one and hence was placed next to the psychological pathway. ***Rosa***. Psychological account of mental process and content.

the illness: we must avoid the solution of a unique atheoretical consensus like ICD or DSM. Although it might be of interest in clinical practice, the "unique consensus solution" is at risk to dry out basic research. Factual science does not fit with single-mindedness and takes advantage of diversity to fasten discoveries and the selection of the most adequate model ((Foucher et al., 2020a)(Foucher et al., 2021b)). This narrative review is only one way, among others, to mitigate incommensurability while preserving our diversities. It aims to raise clinicians and researchers' awareness of these conceptual shifts by reminding their historical roots and adopting consensual accounts… but for each of them.

2. Method

A selective literature search was conducted. Original articles, books, PubMed, various dictionaries, Wikipedia and Google Scholar, up to 31 May 2021, were screened using English terms "psychomotricity", "psychomotility", "psychomotor" together with the German ("*Psychomotorik*", "*Psychomotilität*", "*psychomotorisch*"), Spanish ("*psicomotricidad*") and French translations ("*psychomotricité*", "*psychomoteur*"). Original quotes have been translated and provided in the Supplementary material together with notes (indicated by a "§" and indexed by a number). The sources included in this narrative review are not assumed to be exhaustive. Once the different concepts were defined (multiple consensus), each author ranked his preferences.

3. Results

3.1. "*Zeitgeist*": the spirit of the age

It is impossible to capture the original meaning of psychomotricity and catatonia without having a flavor of the conceptual background that framed the 19th century thinking. At that time, the general paresis of the insane (GPI) founded the neuropsychiatric current whose pathophysiological models were drawn up in the sensualism philosophy (see timeline in Figure 1).

3.2. Connecting the mental to the soma: Bayle's GPI paradigmatic model

The naturalistic framework made its first steps at the beginning of the 19th century. Diseases became considered as "natural morbid entities" being subtended by biological causes that could be demonstrated from clinicopathological correlations. The causal principle, i.e. "same cause → same effects", explained why patients were summarized in phenomenological types or phenotypes (Foucher et al., 2020a). The power of the clinicopathological paradigm was first established by the finding of pathological causes to both acute and fatal conditions, in other words, cross-sectional clinical pictures. However, mental disorders were initially thought to be an exception due to the prevailing dualistic conceptions of the times: mental disorders were thought to be of different nature than somatic illnesses.

In his seminal 1822's publication on the general paralysis of the insane (GPI) phenotype, Antoine Laurent Bayle's introduced two novelties: (i) a diachronic description and (ii) a somatic cause for a mental disorder. On one side, the term "diachronic" means that the course, i.e. how clinical pictures are changing in time, is part of the phenotypical description. On the other side, the assignment of mental signs to brain causes was a revolutionary monistic view. Dualism was so deeply anchored that, although GPI's neurological and mental manifestations were known for decades, they could not be related to the same organic origin (Pérez-Trullén et al., 2015). At best, "paralytic insanity" was primary a mental illness with psychosis, mania, and depression, and paralysis was the consequence of mental anomalies.

Bayle intended to prove that the same somatic cause accounted for both neurological and mental manifestations and his diachronic description was a case against dualism.

The essence of his argument was the parallel progression of somatic (neurological) and mental phenomena along 3 stages of increasing severity (§1): 1) ataxia and mild cognitive impairments, 2) seizures and maniac delusions, 3) paresis and dementia. The observation of the same pathological finding, i.e. a chronic meningoencephalitis, supported the adequacy of his staging model.

Bayle's monistic hypothesis has been much criticized, and it took a generation for GPI to be accepted by the medical community (Pérez-Trullén et al., 2015). But once acknowledged, the success of GPI made it the paradigmatic model of the emerging neuropsychiatric research program. In the field of neurology, it initiated a century of phenotype discoveries and refinement, thanks to pathological and latter histopathological correlations. In psychiatry, the chronic nature of illnesses let the course being considered as a key descriptor. In the middle of the 19th century, the first generation of neuropsychiatrists endorsed Bayle's diachronic model up to the point of considering staging more than a simple feature but as a promise of adequacy like Joseph Guislain in Belgium (Guislain, 1835), Jean-Pierre Falret in France (§2)(Falret, 1864), and Ludwig Kahlbaum in Germany (§4a)(Kahlbaum, 1863).

3.3. Associationism-sensualism philosophy: psychomotricity as the physiological model

The acceptance of monistic assumptions allowed the emergence of a neurophysiological model of the mind in which the concept of psychomotricity is embedded. Its breeding ground can be traced back to mid-18th century, sensualism philosophy of Bonnot de Condillac's which itself takes its root in English associationism (de Condillac, 1754). According to sensualism, mental life has three components: thought, emotion and will; all three being fed by the senses. Percepts, either of external or internal origin, elicit a first representation which leads to another and so on and so forth. The associations one forms between representations are driven by the repetition of the same experience, but also by logical "principles" like resemblance or causality (Kant, 1781). Though representations originally emerge from perceptions, they can depart from concrete to more abstract contents like words, ideas, emotions, or intentions. The flow of thought describes the inner experience of jumping from one representation to the next along these associative links. One might recognize this conceptual framework behinds Bleuler's psychological model for "schizophrenia", i.e. defective association process or "*Spaltung*" (Bleuler, 1911; Bleuler and Zinkin, 1950; Moskowitz and Heim, 2011), while by introducing the possibility of "unconscious" flows, sensualism laid the foundations of Freud's psychoanalytic model.

Ultimately, representations translate into a willingness to act from which motor behaviors ensue. Inspired from the discovery of motor reflexes by François Magendie (Tubbs et al., 2008), Wilhelm Griesinger's developed a neurophysiological theory of these sensualist view of the mind. "Psychic reflex actions" were just more elaborated associative/reflexive loops ending on a volitional or "psychic-motor" system which was ultimately driving motor systems (Griesinger, 1861). Anticipating Kraepelin's "weakness of the will", Griesinger already suggested that the weakness of "psychic-motor" processes could account for catalepsy (§3a).

The term "psychomotor" appears in the 1870s in the writing of Richard von Krafft-Ebing and Heinrich Schüle. Though it derives from Griesinger's "psychic-motor" concept, it referred to a new neurophysiological level, in-between motor and psychic domains. Here, psychomotor phenomena are a new kind of motor outputs: too complex to be of neurological origin since they have the appearance of intentional acts, yet without resulting from any psychic drive (§3b,§6 g)(Schüle, 1878). Unfortunately, the term "psychomotor" has been embezzled by the proponents of sensualist psychology to refer to the same function as "psychic-motor" or will.

3.4. Karl Ludwig Kahlbaum's catatonia

3.4.1. Catatonia designed according to the GPI model: staging and somatic-mental combination

Although Kahlbaum rejected pathophysiological modeling, he clearly developed a neuropsychiatric research program. Throughout his 1874's monograph on "tension insanity" Kahlbaum refers to GPI as the paradigmatic model to be reproduced. Beyond his pathological findings (de Billy et al., 2021), he repetitively stresses the compliance of catatonia with Bayle monistic arguments: stages of increasing severity and co-occurrence of mental and somatic phenomena (§4a)(Kahlbaum, 1874). As for GPI, all clinical pictures were already known, but described independently. Kahlbaum essentially introduced a typical episodic course in 2 to 4 stages: 1) melancholia, 2) manic rage (inconstant), 3) stuporous melancholia, 4) occasionally progressing up to a "terminal dementia" (inconstant). Stuporous melancholia is the most important and constant. At the time, its clinical picture was already well established in German, French and English psychiatry ("*mélancholie stuporeuse*" or "*Melancholia attonita*"; "*attonita*" meaning "thunderstruck") (Berrios, 1996; Kenneth S. Kendler, 2020; Edward. Edward. Shorter, 2005). Importantly, the term "stupor" means more than the absence of spontaneous activity and of active relation to the environment (American Psychiatric Association, 2013). Beyond the absence of self-initiated and reactive movements, the patient is mute, non-responsive, presents with staring, rigid mask-like facies and sometimes catalepsy. It is worth stressing that "melancholia" is not supposed to be related with a disorder of mood but is used as a mere descriptor to refer to a reduction of behavioral outputs (§4b). The current understanding of melancholia only developed in the second half of the 20th century (Paykel, 2008).

3.4.2. Somatic manifestations: muscular signs

Muscular signs are so central to catatonia that Kahlbaum even devised its second name according to them, i.e. "tension insanity" (Kendler and Engstrom, 2017). He described them as tonic resistance to passive mobilization during cataleptic states, and more phasic, dyskinetic phenomena like muscles twitches, facial spasms, choreoathetosis or even cramps-like movements that could appear on any part

of the body (§4c). Up to the second half of the 20th century, dyskinetic and dystonic phenomena were considered to be of muscular origin (Newby et al., 2017). The reason for Kahlbaum's insistence in reporting muscular signs, might not be purely driven by observation. It is clear from the reading of his monograph that he was actively looking for them (§4c). Arguably, muscular signs support his view of catatonia as another instance of a mental disorder due to a brain disease: they are for catatonia what paresis is for GPI. Surprisingly, the central role of muscular signs in the phenomenology of catatonia remains barely ever mentioned despite Kahlbaum's emphasis (Lund et al., 1991; McKenna et al., 1991; Rogers, 1991). The reason might be that most current psychopathological frameworks do not offer a reading grid to figure out which phenomena Kahlbaum was referring to, especially for the phasic component (Foucher et al., submitted).

3.5. Emil Kraepelin research program: *dementia* as deficit state

3.5.1. The concept of "deficit state"

Though Kraepelin called his classification principle "unity of course and outcome", he clearly emphasized the "outcome" (Kraepelin, 1899). In his times, prospective studies had shown GPI to be better described as the progressive buildup of permanent deficits than a stepwise progression. Hence, rather than attempting to find a sequential arrangement in the clinical pictures during the acute states, he focussed on the residual symptoms between the episodes, referred to as "deficit" or "final" state (the outcome). To him, most acute phenomena are unspecific and merely reflect generic brain reactions to an ongoing degenerative process. It is only when this process abates that the manifestations specifically related to the degenerated brain areas can be determined (§5a). An analogy can be made with herpes encephalitis in which the active pathogenic phase is generally accompanied by an unspecific confused state while the permanent deficit in episodic memory and personality changes are specific to the affected brain regions (insula, medial and polar temporal lobe) (Whitley and Gnann, 2002).

For Kraepelin the prevailing deficit ensuing acute episodes is similar to the one of GPI, i.e. dementia, which he broadly defines as the deterioration of higher mental functions. According to him, the precise symptom-constellation in which dementia appears is not important and somewhat unstable. So catatonia ought to be lumped together with paranoid dementia and hebephrenia in a single entity: *Dementia Praecox* (Jablensky, 2010). The idea is already in germ in the 4th edition of his "*Lehrbuch*" (1893), but Kraepelin only achieved the fusion in the 6th edition, published the year of Kahlbaum's death (1899). Kraepelin's interest in *dementia praecox* as a residual state is inseparable from his research program: like in GPI, *dementia praecox* should come with brain changes. Yet, given the numerous pathological examinations of patients with mental disorders over the last decades of the 19th century, other macroscopic changes like GPI's "chronic arachnoiditis" should have been reported already. Kraepelin got around the problem by talking about histopathological rather than pathological correlations (Géraud, 2007). He took advantage of the emergence of neuron staining methods to gather a unique group of brain pathologists and to initiate one of the most impressive research programs in psychiatry.

3.5.2. Psychological turn: dissolving catatonia in *dementia praecox*

The disassembling-reassembling process which occurred between the 4/5th and the 6th edition of Kraepelin's "*Lehrbuch*" has had profound consequences. We shall only raise some few points and refer the reader to recent publications for more in-depth historical accounts (Heckers and Kendler, 2020a; (Kendler, 2020)(Kendler, 2020a), 2020b; Shorter and Fink, 2018). Kraepelin states that catatonic features can be observed in all sub-forms of *dementia praecox* (Kenneth S. Kendler, 2020). While this is often regarded as the definitive argument to conflate catatonia with hebephrenia and *dementia paranoides*, it should be taken with caution. Indeed, Kraepelin does not refer to Kahlbaum's catatonia as a diagnostic entity but only to a limited set of catatonic symptoms that fit his own psychopathological constructs.

Indeed, Kraepelin's conception of the mind is not framed in neurophysiological systems, but in psychological functions or constructs (Heckers and Kendler, 2020b). Accordingly, Kraepelin poorly uses the physiology-laden term of "psychomotor", e.g. only once in the whole chapter on *dementia praecox*, without relating it to catatonia (§5b). For him, mental states cannot be reduced to neural states. While Kraepelin promoted histopathological correlations of mental disorders, he considered them of lesser importance than psychological features and never published in the field (Heckers and Kendler, 2020b). Influenced by Wilhelm Wundt, the founder of experimental psychology, Kraepelin favored introspective approaches (Steinberg, 2002). Hence, *dementia praecox* is defined as a quasi-pure mental disorder (Bräunig and Krüger, 2004): tonic muscular signs are conflated with negativism while phasic muscular signs are subsumed to peripheral somatic manifestations (§5c). Kraepelin's primacy of the mental over the soma is illustrated by the 31 pages dedicated to the "disorder of will" vs only one page to "grimacing" and "epileptiform cramps". Another example of Kraepelin's psychological orientation is the importance given to patients' introspective accounts, taking about one third of the text and presented as trustful evidence of the mental origin of the symptoms (Danziger, 1980).

According to Kraepelin, each patient suffering from *dementia praecox* should present at least one of the numerous symptoms indicative for an impairment of volition which descriptions stretch over nearly half of the chapter (31/70 pages: 44%). With the sole exception of muscular signs, all Kahlbaum's catatonic symptoms are re-interpreted as a disorder of the will so that Kraepelin merely moves them from one chapter to another; negativism, stereotypies, mannerisms, impulsive actions etc., become "psychological signs of *dementia praecox*" (§5d). Catalepsy is grouped together with echo-phenomenon in his "command automatism" or "will influenceability" construct, conceived as the mirror image of negativism, leading some authors to rename it "positivism" (Fink et al., 2010). Kraepelin's catatonia subtype remained solely characterized by catatonic excitation (manic rage) and catatonic stupor (stuporous melancholia).

3.5.3. Kraepelin's catatonia as a case of incommensurability

Kraepelin did not realize that by excluding phasic muscular signs and by "mentalizing" the other catatonic phenomena (Kendler and Engstrom, 2017) he did not refer to the same kind of patients as Kahlbaum's ones. Unfortunately, by using the same name, the meaning gap remained unnoticed, initiating decades of misunderstandings between Kahlbaum, Kraepelin and their followers. Replication studies yields strikingly consistent results when they refer to the same framework, whereas their outcomes differ with equal consistency when one study referred to Kahlbaum and the other to Kraepelin (Kenneth S. Kendler, 2020). It might be interpreted as a loyalty or groupthink bias (Kenneth S. Kendler, 2020), but the reason might be more trivial: investigators are not talking about the same group of patients. Kraepelin's shift resulted in a case of incommensurability (Kuhn, 1996): Kraepelin's catatonia did not sufficiently overlap with Kahlbaum's catatonia to confront their adequacy on evidence collected separately and on different samples.

3.6. The Wernicke-Kleist-Leonhard research program (WKL-RP)

The WKL school pursued what is arguably the most advanced neuropsychiatric research program embedded in what we nowadays call system neuroscience. Though mostly forgotten, its contribution to the concepts of catatonia and psychomotricity is substantial (Foucher et al., 2020; (Shorter, 2005b); Ungvari, 1993)(Foucher et al., 2020c)(§3). The WKL-RP acknowledges clinical entities that differ from current ones. Psychopathological descriptions poorly rely on unitary symptoms which are considered unspecific in that they can be realized in multiple ways. More often clinical reasoning is based on symptom-complexes in which signs and symptoms are arranged according to an intrinsic logic: some are "elementary" (primary), while others are "secondary". Primary manifestations directly result from the dysfunction of specific systems, e.g. hypnopompic hallucinations during sleep paralysis. Secondary symptoms emerge from normally functioning processes supplied by abnormal inputs from dysfunctional ones, e.g. delusional interpretation of a sleep paralysis as having been abducted by aliens (§6b) (Foucher et al., 2021b). Symptom-complexes must be distinguished from symptom-clusters, symptom-checklists, and DSM's polythetic approaches (3 out of 5 rule) (Foucher et al., 2020c).

3.6.1. Psychomotor phenomena are primary manifestations of dysfunctional psychomotor systems

Wernicke's "psychic reflex arc" is a more elaborated version of Griesinger's "psychic reflex actions" physiological model of the mind in which processes are disentangled from representations (§6a). Wernicke acknowledges Schüle's separation of psychomotricity from will processes and defines psychomotor systems as the ones translating various inner mental representations into outwardly oriented motor commands. Psychomotor systems account for the conversion of various drives in coherent behaviors, actions and motions sequences that can be sent to lower-level motor/sensorimotor systems. According to WKL's model, these drives are the outputs of various conscious and non-conscious upstream processes, e.g. instinctual, intentional, appetitive, orienting, reactive, emotional. Of note, praxis like object affordance or knowledge-based tool-use are intrapsychic and not psychomotor functions (Kleist, 1934).

This introduces a major shift from sensualist accounts: WKL-psychomotor phenomena refer to the signs and symptoms that are primarily accounted for by the impairment of psychomotor systems and no longer by a disorder of the will (intrapsychic). WKL-psychomotor phenomena have specific characteristics (§6 g) as illustrated by WKL-negativism. Everything Kraepelin would have referred to as negativism is considered by WKL as secondary reactions: intentional opposition due to a delusional thought, command hallucinations or the blocking of the will (§6c). WKL-negativism results from the dysfunction of high-level psychomotor systems responsible for the selection of a univocal and coherent behavior which has nothing to do with *Gegenhalten*. This is clinically tested by inducing an ambitendency. For instance, if the patient turns away from the examiner when approached (aversion), the examiner might be able to induce the voluntary antagonistic tendency by friendly and repetitively asking the patient to turn towards him, to look at him in the eyes and to take his stretched hand. In case of a psychomotor negativism, the patient fails to select a single behavior and implement both actions although they are mutually exclusive. A psychomotor ambitendency shows up as an inner struggle between the two drives: the patient attempts to comply by slowly turning towards the examiner, but seems to be hindered by an internal force that drives him to turn away again unless he is being continuously encouraged (Leonhard, 2003, 1999a). As opposed to Kraepelinian negativism, the patient is willing to comply but impeded by his failure to inhibit his aversive drive.

3.6.2. Kahlbaum's muscular signs: parakinesias and *Gegenhalten*?

In the 1920s, i.e. decades before the introduction of antipsychotic medication, Karl Kleist described many psychomotor signs with great details thanks to what is likely the first systematic use of film recordings in the history of movement disorders research ((Foucher et al., 2021a); Strauss, 1928). Some of them are known outside the WKL-community like *Gegenhalten* for instance, which likely corresponds to the tonic component of Kahlbaum's "muscular signs". Though Kleist precisely coined the term to separate this *Gegenhalten* from negativism, the two often remain erroneously conflated though *Gegenhalten* is acknowledged to be one of the most frequent form of hypertonia, i.e. a neurological rather than a psychomotor sign (§6d)(Adams, 1973). Other signs are poorly known out of the WKL-community like parakinesias which plausibly correspond to the dyskinetic component of Kahlbaum's muscular phenomena. Parakinesias consist in various deformations of the motor flow which loses its natural grace (Fish, 1962). This is sometime captured by the concept of "mannerisms" (Northoff et al., 1999). Deformations can grow up to dyskinetic- or dystonic-like additional movements of pseudo-expressive appearance ((Foucher et al., 2021a); Kleist, 1934; Leonhard, 2003). When not masked by first-generation antipsychotics, parakinesias are typically mistaken with inborn psychomotor peculiarities or tardive

dyskinesia while the concept of "grimacing" only captures the severest ones. According to WKL, parakinesias have distinctive features such as being prominent on the upper part of the face or being experienced as "self-syntonic", i.e. patients are frequently unaware or at least undisturbed by them (§6 g)(Foucher et al., submitted). Parakinesias are predictive of a progressive psychomotor deficit (§6e,f)(Foucher et al., submitted; Kleist, 1934).

3.6.3. Psychomotor ≠ catatonic: another risks of incommensurability

Aside from the mainstream psychiatry, the WKL-RP aimed at describing life-long stable "natural" phenotypes rather than "consensual" disorders (Foucher et al., 2021a). Eight decades of trial and errors have led to the description of 35 major phenotypes which contours have remained stable since the 4th revised edition of the classification in 1968 (Leonhard, 2003, 1999a). These phenotypes account for nearly 90% of patients suffering from an endogenous psychoses (Foucher et al., 2020c) and their stability throughout life is confirmed by the longest prospective test-retest diagnostic study ever conducted of 30-years interval (Tolna et al., 2001). Unsurprisingly, multi-diagnostic studies showed that the WKL-RP carves the endogenous psychosis-spectrum in a completely different way than the international classifications (Jablensky, 2011): the global concordance rate between WKL-phenotypes and ICD-DSM-disorders is about $\lambda \approx 0.5$ (Peralta et al., 2016).

The mismatch might be even worse between the eight WKL-psychomotor phenotypes and ICD-DSM catatonia (Figure 2). Only about ⅔ of the patients diagnosed as ICD-DSM catatonia, will be diagnosed for a WKL-psychomotor phenotype, i.e. the ones accounted for by the primary impairment of psychomotor systems. In the remaining third ICD-DSM catatonia, manifestations are secondary to the impairment of intrapsychic systems, e.g. thought inhibition, overwhelming anxious or ecstatic mood (§6b). The situation is even worse if we only consider the seven WKL-catatonia phenotypes: they are accounting for less than half of ICD-DSM catatonias. The reason is a matter of convention: the WKL school only uses the label of "catatonia" for phenotypes coming with the buildup of a psychomotor residuum (§6e,f,h)(Pfuhlmann and Stöber, 2001). But less than half of the patients diagnosed with either form of WKL-catatonia has an ICD-DSM diagnosis of catatonia: only 20% of patients with WKL-periodic catatonia (Krause, 2012; Stompe et al., 2002)(Foucher et al., 2020b), while many of the six other forms (system catatonias), are diagnosed as ICD/DSM autism spectrum disorder (Leonhard, 1999b). Conversely, patients with motility psychosis, a purely relapsing remitting WKL-psychomotor phenotype (non-catatonic according to WKL) are quite constantly diagnosed as ICD-DSM catatonia.

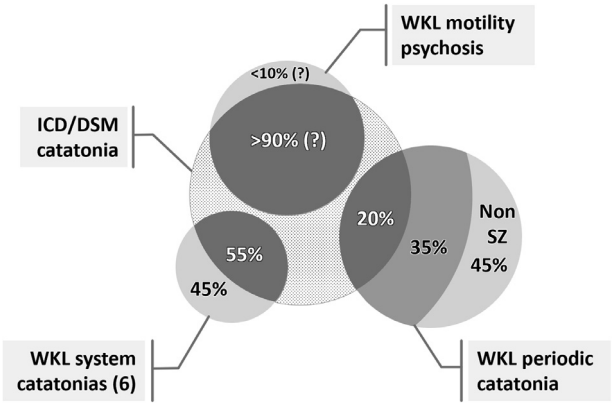

Fig. 2 ICD-DSM – WKL catatonias mismatches. The dotted surface represents the patients diagnosed for ICD-10 or DSM-IV catatonias. gray circles represent WKL-psychomotor phenotypes (§6 h). These are only accounting for about two thirds of ICD-DSM patients. The other one third are secondary to affective overwhelm (e.g. anxiety-happiness psychosis), or severe thought inhibition (e.g. confusion psychosis, cataphasia). The two lower surfaces represent WKL-catatonic phenotypes (periodic and systems) which probably account for less than half of ICD-DSM catatonic patients. Of note, after an average of 16-years since the beginning of the illness, only 20% WKL-periodic catatonia are diagnosed as ICD-catatonia (dark gray) and just over a half are diagnosed as ICD-DSM schizophrenia. Up to ¼ of them are not even diagnosed in the psychotic spectrum (light gray)(Krause, 2012; Stompe et al., 2002).

3.7. Nowadays

The neuropsychiatric orientation of Griesinger and WKL schools has been left aside by mainstream psychiatry, which embraced Kraepelin and Bleuler's psychological interpretation of catatonic and even motor phenomena (§7a). Only Karl Jaspers had a more balanced perspective in acknowledging the idea of bridging processes between mental and motor functions (§7b) (Bleuler, 1911; Bleuler and Zinkin, 1950; Jaspers, 1946).

3.7.1. Current understanding of psychomotricity: mentally driven motor outputs

Surprisingly, there is virtually no entry either for "psychomotricity" or for "psychomotility" in most UK or US dictionaries, wiki-pages or pubmed (§8a). The situation is different in Germany, Spain and France as "*Psychomotorik*", "*psicomotricidad*" and "*psychomotricité*" is the name given to a profession which re-emerged from the same sensualist philosophy in the mid-20th century. It is concerned with human development approached from a holistic perspective in which the motor behavior is viewed at the interface between the subject's psychic life, his body, and the social-physical world (§8b-d); a concept that can be related to "embodiment" in English. According to this view, "psychomotricity" is about all non-verbal motor outputs which can be interpreted as reflecting mental, intentional, affective, or emotional states. Instances of such readouts could be postures, gait, facial expressions, gestures, manners, tempo, dexterity, or gracefulness of movements. Though the definition excludes the informational content of speech, it encompasses its vocal component, e.g. prosody, pitch, loudness.

3.7.2. ICD-DSM: the a-theoretic use of "psychomotor"

Both the DSM-5 and the ICD-11 use psychomotor term as a component of two-words names that are only meaningful as a whole; there is no specific significance attached to "psy-

chomotor" per se (§9). The second words avoid any reference to a physiological understanding and remain purely descriptive: "psychomotor excitation" becomes "psychomotor agitation", and "psychomotor inhibition" turns into "psychomotor retardation". These are the only definitions appearing in glossaries. "Psychomotor features" or "disturbances" are only mentioned in the chapters about catatonia. Again, the two-words seem to form a whole in which "psychomotor" is used as a synonymous of "catatonic" as illustrated by the ICD-11 category of "psychomotor symptoms in primary psychotic disorders" (§9b, 6A25.4). In line with ICD-DSM's a-theoretical stance, it has recently been proposed to define psychomotor phenomena as any motor manifestations occurring within the context of a psychiatric disorder, including neurological soft signs and drug-induced extrapyramidal symptoms, with the sole exception of conversion phenomena (§10a)(Walther and Morrens, 2015).

3.7.3. Psychomotricity in the age of sensorimotor neuroscience

In last decades, a growing number of neuroscientists reexamined motor abnormalities in chronic psychoses. The main focus was originally on neurological soft signs framed in the "neurodevelopmental theory" of schizophrenia (King et al., 1991). Then, the specific therapeutic response of catatonia, early intervention / schizophrenia spectrum disorder studies, and the introduction of second generation anti-psychotics contributed to the renewed interest in spontaneous dyskinesias and parkinsonism (Peralta and Cuesta, 2011; Walther et al., 2020; Walther and Strik, 2012). Echoing WKL-parakinesias, both have been found to be heritable (Koning et al., 2010), to have prognostic values (Cuesta et al., 2014; Dean et al., 2018; Mittal et al., 2008; Peralta and Cuesta, 2011; Sambataro et al., 2020) and to have specific neurobiological substrates (Hirjak et al., 2019; Strik et al., 2010; Walther et al., 2017). Spontaneous parkinsonism and dyskinesias are often referred to as "neuromotor" (Peralta and Cuesta, 2011), rather than psychomotor phenomena, and mostly supposed to result from the impairment of classical sensorimotor systems, e.g. pyramidal, striatal and cerebellar systems (Bernard et al., 2014; Hirjak et al., 2020; Mittal et al., 2017; Northoff et al., 2021; Strik et al., 2010). The use of the qualifier in "psychomotor retardation", refers to the addition of sensorimotor and psychological component, i.e. cognitive, to the slow response (§10c)(Osborne et al., 2020).

On the other hand, complex catatonic behaviors like negativism often remain qualified as "psychomotor" to mean "of mental origin", which is modelized in two ways. Most models endorse the same kind of functional segregation than sensualism. They suppose the existence of a specific psychomotor/volitional function (§10c, §13b)(Walther et al., 2019) which is mapped on a network commonly including the supplementary motor area (Walther et al., 2019). Depending on the model, the latter is variously associated with striatal loops (Strik et al., 2017), lateral premotor (Foucher et al., 2018; J. R. Foucher et al., 2020b; Jacobson et al., 2018) and other medial prefrontal cortices (Mittal et al., 2017).

A recent functional integration model makes it possible to dispense with psychomotor-specialized brain regions. It defines psychomotor mechanisms by which sensorimotor functions are modulated by cognition and emotion and stipulates that they are intrinsic to every psychological processes (§10b)(Northoff et al., 2021). In this model, motor and behavioral catatonic phenomena involve the same regions than in segregated models but supposes different mechanisms, e.g. functional dysconnectivity (§13e)(Hirjak et al., 2020). Another difference is that affective psychomotor manifestations correlate with other brain regions, i.e. a right-sided orbito-frontal, frontal and parietal network (Hirjak et al., 2020). Interestingly, this is only found using the sole scale assuming that emotional phenomena are intrinsic to catatonia (Northoff et al., 1999); a nice illustration of how preconceptions influence observations (Foucher et al., 2020a).

The research domain criteria or RDoC deserve to be considered separately due to their embedment in psychological constructivism (Foucher et al., 2020a). Sensorimotor constructs (Simmons, 2018) are no exception to the rule: four sensorimotor functions were first determined and their putative biological substrates were subsequently defined by consensus. Catatonic manifestations are scattered among subconstructs together with neurological phenomena, e.g. catatonic stupor with stuttering vs catatonic immobility with tics (§11a, §13d). This implicitly suggests that catatonia is a neurological disorder, and that catatonic stupor should be distinguished from catatonic immobility.

3.7.4. Authors' preferences

The authors ordered their preferences for the four main accounts: sensualist-psychomotor systems (Griesinger), WKL psychomotor systems, sensorimotor systems (like Kahlbaum) or sensorimotor constructs (RDoC). As shown on Figure 3, the WKL-account ranked first followed by the two sensorimotor proposals which are roughly equally rated (§12).

4. Discussion

This overview warns us about the risk of misunderstanding by using the terms "catatonia" and "psychomotor" without minding the conceptual gaps existing between the different reference frameworks: the same words have multiple meanings (Foucher et al., 2020; Sousa, 2010). In the following, we shall argue that the so-called return to Kahlbaum's catatonia serves a practical purpose. However, from a basic science perspective, its reification up to considering it as a natural entity would not be less fallacious than for other, longer existing ICD-DSM disorders (Kupfer et al., 2002). Hence, we will return to the use for which "psychomotor" was first coined, and attempt to map the field of theories in order to mitigate the risk of incommensurability (Kuhn, 1996).

4.1. A return to Kahlbaum's catatonia or a new diagnostic chimera?

Following the careful reading of the original description, the alleged return to Kahlbaum's catatonia sounds at best like a figure of speech, resorting to an argument from authority to promote the creation of an independent "ICD/DSM-catatonia" entity (on the same level with affective disor-

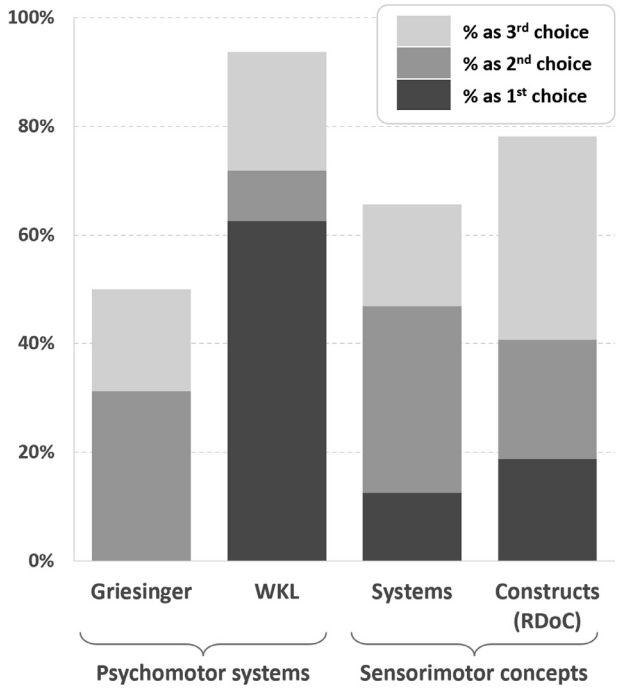

Fig. 3 Authors' preferences. Author's preferred accounts of psychomotor and sensorimotor concepts. First choices in dark gray, up to third choices in light gray. A none/other item could also be chosen (not shown). See §12 for details.

ders and schizophrenia) (Fink et al., 2010; Reed et al., 2019). In fact, this new disorder makes no mention of the features highlighted by Kahlbaum as the most important: no muscular signs (*Gegenhalten*, parakinesias) and no staging. ICD/DSM-catatonia is still a cross-sectional (episode) diagnosis and not a longitudinally defined entity (phenotype). The phenomenological description remains embedded in Kraepelin's will-construct as illustrated by the conflation of *Gegenhalten* with negativism. Though ICD-DSM definitions are using "stupor" as a criterion, their understanding of it lacks important concurrent features explicitly reported by Kahlbaum, such as the rigid (tensed) facial expression and staring.

However, this ICD/DSM diagnostic chimera of catatonic disorder serves important practical purposes: reducing the current under diagnosis of a treatable condition (Anand et al., 2019). From a diagnostic perspective, "staring" deserves to be highlighted. It is missing from the criteria though it has been shown to be easily recognized and predictive of therapeutic response (Foucher et al., 2020c; Wilson et al., 2015). Benzodiazepines and electroconvulsive therapy are widely acknowledged as effective in ICD/DSM catatonia (Pelzer et al., 2018). However, this is mostly ascertained for acute forms whereas chronic catatonic phenomena have been proposed to be much less responsive (Ungvari et al., 2010). Yet the best evidence for that used benzodiazepines in WKL-system catatonia phenotypes (Ungvari et al., 1999). Though they are highly chronic forms, they poorly overlap with ICD/DSM catatonia (Leonhard, 1999b). Hence the predictivity of chronicity and cut-off duration in benzodiazepine non-response deserve to be further evaluated. Last, the benefit of clozapine is debated. WKL-clinicians long believe clozapine to essentially advantageous in periodic catatonia (Foucher et al., 2020c; Stöber, 2000), while others have recently suggested that clozapine would be of interest for all patients diagnosed with ICD/DSM catatonia (Hirjak et al., 2021).

4.2. Theories of catatonic phenomena: sensorimotor, psychomotor and/or psychological

As important as it may be for clinical purposes, the reification of ICD/DSM-catatonia up to the point of considering it as a natural entity bears the same risk of slowing down pace of progress as for other ICD/DSM disorders (Foucher et al., 2020a). Therefore, basic science is needed to improve the adequacy of our representations with reality. In the following lines, we shall distinguish the *explanandum*, i.e. the phenomena to be explained, from their *explanans*, i.e. the etiological theories to explain them. If we return to the theory-laden usage for which "psychomotor" was first coined: "catatonic" qualifies the *explanandum*, i.e. the phenomena to be explained, while sensorimotor, psychomotor, and psychological, qualify three possible etiological levels, i.e. the *explanans* (see Figure 4 for a field map and §13 for the concept maps of specific theories). In other words, "catatonic manifestations" merely refer to the phenomena (a-theoretical *explanandum*) while "psychomotor phenomena" must be understood as "catatonic manifestations that are supposed to be accounted for by a specific psychomotor theory". To remain consistent with original proposals, we kept their use of the term "psychomotor" but warn our reader about its polysemy: as equivalent to "psychological", as combination of "psychological" and "sensorimotor" etiology or as a new level, in-between "sensorimotor" and "psychological" ones.

4.2.1. Single level theories

Here all catatonic phenomena are accounted for by a single functional level, either sensorimotor or psychological. Though not clearly stated as such, the RDoC seem to propose a quasi-exclusive sensorimotor theory of all catatonic phenomena up to complex ones like negativism or automatic obedience. Conversely, Bleuler is probably the author who has gone furthest in this opposite direction in considering all catatonic phenomena to be of psychological origin. For him, even the simplest catatonic manifestations were mentally driven, e.g. snout spasm, *Gegenhalten* (§7a).

4.2.2. 2-levels theories

Most theories of catatonic phenomena are balanced between these two extremes in acknowledging both sensorimotor and psychomotor explanations for catatonic phenomena. For instance, parakinesias or hypertonia are supposed to be sensorimotor phenomena, e.g. related to the dysfunction of striatal and/or cerebellar loops (Mittal et al., 2017; Strik et al., 2010). Conversely, more complex catatonic behaviors are qualified as psychomotor because they are supposed to be of pure psychological etiology (Mittal et al., 2017; Northoff et al., 2021; Walther et al., 2019). Most psychomotor theories are segregationist and assume the

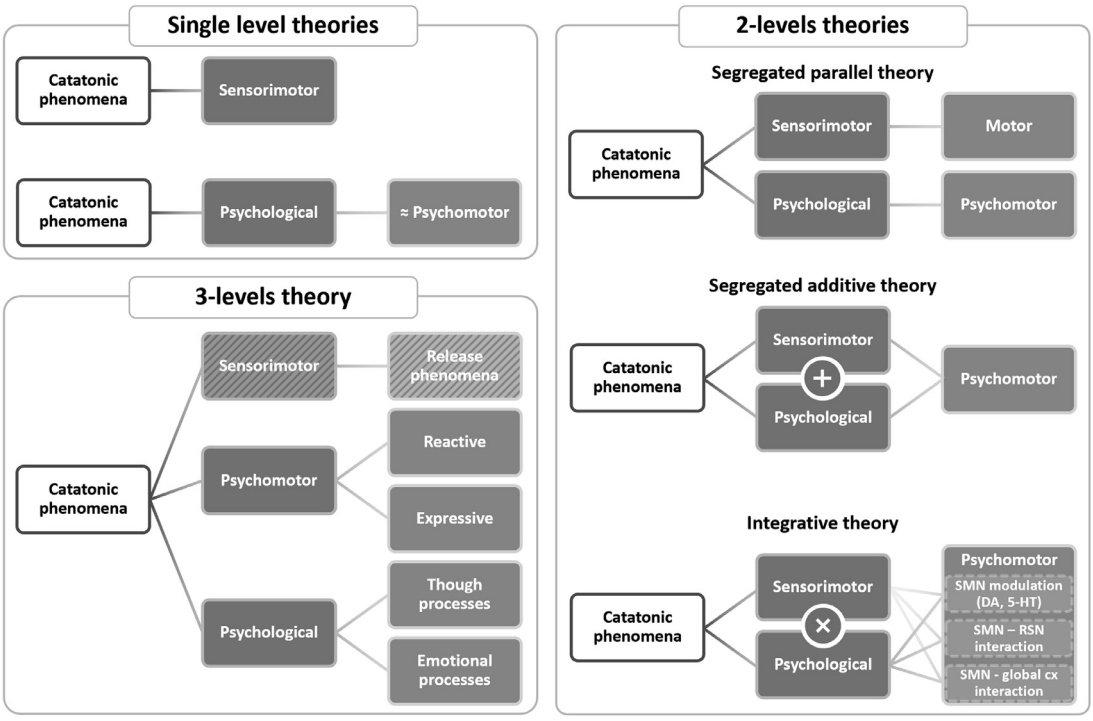

Fig. 4 Field map of theories for catatonic phenomena. See main text and §13.

existence of specific psychomotor function(s) and/or system(s) which dysfunction result in some catatonic phenomena. While psychological, psychomotricity is part of a functional hierarchy in which it is positioned between other psychological functions and the sensorimotor system. In other words, psychomotricity is believed to be the only output to sensorimotor systems compelling all psychological functions to go through them. The simplest version was Griesinger's psychic-motor theory according to which psychomotricity corresponded to conscious will (Walther et al., 2019). Recent versions rephrase the concept in terms of executive and emotional control (§13b)(Mittal et al., 2017; Northoff et al., 2020). Though mostly limited to the concept of "psychomotor retardation", here "psychomotor" refers to the addition of psychological and sensorimotor etiologies, i.e. the sum of cognitive and motor slowdowns (§10c)(Osborne et al., 2020).

The recent integrative theory has proposed that psychomotricity is neither a function nor a system, but an intrinsic property of all psychic functions (§10b, §13e)(Hirjak et al., 2020; Northoff et al., 2021). These psychomotor mechanisms are so embedded in psychological processing that the two seems to be inseparable, explaining why the model does not dissociate affective manifestations from motor and behavioral catatonic phenomena (Hirjak et al., 2020; Northoff et al., 1999).

4.2.3. Psychomotricity as an independent level: a 3-levels theory

WKL theory pushes functional segregation further in distinguishing many systems integrated in 3 hierarchically embedded levels: psychological, psychomotor, and sensorimotor. However, the level accounting for the manifestations should not be confused with the affected one. For instance, positive symptoms like parakinesias and *Gegenhalten*, are likely produced at the sensorimotor level. But these are not considered to be intrinsically impaired. Their abnormal functioning results from the release of control of upper-level systems, i.e. the downward consequence of a primary failure at the psychomotor or the psychological levels. Catatonic phenomena are constant in psychomotor dysfunctions while they are less frequently associated with psychological ones, e.g. massive thought inhibition, freezing reaction due to an overwhelming anxious or ecstatic affect (§6c).

4.3. Preventing the risk of incommensurability by directly confronting theories

Most above-mentioned theories use the same label to name different phenomena and gather them differently (Foucher et al., 2020a). This is at risk of incommensurability if we stick to the terms and do not pay attention to their meaning as for Kahlbaum and Kraepelin. The only way to get around it is the "method of multiple working hypotheses" (Chamberlin, 1965), i.e. to assess the same group of patients according to the different phenomenological scales and diagnostic frameworks. Such poly-diagnostic studies allowed to figure out the mismatch between ICD-DSM and WKL (Figure 3), and their very different correlates (Cuesta et al., 2007; Foucher et al., 2018; Peralta and Cuesta, 2005). A recent systematic review suggests that this might also be the case by simply using different phenomenological scales (§13e)(Hirjak et al., 2020). However, because studies assessed patients with only one scale, this interpretation remains to be directly addressed by multiscale confrontation studies, i.e. assessing the same patients.

5. Conclusion

If the frequently alleged return to Kahlbaum's catatonia is questionable from a historical perspective, the creation of a catatonic disorder is important for nowadays clinical practice. The condition remains insufficiently recognized while often responsive to treatments, making these patients lose chances. But ICD/DSM catatonic disorder is unlikely to be a natural entity. If we want basic science to guide future therapeutics, such real natural entities remain to be found; this is the essence of precision medicine (Foucher et al., 2020b). The present examination and discussion of historical and current theories for catatonic phenomena is provided to set the field, attract attention on some discrepancies and how we might get around the risk of incommensurability. We hope this overview will help laying the foundations for future multi-scales, poly-diagnostic studies to confront sensorimotor, psychomotor, and psychological theories of catatonic phenomena.

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Contributors

JF wrote the first draft of the manuscript. All authors contributed to and have approved the final manuscript.

Declaration of Competing Interest

Authors have no conflict of interest to declare.

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