

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

Quotations and notes

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NOTES AND QUOTES

§1 – Bayle’s general paralysis of the insane

The general paralysis of the insane (GPI) was first described in 1822 by Antoine Laurent Jessé Bayle (1799-1858) in his doctoral thesis “*Recherches sur les maladies mentales*” (Research on mental diseases) on 6 cases (Figure 1).

He raised against him the dualistic proponents especially represented by Jean-Étienne Dominique Esquirol (1772-1840), a prominent figure of the time. The staging was better specified in latter writings to defend his monistic thesis against Esquirol’s school (Figure 2). The first stage consisted in impaired gait, decreased intellectual function, and delusions. The second stage presented with seizures, mania, agitation, and violent behaviors. In the third stage, patients had incomplete paresis, having difficulties in articulation, and are demented. The Argyll-Robertson pupil (stage 1) was described latter in the mid-1860s. The connection with syphilis was suggested in the 1880s but only demonstrated in 1913.

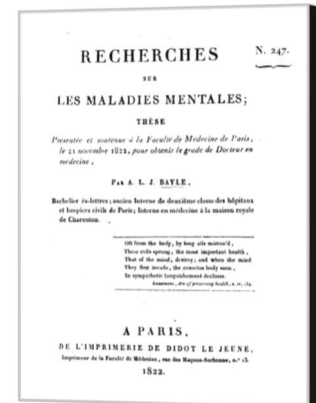


Figure 1: Antoine Bayle’s doctoral thesis (1822).

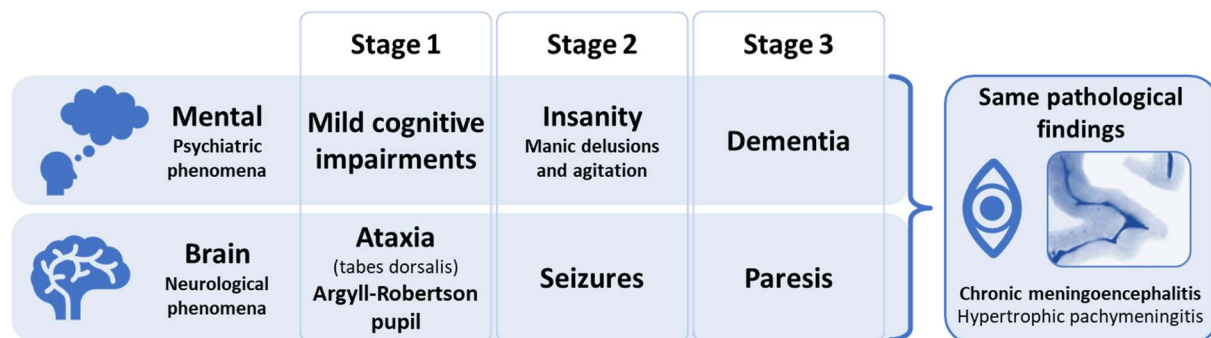


Figure 2: The mature staging model of Bayle’s general paralysis of the insane.

§2 – Jean-Pierre Falret (1794-1870)

Source: “*Des maladies mentales et des asiles d’aliénés: leçons cliniques et considérations générales*” (Mental illness and asylums: clinical lessons and general considerations) – Falret JP, 1864, Paris: Baillière et fils (accessed the 11/03/2021).

<https://gallica.bnf.fr/ark:/12148/bpt6k85072p/f35.item.texteImage>

*We could never state enough that insanity is not a single illness, which would take on the most various aspects, infinitely changeable according to individualities and circumstances, depending on the education or the environment. These incidental conditions may well account for accessory changes on the manifestations of insanity, yet these are more apparent than real and do not alter the very essence of the diseases. The most serious progress that could be made in our field consist in the discovery of truly natural species, each being characterized by the unfolding of physical and mental symptoms of **systematic course**. – p. XXXI.*

Note: Jean-Pierre Falret (1794-1870) believes in the naturalistic framework in psychiatry research. In line with the above-mentioned commitment, he described “*la folie circulaire*” (circular insanity) the same year (§04). However, the precedence for the phenotype description was disputed as Jules Baillarger

independently described “*la folie à double forme*” (double form insanity). Both were describing what Kraepelin would encompass in his manic-depressive illness.

§3 – Who coined the term “psychomotor / psychomotricity”?

Despite the very helpful contribution of Prof. Edward Shorter who oriented our attention towards the *Illenau*’s school, we are unable to provide a definitive answer to this question and would be thankful for any input on this matter.

§3a – Wilhelm Griesinger (1817-1868): “psychic-motor”

Though Griesinger is sometime credited for having quoted the term “psychomotricity” in occupational therapist textbooks, we were not able to find a single occurrence of it in his writing. Yet it is a matter of a few letters as he coined the term “psychic-motor” (“*psychisch-motorisch*”) in 1844 to refer to the same sensualist concept rephrased in his physiological model of “psychic reflex actions” (“*psychische Reflexactionen*”).

Griesinger’s “psychic reflex actions” model applies François Magendie’s stretch-reflex to sensualist account of mind*. By “reflex”, Griesinger meant “a response to a stimulus”, our current understanding, which narrows the concept to non-voluntary responses only emerged in the last half of the 20th century. According to his view, more complex behaviors were nothing more than more elaborated stimulus-response “reflex circuits” starting from the sensory organs up to the psychic apparatus following associations pathways to generate a will which projects down to the motor system to be acted. The qualifier “psychic-motor” was the physiological equivalent for the direct effect of will on motor apparatus (see concept map §13b). He used the term to illustrate how “hypotonia” could be understood in terms of “psychic reflex actions” by stuporous patients and even related it to cataleptic behavior, laying the foundation of Kraepelin’s “disorder of will” model (Griesinger, 1861).

Source: “*Die Pathologie und Therapie der psychischen Krankheiten, für Ärzte und Studierende*” (Pathology and therapy of mental diseases, for physicians and students) – Griesinger W, 1845, Krabbe (Stuttgart), translated from the 1st edition (accessed the 11/03/2021)

https://www.deutschestextarchiv.de/book/view/griesinger_psychische_1845/?p=101&hl=cataleptischem

[In stuporous melancholia, the] “lessening of will’s influence on muscles which result in great sluggishness of movement, up to cataleptic maintenance of forced positions” – p. 87.

As we were not able to find further use of this term in Griesinger’s follow-up publications, he cannot be considered as a strong promotor of the concept.

In the same vein Otto Müller (1863) referred to the “will” domain as the “*Psychomotoriums*” (De Boor, 1954). Both ascribe to the sensualist understanding.

Note: Griesinger was one of the most influential promoters of the German neuropsychiatric research program. While the citation “Mental diseases are brain diseases” seems to be apocryphal, it well reflects his beliefs anchored in his endorsement of the mind-brain identity theory. Yet he did not mean that all of what we currently refer to as “psychiatric disorders” were disorders of the brain! Conversion disorders were out of the play. He mostly talked about what will be named short after “psychoses” either of already known origin (“exogeneous” as the GPI – we would say “secondary” today) or which brain etiology or physiopathology remain to be discovered, i.e., “endogenous psychosis”.

Wilhelm Griesinger born as the youngest son of the administrator of the Stuttgart hospital. He was a precocious child, fluent in French and English. He passed the Abitur at 16 and obtained his doctorate at age

* Wilhelm Griesinger attended François Magendie’s public lessons on the physiology of reflexes in 1839 at the *College de France* in Paris. Griesinger was fluent in French and English allowing him to interact with other psychiatric figures through his travel around the world. His main books were translated in both languages.

21. He spent the following year in Paris, where he attended Magendie's lessons and the 2 next years as assistant physician in Winnenthal, one of the greatest psychiatric institution of the time headed by Zeller. He was 28 when he published his influential "pathology and therapy of mental diseases"). The epitome of a Griesinger in tune with the Prussian institutions of his time does not do justice to his rebellious, even revolutionary spirit, which he had to temper not to risk prison and to make his career. In 1860, Griesinger took over the management of the clinic for internal medicine in Zurich. He was the promotor and participated in the planning of the Burghölzli mental hospital in Zurich before holding the first chair of psychiatry in Germany in Berlin's Charité hospital in 1864.

During his two years spent in Winnenthal, Griesinger rejected the psychology of faculty and the "mentalists" style of "German romantic psychiatry". His physiological proposals were completely opposed to the mentalism which prevailed in the Asylum. These views were part of a radical package of reforms that he attempted to enforce, raising all non-university psychiatrists against him. He was not able to push them further as he suddenly passed away due to an appendicitis, only 2-years after his nomination.

Griesinger's early engagement in neuropsychiatry might find its roots in his father's murder by the family's mentally ill piano teacher when he aged 14. It is tantalizing to view this event as part of his motives for reforming asylum's psychiatry as the hostility he had to face might have made more than one step back.

§3b – Illenau's friends: Richard von Krafft-Ebing (1840-1902) & Heinrich Schüle (1840-1916)

Though many German authors credit Carl Wernicke for having coined the term of "psychomotor" ("psychomotorische") or "psychomotricity" ("Psychomotorik")*, we were unable to find a citation before the publication of his "Grundriss" in 1900. Edward Shorter observed that "psychomotor" does not appear in Neumann "Lehrbuch" (1859) which is a fair indication that the term was probably not in use at the time.

1874 - First occurrence of "psychomotor"

The earliest occurrence of "psychomotorisch" we could find comes from Krafft-Ebing's monography on "*Die Melancholie: eine klinische Studie*". published in 1874[†] (Melancholia, a clinical study, Figure 3). Considering that he referred to quite the same clinical picture as Kahlbaum's "Katatonia", the fact that both monographs were published the same year demonstrate the interest that German psychiatry had in these clinical pictures. At the time "melancholia" was merely referring to a reduction of motor outputs, without any a priori on a possible connection to a mood disorder (Berrios, 1988).

Die der Melancholie eigenthümliche, in psychischen Missgefühlen, Verlangsamung des Vorstellungsablaufs, gehindertem Umsatz der Vorstellungen in Bewegungsanschauungen und Willensimpulse begründete Störung der centrifugalen Seite des Seelenlebens, die sich klinisch als Energie- und Willenslosigkeit, Unschlüssigkeit und Unthätigkeit kundgibt, kann bis zu einer vollständigen Hemmung der psychomotorischen Leitung sich steigern (Melancholia passiva). Der Kranke, obwohl sein Bewusstsein der Schauplatz der qualvollsten Affekte, Vorstellungen und Sinnesdelirien ist, vermag sie nicht mehr in einer erleichternden That zu lösen, der quälenden inneren Spannung Luft zu machen, sei es weil das Gegengewicht hemmender Vorstellungen zu gross ist, sei es weil die treibende Vorstellung zu dämmerhaft im Bewusstsein erscheint, um als genügender Reiz zu wirken und der Leitungswiderstand auf der psychomotorischen Bahn ein zu grosser geworden ist. Für diese Vermuthung spricht wenigstens die Thatsache, dass diese Zustände immer mit einer ziemlich bedeutenden Bewusstseinsstörung einhergehen, dass ferner die wenigen dennoch zu

Figure 3: Reprint from page 38 of Krafft-Ebing's "Melancholie". The two occurrences of "psychomotor" have been put in red.

1878 – "Psychomotricity" as in-between processes

The first clarification of the concept we found is in Schüle's "Handbuch", published in 1878 while Krafft-Ebing's own account was published in his "Lehrbuch" in 1879. None of them has taken credit for the term. Both were life-friends, even before working together in "Illenau", one of the leading asylums at the time. It can be speculated that the term emerged as a simplification of Griesinger's "psychisch-motorisch" during their discussions though the meaning they gave to it differed. Krafft-Ebing listened to Wilhelm Griesinger's lectures when he spent 1863's summer months in Zurich for convalescence of bout of fever (possibly typhoid). We know from Schüle that together with Morel, Griesinger was their most commented authors during their 1864-1868 years' as junior assistants (Hauser, 1992). Both embraced Griesinger's neuropsychiatric approach and sought to link the description of clinical

* See for instance Otto Kauders' (§6a) or Wilhelm Witte's accounts (<https://www.schwabeonline.ch/schwabe-xaveropp/elibrary>)

† See p.38 for instance (online version: <https://books.google.de/books?id=O4M AAAAYAAJ>).

phenomena with brain anatomy and neurophysiology. They refer to a psychomotor domain (not systems) less as in-between than as intermediate motor functions.

Source: “Handbook of mental illnesses” (“*Handbuch der Geisteskrankheiten*”) – Schüle H. Leipzig, Germany: von Vogel; 1878. 732 p.

<https://opacplus.bsb-muenchen.de/Vta2/bsb11377925/bsb:BV014123363?page=9>

Motor disorders related to the mental domain are much more manifold than emotional disorders [...]. By “moving” the developing human mind creates the first perceptions for the understanding of the physical self as being distinct from the outside world, that is distinguishing between subject and object. The determinant role played by the mind-motor domain in the development of thinking, is illustrated by the intimate proximity between motor and intellectual centers in the cerebral cortex. There, more and more finely structured continuity of ganglion complexes and connecting conduction fibers is emerging, which is paralleled by a growing variety of [...] mentally higher forms of movement. [Whatever its level], the movement presents itself as a reflex process. The first processing centers (the spinal cord) react directly to body stimuli; higher up, other perceptions insert themselves between stimulus and action (primitive judgments). These allow to adapt the movement and its strength to the kind of stimulus and implement the instinct of self-preservation (motive). These low and intermediate motor centers (spinal cord and basal ganglia) adapt unconsciously. Conversely, mental acts occur when perception reaches consciousness and spreads in the hemispheres through multiple intermediate circuits in-between the stimulus and the behavior. [...]

Hence, the field of motor disorders can be divided into three large groups:

- 1. Anomalies of the lowest motor domain.*
- 2. Anomalies of the intermediate psychomotor domain.*
- 3. Anomalies of the psychic domain of movement.*

p. 53-55.

Anomalies of the intermediate (psychomotor) domain could either preserve the forms of a “mental act” or not. This will be latter referred to as quantitative and qualitative changes by Wernicke. For instance, according to Schüle, the movements are just increased or decreased in mania and melancholia while they preserve their natural course. Conversely psychomotor disorders do not preserve the aspect of a mental action. Schüle group immobile and reactive states, (immobility with the admixture of waxy flexibility and command automatism) and “tetany” that is characteristic of Kahlbaum’s catatonia.

The tetany.

By tetany, Arndt means all forms of motor tension phenomena which accompany various psychopathic brain states. They are more or less intense contractures of the flexor muscles of the face, neck, hand and fingers, more rarely of the lower extremities.*

Kahlbaum has already emphasized this symptom and has recently used it to characterize a “natural form of psychosis”, the so-called “catatonia”. [...] In the most severe forms, patients sit on the floor “like contracted masses”, must be dressed, undressed, and fed.

* Rudolf Gottfried Arndt (1835-1900), a psychiatrist and brain histologist who also embraced Griesinger’s neuropsychiatric program. The reference is “*Ueber Tetanie und Psychose*” (About tetany and psychosis) *Allgemeine Zeitschrift für Psychiatrie*, 1874, 30(1): 53-62 (<https://digital.zbmed.de/zbmednervpsych/periodical/structure/4238346>). The article is about a meeting held in 1872 in which the comments of Ewald Hecker (1843-1909) making the link with Kahlbaum’s catatonia are reported.

In this state they let themselves be carried away like pieces of wood, pinched and pulled, without reacting [...]; they sometimes do not simply react defensively, but brusquely, irritably, often in explosive motor discharge. This avalanche-like vehemence, if the stimulus is at all able to penetrate to a reaction, proves that even that rigid immobility, the highest degree of tetany, is only apparent. Under the external abulia there is rather an inner state of excitement, the painful excess of which leads to that psychic bondage and psychomotor tension, by cause an almost complete areflexia and the intense contracture. – p.61.

In his “*Lehrbuch*” Krafft-Ebing endorsed quite all Schüler’s account and description and cited his book. Yet he refined/simplified the definition for psychomotor disorders:

Source: Textbook of Psychiatry on a Clinical Basis for General Practitioners and Students, Vol 1 (“*Lehrbuch der Psychiatrie auf klinischer Grundlage für praktische Ärzte und Studierende*”) von Krafft-Ebing R. 2nd edition. Enke: Stuttgart, Germany. 1883, 397 p.

<https://books.google.de/books?id=UNWQET6x7FAC>

These are movements which have the character of intentional acts, which are triggered in the psychomotor centers of the brain, but which occur without the influence of the will, due to internal organic stimulation processes. – p. 89.

§4 – Karl Ludwig Kahlbaum (1828-1899)

Source: “*Die Katatonie oder das Spannungsirresein: eine klinische Form psychischer Krankheit*” (Catatonia or tension disorder: a clinical form of mental illness) – Kahlbaum KL, 1874, Hirschwald (Berlin). 118p. translated from the 1st edition (accessed the 30/12/2020)

https://archive.org/details/BIUSante_43026/

§4a – The clinical method

The first step in the clinical method was made by psychiatry with the delimitation of the so-called “general paralysis of the insane”. The fact that paralytic symptoms occur in several mentally ill patients was already known from the observations of the older somatic school. But it was only through the delineation of a special clinical picture, in which paralytic phenomena form a single series of symptoms[†], that these barely mentioned manifestations, formerly taken to be mere “complications of insanity”[‡], gained a so extraordinary importance [...]. This one form of disease[§] remained for long almost the only fruitful subject of pathological examination in psychiatry, showing that pathology needs clinical preliminary works [...]. Only the French, who discovered this first form^{**}, have so far, almost alone, proposed new clinical entities (Folie circulaire) [...]. The somatic symptoms of psychoses had already been eagerly observed and collected by psychiatrists for decades, but it was not the presence of the somatic symptom that gave this form of disease its important scientific and practical significance, but the clinical*

* Kahlbaum viewed in Bayle’s GPI the pure success of clinical observation. He does not consider the role of the inverse clinicopathological correlation in Bayle’s refinement of the GPI-phenotype.

[†] Staging.

[‡] Here Kahlbaum tackles Griesinger’s belief about GPI. He repeatedly attacks Griesinger’s position throughout his introduction without mentioning him. Six years before Kahlbaum’s publication, Griesinger took advantage of his forceful position at the head of Berlin’s Charité hospital to reform asylum’s psychiatry. His reforming attempt aborted only because of his death, but it raised all asylum’s psychiatrists against him. It is likely that Kahlbaum’s resentment against Griesinger remained vivid at the time he wrote his introduction.

[§] Phenotype.

^{**} GPI.

method of its delimitation and description, and it was due to the fact that the method used in general pathology was more practiced in somatic processes that this form of disease first attained such significance for the science of psychiatry. – p. VII.

[The clinical method is] the detailed consideration of [...] the somatic as well as the psychic, and among the psychic the intellectual as well as the affective and the ethical, the conscious and voluntary as well as the unconscious and involuntary [...]. The clinical method contrasts with earlier methods working according to uniformly psychological or one-sided somatic principles. It formerly allowed to establish the “general paralysis of the insane”. It now permits to discover a completely new type of mental illness [: catatonia]. Another of such new type is the group of illnesses to which I have given the name of juvenile insanity or hebephrenia. It has been described by Dr. Hecker according to my approach on my clinical material [...]. – p. XIII.

§4b – Stuporous melancholia (*Melancholia attonita*)

The extract is provided to allow the reader to capture the discrepancy between our current understanding of the term “Melancholia” and its meaning at the time of Kahlbaum (extract from chapter 1). According to Berrios (p.293), up to the modern period, melancholia referred to a mixture of irrationality and reduced behavioral output (Berrios, 1996a).

*Stuporous melancholia represents [...] that state in which the patient sits silently, or completely mute and motionless, with rigid facial expressions, 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 at a slight yet clearly recognizable degree. – p. 4.*

Note: “Catalepsy” has a much longer history. The first accounts are from Hippocrates who named it “catoche” (κατοχή, “I hold, I stop”, 4th century BC) while Galen used a synonymous: “*catalepsis*” (κατάληψις, same meaning, 2nd century AD). In Galen’s writing, *catalepsis* referred not only to motor arrest and posture maintenance, but its combination with the suspension of mental activity (*mentis stupor*) (Puel, 1956). In the late 14th century the term “catalepsy” referred to “an attack or abnormal state of muscular rigidity in the limbs”*, but catalepsy as a motor behavior remained sometimes conflated with stupor, i.e. a mental phenomenon, depending on the author up to the 18th century (Puel, 1956; Shorter and Fink, 2018). “*Catalepsis*” was introduced in the English medical literature by Philip Barrough who defined it as “congealing” or “taking” states and the term was changed into “catalepsy” by Robert Bayfield in 1663 (Fink and Taylor, 2003). However Kahlbaum made a precautionous use of the term because catalepsy was believed to be of psychogenic origin: epidemic of cataleptic behavior have been reported in cloister and catalepsy could be reproduced by suggestion[†] (Shorter and Fink, 2018).

Catalepsy refers to a single sign which cannot be equated with *Melancholia attonita* or catatonia. The former is a clinical picture, and the latter is a phenotype. Both are defined by much more complex symptomatology which cannot be summarized by “catalepsy” as a single phenomenon. Furthermore, as most isolated signs and symptoms, “catalepsy” is not pathognomonic. It has his own history in neurology, e.g. seizure of the SMA (Grosu and Popescu, 2015), and in psychiatry, e.g. psychogenic forms were much more common than catatonic ones during the first World War (Berrios, 1996b).

§4c – Muscular signs

Extract from chapter 2. We kept the word of “convulsion” used by Kahlbam in his description of the phenomena, but we remind the reader that the term was not so much related to epilepsy as it is

* <https://www.etymonline.com/word/catalepsy>.

† “Magnetization” or “animal electricity”. At the beginning of the 19th century, the Austrian Franz Mesmer, then in Paris, reproduced cataleptic-like behavior by suggestion.

nowadays. Indeed, Kahlbaum never talked about real epileptic phenomena (they would be too long), but as about “epileptiform states”. “Convulsion” rather refers to tonic muscle contractions.

Note: Kleist latter differentiated tonic muscle contractions from negativism under the label of “Gegenhalten”. This is a low-level motor sign giving a tense and contorted appearance to catalepsy and opposing to passive mobilization (Kleist, 1927). Last Kleist accounted for phasic muscle contractions (dyskinetic-like) as part of his concept of parakinesias (Foucher et al., n.d.).

*Let us now proceed to the somatic symptoms. Already with the last-mentioned phenomena of disturbed volitional activity it is obvious to think of a pathological innervation of the motor nerves. This assumption becomes extremely probable considering that cramps are essential symptoms of this form of disease. The spasm form of *flexibilitas cerea** is a very frequent symptom in stuporous melancholia, and illustrated by the above-mentioned casuistics, other kind of spasms do occur. The 1st case presented chorea-like convulsions of the face and extremities. Epileptiform outbreaks were reported in the file or the 2nd case. In the 3rd case hysterical spasms are mentioned involving the feet, then the arms and chin cheeks, with a sound like the ticking of a clock in the mouth; later spasmodic crying and laughing. In the 4th case, I did myself observed these spasmodic and convulsive states in the institution: initially as convulsions† of all muscles of the extremities (twitching of individual muscle in alternation), then tetanus and trismus. In the 6th case the convulsions were also observed in the institution, and like the previous case it was first convulsions* over the whole body, then an epileptiform state. In the 11th case, the convulsions occurred for several days mostly accompanying bouts of raving madness‡. Individual convulsions in the upper half of the body were observed in the case of hanging. In two cases there was no mention of convulsions in the medical report sent to the institution, and I only learned about the convulsions later by questioning the relatives. As already stated above, especially in the cases with stuporous melancholia, the frequent occurrence of convulsions is already mentioned in the literature. These convulsions occur in most cases during the first stage of the disease; only in cases 5, 6 and 11 did they occur later in the course. It is not improbable that they occur much more frequently, remaining mostly unreported to the physicians because they occurred when the patient was not yet considered to be mentally ill, or because these cramps-signs were not considered to be part of the mental illness. Some abnormality in the condition or in the functioning of the motor organs can be found in all cases. In most cases it is continuous during the whole disease, in other cases only intermittently. Some of these motor abnormalities could be regarded as psychological, even dependent on volition [...]. One part could be understood as cerebro-spinal, like *flexibilitas cerea* and the contracture-like bent postures of the limbs. Actual paralysis occurs so rarely in catatonics that they must be considered as not belonging to the picture of this form of disease. However, the patient seems poorly reactive up to quasi-complete anesthesia. Especially in cases of stuporous melancholia it is often reported how the deepest punctures are endured without the slightest expression of pain, [...]. But the abolition of the pain sensation [...] seems mostly to be due only to the abolition of reactions for motor reasons, since when the psychic reaction remits, the patients show*

* Waxy flexibility. Here Kahlbaum refer to a case presenting with rigidity. Rigidity is a more severe form of hypertonia that unfrequently occurs in the context complete akinesia and catalepsy.

† “Convulsibilität”: the term had no relation with epileptic seizures as it is frequently the case nowadays, but merely described muscular spasms.

‡ “Tobsuchtsanfällen”: bouts of manic rage.

again full sensitivity and sometimes have retained the memory of the punctures painfulness. – p. 50-2.

§5 – Emil Kraepelin (1856-1926)

We used the last edition of Kraepelin's "*Lehrbuch*" (8th) published in 4 volumes between 1909 and 1915. Dementia praecox is found in volume 3 (published in 1913).

§5a – Course and outcome

Source: "Psychiatry, Textbook for Students and Physicians" ("*Psychiatrie – Ein Lehrbuch für Studierende und Ärzte*"), 8. Auflage, I. Band. Kraepelin, E. 1909, Johann Ambrosius Barth, Leipzig. pp. 676.

Introduction

Today, a diagnosis means for us the recognition of a disease process of a certain kind underlying the given condition. It should contain much more than a summary of the just observed symptoms of the disease; it must also include a more or less definite view of the history of the development and the presumed further course of the diagnosed case. Accordingly, we can only consider a concept of disease to be complete and clearly defined when we are precisely informed about the causes, the manifestations, the course and outcome of the ailment, and finally also about the pathological anatomical changes peculiar to it. – p. 1-2.

It must be admitted that the diversity of the processes of the disease tends to emerge most clearly in the course of the illness. For this very reason, the consideration of the course and outcome of mental diseases seems to me to be of extraordinary importance for their delimitation. Essential features of the illness will imprint the clinical manifestations more permanently than secondary and accidental ones. Accompanying symptoms and transient irritation symptoms will recede behind the pure deficit symptoms caused by the essence of the pathological process itself, in the same way than it is possible to know the true seat and extent of a focal cortical lesion only from the permanent disturbances. The final states of chronic mental illness should therefore give the purest picture of the essential features which characterize the condition. To a certain extent this is indeed the case, and it is precisely the study of the deficit states that has given us extremely important information about the inner relationship of apparently quite different conditions. Nevertheless, it must be considered that under certain circumstances, once the actual pathogenic process has run its course, important symptoms may have already become completely blurred. On the other hand, deficits, which were detectable earlier, can be compensated by other parts of the brain by vicariance. It is therefore advisable, when creating forms of disease, not only to consider the last final states, but always the whole course with all its details, the more so as we have to reckon with all possible gradations in the conclusion of a disease process, depending on its severity, from complete healing to the deepest stupor. – p.9-10.

§5b – Kraepelin's usage of "psychomotor"

Due to his psychological orientation, Kraepelin refrained from using the physiology-laden term of "psychomotor". Considering only the classification parts, from the 4th tot the 8th edition, he used it 96 times for a total of 4835 pages (less than 1 time every 50 pages). When he used it, his understanding remains mostly in line with Griesinger's model of the sensualist tradition (§3a). Two third of the

occurrences are to describe hypo- or hyper-kinetic states related to mood and substance-induced disorders: “psychomotor inhibition” (34%) (“*psychomotorischen Hemmung*”) and “psychomotor excitation” (38%) (“*psychomotorische Erregung/Reizbarkeit*”). The other words that are associated to the “psychomotor” qualifier are “activity”, “performance”, and “domain” to refer to those motor outputs driven by will processes.

All occurrences of “psychomotor” (“*psychomotorisch*”) appear out of the chapter on *dementia praecox* (Figure 4), with only one exception in the part on “somatic signs”. Yet it refers to tremor, balance, and gait disorders, which are not related to muscular signs. Kraepelin does not mention Wernicke.

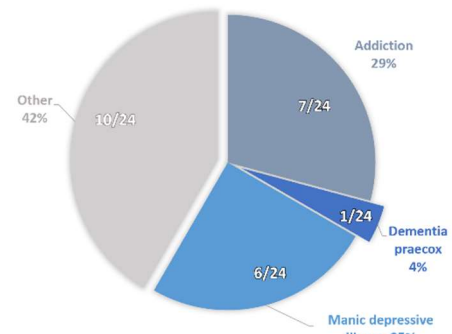


Figure 4: Kraepelin's usage of “psychomotor” as a qualifier in the 8th edition of his textbook.

Source: “Psychiatry, Textbook for Students and Physicians” (“*Psychiatrie – Ein Lehrbuch für Studierende und Ärzte*”), 8. Auflage, III. Band. Kraepelin, E. 1913, Johann Ambrosius Barth, Leipzig. pp. 667-1395

Severe and manifold disorders are found in the psychomotor field. Dufour has described disturbances of balance, staggering, adiadochokinesia, trembling, which he regards as an expression of a “cerebellar” form of dementia praecox. – p. 751.

§5c – Muscular sings

It is interesting to remark that Kraepelin did not defined catatonia by its association with what would be referred today as “catatonic signs”. These are well mentioned as attacks and chorea-like movements. Yet they are taken to be relevant for the whole “*dementia praecox*” illness, and not at all restricted to the catatonia subtype.

Source: “Psychiatry, Textbook for Students and Physicians” (“*Psychiatrie – Ein Lehrbuch für Studierende und Ärzte*”), 8. Auflage, III. Band. Kraepelin, E. 1913, Johann Ambrosius Barth, Leipzig. pp. 667-1395

The attacks already very well described by Kahlbaum and Jensen deserve special attention. They are mostly dizzy spells, fainting or epileptiform convulsions, which soon occur sporadically, soon more frequently in our patients. Less frequent are convulsions in single muscle areas (face, arm), tetany or even apoplectiform† attacks with prolonged paralysis, but I have been told of such a case a few times from previous history. Once I saw a severe attack with convulsions in the left side of the body and in the right face. Not quite seldom such convulsions are the first sign of the illness. Among others, I observed an elderly student, particularly gifted from his youth, who was suddenly seized by a deep comatose state, from which he awoke only very gradually. Apart from a slight difference of pupils, facial convulsions and hyperreflexia, he was free of other neurological symptoms. But when I examined him a few weeks later, the patient presented the distinct picture of dementia preacox, which persisted for years. Hüfler also describes equivalents of the catatonic attacks; he understands by them transient disturbances of innervation in the arm, in the facial musculature, in the tongue, feeling of discomfort or pain, vascular and pupillary disturbances, vomiting, episode of abundant sweating with or without clouding of consciousness. – p. 454.*

* “Anfälle”

† Sudden suspension, more or less complete, of brain activity.

The most peculiar disturbances are the frequently occurring spasmodic symptoms in the facial and speech musculature. A part of them completely resembles the expressive movements, the frowning, distorting of the corners of the mouth, irregular movements of the tongue and lips, twisting, opening and squinting of the eyes, in short, those movements which we summarize under the designation of face cutting or grimacing; they remind of chorea. Opsoclonus might belong to the same feature. The smacking and clicking of the tongue, the sudden sighing, sniffing, laughing, coughing are also related to them. Furthermore, we observe, especially in the lip muscles, fine, lightning-like or rhythmic twitching, which in no way bear the stamp of arbitrary movements [...]. Occasionally one sees unequal muscular tension in both halves of the face, either temporarily or for a longer period, as Hüfler has pointed out. The splayed fingers often show fine-beat tremors. Several patients constantly performed peculiarly erratic, disordered, chorea-like spreading movements, which I believe can best be characterized by the expression “athetoid ataxia”. – p. 755-6.*

§5d – Main symptoms of *dementia praecox*

Dementia praecox was characterized by a “peculiar destruction of the inner coherence of the psychic personality with predominant damage of the mental life and the will”. The translation of the table of content for the main symptoms of *dementia praecox* (p. 670-746) shows that all Kahlbaum’s catatonic manifestations but muscular signs were considered to be part of the main features of the illness.

Source: “Psychiatry, Textbook for Students and Physicians” (*“Psychiatrie – Ein Lehrbuch für Studierende und Ärzte”*), 8. Auflage, III. Band. Kraepelin, E. 1913, Johann Ambrosius Barth, Leipzig, pp. 667-1395.

*Disorders of understanding and attention, sensory illusions, (thought-sounding, thought-influencing), disturbances of orientation, consciousness, memory (memory falsifications), train of thought (loss of mental alertness, looseness of association, paralogical thought), disturbances of mental efficiency, judgment, delusions, **apathy**[†], “ataxia of feelings”, **reduction of volitional drives, command automatism (catalepsy, echolalia, echopraxia), impulsive actions, posture and movement stereotypies, mannerism, parabulia**[‡], **negativism (“autism”)**, disorders of practical efficiency, verbal expression (absence of the need to communicate, disjointedness, punning, stereotypy, verbigeration, derailments of word finding, word neologisms, akataphasia) – p. VII.*

§6 – The Wernicke-Kleist-Leonhard (WKL) pathway

This neuropsychiatric oriented way of thinking supposes the existence of various “neuro-psychological” systems, interconnected in a more or less hierarchically organized way (see concept map §13c). The clinical reasoning is therefore close to that of neurology, in which signs and symptoms are interpreted as the impairment of the smallest possible number of these systems.

Note: Leaders of the WKL pathway were Karl Kleist (1879-1960) in Frankfurt, Karl Leonhard (1904-1988) in Berlin’s Charité hospital (Est-Berlin). Prominent figures are Helmut Beckmann (1940-2006) and Gerald Stöber (1961-2017) from Würzburg (Germany), Carlo Perris (1928-2000) in Umeå (Sweden), Frank Fish (1917-1968) in Liverpool (UK), Christian Astrup (1921-1989) in Oslo (Norway), Barahona Fernandes (1907-

* “*Bulbusunruhe*”: multidirectional chaotic eye movements.

† “*gemütliche Stumpfheit*”: leisurely dullness.

‡ Kraepelin refer to the interference of “secondary” drive distorting the will’s representation of the action. The action can be improperly transformed from the start, e.g., a patient who is supposed to put on his jacket puts his legs into the sleeves, or the action is changed during its execution. In the last case Kraepelin gives examples that would be referred to as ambitendency, e.g., giving the hand, the movement stops halfway.

1992) in Lisbon (Portugal), Demetrio Barcia in Murcia (Spain), the “Budapest-connection” with Bertalan Pethő (Budapest, Hungary), Thomas Ban (Vanderbilt university, US) and Gabor Ungvari (Hong-Kong, UK, then Perth, Australia)...

§6a – Carl Wernicke (1817-1868): “psychomotor disorders of the psychic reflex arc”

Carl Wernicke did not coin the term “psychomotor” (“*psychomotorische*”) or “psychomotricity” (“*Psychomotorik*”) and even refer to Krafft-Ebing’s *Lehrbuch* in lesson 32 about their “pseudo-spontaneous or -intentional” appearance (§3b). Yet at the beginning of the 20th century his definition of psychomotor systems superseded the one of the *Illenau*’s school. For Wernicke, psychomotor systems stood in-between the neuropsychological, e.g., praxis, and the motor systems (Kauders, 1931).

Source: “Clinic and analysis of psychomotor disorders” (“*Zur Klinik und Analyse der psychomotorischen Störung*”) – Kauders O, Key Issues in Mental Health, No. 64. 1931, 132 p. We reproduced Wernicke’s graph which was assumed to be sufficiently known to all German readers at the time but might have been forgotten since (Figure 5).

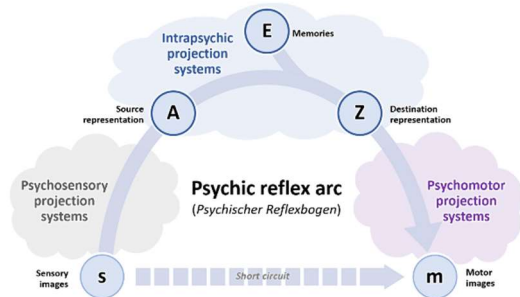


Figure 5: Wernicke’s “psychic reflex arc”. See excerpt from Kauders.

I. Introduction

Over the last decades three major directions can be distinguished in the development of psychiatric science regarding [...] the disorders of movements in mentally illnesses. The first approach is characterized by the names Wernicke and Kleist. Wernicke himself coined the term psychomotor and psychomotor disorders, which have to be understood as “psychomotor disorders of the psychic reflex arc” according to Wernicke’s model. The structure of Wernicke’s psychic reflex arc is basically the same than for primitive reflex in the flow from the sensorium (s) to the motorium (m); the passage through the psychic apparatus does not appear necessarily, but like an epiphenomenon, as this appears from the well-known graphic representation of Wernicke [...].

That disturbance or dissociation* in the reflex arc, whether it is thought of as irritation or as paralysis, as the results of which we observe the changed motor behavior in the mentally ill, begins between Z and m. It is a disturbance of the secondary identification from the target idea Z to the motor projection field m. It is a disturbance of the secondary identification from the target Z to the motor projection field m. If the marking at this point of the psychic reflex arc already sufficiently demonstrates that the psychomotor disturbance, must be thought of as extra-psychic, then this view of Wernicke’s becomes even clearer by the fact that the altered psychomotor behavior itself first forms a source for affects and thought processes. The psychomotor movements themselves, on the other hand, are executed independently “of thought and will” and, corresponding to the interruption of conduction in the psychomotor part of the reflex arc, a profound split arises between a new machine of movement obeying laws, detached from the consciousness of the personality, and the personality itself, which is thought to be, as she was confronted with her own motor disorders as a reflecting spectator and is only secondarily affected. p. 1-2.

* “Dissoziation”. Some years before Otto Gross proposed Wernicke’s “*Sejunction*” theory to be the physiological counter part of Bleuler’s “*Spaltung*” psychological theory for psychoses (Gross, 1904).

Remark: Wernicke’s definition of “psychomotricity” is inseparable from his revisiting of Griesinger’s “psychic reflex actions” and the *Illenau*’s concept of psychomotor functions (§3b). His “psychic reflex-arc” was the physiological foundation for a biological theory of psychological phenomena, a project that Wernicke’s premature death precluded him to complete (Pillmann, 2007). According to Wernicke, the representation of a psychic goal is accounted for by specific neural assemblies, distributed across the whole cortex. These were connected according to an inner-(mental)-logic while the well localized motor output centers are organized according to an “outward-logic”. The translation between the two was precisely what the “psychomotor (projection) systems” were supposed to do: converting intentional “inner representation” into “motor sequences” (Braun, 2020; Jäger, 2019; Wernicke, 2015, 1900). These systems are responsible for the translation and the fluid coordination of movements, actions and behaviors originating from conscious and non-conscious processes. Hence, they are accounting for intentional, reactive, orienting, and expressive motions, including elementary speech acts. Psychomotor functions are currently assigned to the various premotor networks (Jacobson et al., 2018).

§6b – Elementary signs and symptoms

Let us illustrate a typical WKL-diagnostic approach of a mute and stuporous patient (according to DSM-5 definition, i.e., spontaneous immobility and poor reactivity to external stimulation). This behavioral output could be multiply realized (Foucher et al., 2021a). First it might not result from an endogenous psychosis:

1. Exogeneous causes must be excluded. The condition should not be related to a medical illness such as NMDA antibody encephalitis, or induced by a substance (toxic or withdrawal effect), etc.
2. A “normal psychogenic” reaction must be excluded, to a stressful event (emotional shock) for instance. This is the classical distinction between psychoses and neuroses: whereas the formers are supposed to derive from the abnormal functioning of one or several brain systems, the later are assumed to result from normally functioning systems confronted to abnormal circumstances. Antecedents, prior personality, previous life events, culture, context, and evolution can substantiate such diagnosis.

Once these two differential diagnoses have been ruled out, we might be in the field of endogenous psychoses which means that we must be able to interpret the clinical picture by the dysfunction of selective systems in coherent “symptom-complex” in which signs and symptoms cannot be considered separately but as interacting with one another*. The clinicians have to make hypotheses about the causal relationships between symptoms in trying to minimize the number of affected systems (parsimony principle) and have to test them. The key is to find the elementary symptoms, i.e., the ones which directly result from the disordered neurophysiological process (\pm mandatory) (Wernicke, 2015). These could trigger secondary symptoms that might be physiological or psychological more variable manifestations depending on other factors, e.g., personality, life event... These are resulting from normal processes confronted with abnormal inputs. Hence, in the WKL-perspective, a mute and stuporous state might result from many other system failures than the one of psychomotor processes (see Figure 6).

1. First, the patient might not be **willing** to move and speak, due to delusional ideas for instance. Facial expression and the overall patient’s attitude might give some cues, e.g., attention towards the environment even if unresponsive (follow with the eyes), suspicious facial expression, normal automatic movements... Moreover, the patient’s motivations must be questioned as soon as the patient’s state allows it, to reduce the risk of post-hoc explanations.

* The concept of “symptom-complex” is frequently confused with the concept of “syndrome”. “Symptom-complex” used to be the accepted term to designate a set of symptoms and signs that seem to occur together more often than would be expected by chance. Conversely the term “syndrome” used to be preferred when this grouping was proved to be caused by the impairment of one organ or one system whatever the etiology. In a way, a syndrome is a validated symptom-complex.

2. Second, hypokinesia and mutism could be due to major **thought inhibition**. In such cases hypokinesia should dominate on voluntary rather than expressive and reactive movements. Movement made on order, either verbal or non-verbal, should be more impaired and sometime the patient might not even understand what he is asked for. The misunderstanding of the environment is likely to result in a perplexed or dazed facial expression. While the diagnosis might be difficult when the inhibition is severe, the picture should get clearer at some time during the resolution of the episode as the most impaired domain will be the last to improve. Retrospective questioning of the patient is generally poorly informative as such episodes typically come with poor memories.
3. Third, the patient might be overwhelmed by his **emotions**. Although depression and anxiety are more frequent, an ecstatic state is also possible. Here emotional facial expressions and bodily postures should act out the affect. Retrospectively, questioning the patient should reveal the emotional content and its possible related ideas.
4. Forth, the patient might suffer from a direct impairment of the **psychomotor** domain. Expressive movements, either inner-driven or reactive should be much more impaired than voluntary movements, e.g., empty facial expression, poor orientation toward the examiner, other psychomotor distortions.

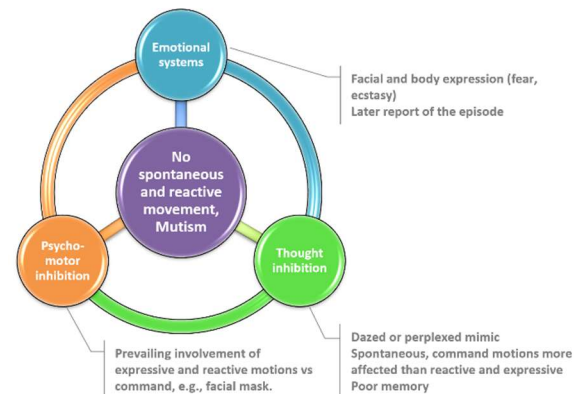


Figure 6: Not all stuporous and mute states are of psychomotor origin according to WKL. See text for the interpretation in terms of elementary (primary) – secondary symptoms.

In this perspective, diagnosing a psychomotor impairment on the mere argument that the most salient manifestations affect the motor output is misleading. The behavior can also be secondary to an abnormal thought content, an abnormal thought process or an overwhelming affect. The reverse is also true. Salient paranoid-hallucinatory or disorganized speech manifestations might indeed be secondary to a less conspicuous impairment of psychomotor functions that has to be looked for, in which case parakinesias are of major interest. This approach departs from the atheoretical stance of the ICD-DSM as allow the clinician to interpret the clinical picture. As unnatural as it seems to psychiatrists, this is nothing more than the classical neurological examination procedure. And as illustrated in the following paragraph, WKL-symptom-complexes, like neurological interpretations are testable: the clinician can (must) challenge his hypotheses by actively looking for and even inducing specific signs.

§6c – Testing the psychomotor origin for negativistic behavior: inducing ambitendency

Source: “Grundlagen der Psychiatrie” (Fundamentals of psychiatry) – Leonhard K, 1948, Ferdinand Enke Verlag (Stuttgart). 246 p. translated from the 1st edition.

Negativism is characterized by a tendency to resist, according to which the patient does not follow orders and may even do the opposite of what is desired. If one bases the assumption of negativism only on an external appearance of rejection, then the symptom is very ambiguous. Even a mere irritation can lead to this behavior. And indeed, it has become usual to talk about “negativism” even in the case of simple rejection. With such definition, this symptom is very common. But “negativism” can be used in a narrower sense, a clearly circumscribed definition which deserves being called **true** [psychomotor]

negativism*. This has nothing to do with irritation and refusal; on the contrary, according to our own observations, [psychomotor negativism] only appears in its pure form once having succeeded in overcoming the irritability which may be present at the same time. If, for instance, the patient refuses to grasp your offered hand with a hostile facial expression, then her behavior might only ensue her refusal. To disclose [a psychomotor negativism], one must put the patient in a friendly mood by talking to her sympathetically and winning her trust in any way. The patient's facial expression should unveil her change in a friendly mindset. If you now offer your hand to her again, you will recognize, in the case of a true [psychomotor] negativism, that she definitely wants to shake hands, but is not able to do so, or only very improperly. She starts to move, often making part of the way, but then get stuck and her hand returns to the starting point. The same behavior can be observed when the now friendly patient is given small orders: she tries to carry them out, might even be close to complete them when the task is routinized, but gets stuck again and again in spite of visibly good will. In this way it is possible to objectivate that the patient's resistance to act persists even when she is willing to do so. In this case, one can assume a true [psychomotor] negativism which no longer has anything to do with a psychologically understandable rejection. Seen from the outside, [psychomotor] negativism might look like a form of kinetic arrest. However, in kinetic arrest the patient is just unable to act despite the will to do so. In negativism, on the other hand, one does not observe a simple hindrance, but a real counter-acting tendency, giving the impression of a hesitancy between wanting and not wanting with the repeated coming and going of the movement. And this double tendency could also show up in the facial expression. It is convenient to speak of [true] **ambitendency**, with which the concept of [psychomotor] negativism can be associated. The concept of "blocking" is too vague as it might encompass the ones of [psychomotor] negativism, kinetic arrest, and mere affective rejection. p.31-33.

§6d – Conflating WKL-concepts with Kraepelinian constructs

Kleist coined the term "*Gegenhalten*" to separate it from psychomotor negativism. In his seminal article, he argued that *Gegenhalten* resulted from the dysfunction of low-level motor systems rather than high level psychomotor ones and could occur out of the context of negativism (Kleist, 1927). Kleist account of *Gegenhalten* is now acknowledged to refer to the same phenomenon than "paratonia" (neurology) which is supposed to be a release phenomenon commonly appearing in degenerative or vascular dementias (Drenth et al., 2020). Yet, *Gegenhalten* is still commonly conflated with Kahlbaum's negativism (American Psychiatric Association. Task Force on DSM-IV., 1994; Bush et al., 1996; Carroll et al., 2008; ICD-10 World Health Organization, 1992; Rajagopal, 2007).

Source: "*Grundlagen der Psychiatrie*" (Fundamentals of psychiatry) – Leonhard K, 1948, Ferdinand Enke Verlag (Stuttgart). 246 p. translated from the 1st edition.

"Gegenhalten" (Kleist) is to be separated from negativism. It is no longer a form of goal-directed tendency but is of reflective kind. It is tested in such a way that one tries to bend the stretched forearm of the patient or to stretch her bent one with a gentle pressure. If "Gegenhalten" is present, then a counter-pressure sets in, which increases when one tries to overcome it. The stronger the counter-pressure is, the faster one can increase it without yielding. As a rule, however, there is no resistance to a movement that is strong from the start. "Gegenhalten" might largely account for the "psychological pillow"

* In the main text, we refer to this concept as "psychomotor negativism".

phenomenon, i.e., the fact that some patients, when lying on their backs, keep their heads raised from the surface. p.33.

The same could be said about *Mitmachen* and *Mitgehen*. The two are frequently supposed to mean the same and conflated with Kraepelin's "command automatism" (Krüger et al., 2003). Yet Kleist's *Mitmachen* is a form of active assistance of the same low-level origin than "*Gegenhalten*"; and has been renamed "facilitory paratonia" in the neurological literature (Beversdorf and Heilman, 1998). Conversely, *Mitgehen*, e.g., Anglepoise lamp sign, confers to higher level psychomotor dysfunctions, closer to the ones impaired in WKL-negativism (Foucher et al. submitted).

§6e – Quantitative vs qualitative dysfunctions: why distinguishing catatonia?

The WKL-school emphasize the distinction between **quantitative** and **qualitative** dysfunctions because of their prognostic significance. This stems from the *Illenau's* school to which Wernicke refers to in his "*Grundriss*". Quantitative changes is when neurological systems are too much or insufficiently excited or inhibited resulting in functional excess or deficit which might result in psychomotor hyperkinesia or akinesia for instance. Such changes can be accounted for by the dysfunction of regulatory processes rather than the higher-level systems themselves. These are dysregulated but operate normally. This is not indicative for a degenerative process and for the buildup of more permanent changes. Hence, this can be observed in purely relapsing remitting phenotypes, e.g., motility psychosis.

At the opposite, systems might be **qualitatively** changed in which case they will not operate normally. In the psychomotor domain, aversion, ambitendency, *Gegenhalten*, *Mitgehen*, or parakinesias cannot be explained by the mere hyper- or hypo-functioning of psychomotor processes (Kleist, 1934). These are qualitative changes indicative for the disintegrations of higher-level processes, supposedly related to a neurodegeneration limited to specific neuronal populations like in some neurological diseases, e.g., familial adult myoclonic epilepsy (Corbett et al., 2019; Florian et al., 2019; Zeng et al., 2019). Hence qualitative changes are predictive for the buildup of permanent signs and symptoms that were not present before the beginning of the illness, i.e., an acquired functional deficit. In case psychomotor systems are implicated, the phenotype will be given the name of catatonia (Foucher, 2021). Such qualitative psychomotor phenomena are catatonic phenomena.

§6f – What is a good and what is a bad prognosis?

Central to Kahlbaum-Kraepelin's misunderstanding was the question of prognosis (Kendler, 2020). Kahlbaum demonstrated that catatonia did not have the bad prognosis alleged by Kraepelin, and the latter provided evidence for the opposite. It depends on how they define a good prognosis: a symptom-attenuation compatible with an institutional discharge (Kahlbaum) or a complete functional recovery (Kraepelin). And Kraepelin even integrated this criterion in his definition.

It is the same for ICD-DSM vs WKL catatonias: ICD-DSM catatonias are claimed to have a good prognosis (Fink et al., 2010), whereas it is not the case for WKL-catatonias (Foucher et al., 2020). Again, this is a matter of definition: ICD-DSM's pragmatic orientation and cross-sectional approach defines "prognosis" as the symptomatic or the functional remission after the episode. Conversely, the WKL-school defines phenotypes of "poor-prognosis" as the ones coming with the build-up of a "deficit" even if it occurs in the long term. In catatonias, this is defined by the occurrence of any new psychomotor dysfunction, i.e. appearing in the course of the illness, whatever its functional consequences (§13c)(Foucher et al., 2020).

§6g – Historical development of psychomotor phenomena and of their characteristics

By the reading of Schüle, "psychomotor" became to refer to a new neurophysiological level, by questioning the limit between sensorimotor and psychological phenomena. Should we only use "psychological" to refer to those motor outputs related to conscious contents? If yes, how to interpret catatonic phenomena that have the appearance of intentional acts but unrelated to any conscious

drive (§3b)(Schüle, 1878)? The concept of “unconscious”, introduced in the 18th century by Friedrich Schelling, was already well established in Germany (Otabe, 2019) and Schüle’s psychomotricity may have been a way to account for the motor outputs from the unconscious.

Wernicke clearly defined psychomotor processes as a new level in the top-down hierarchy of output systems, i.e. in-between psychological and sensorimotor levels. The limit between psychological and psychomotor phenomena could no longer solely rely on the consciousness criterion because for Wernicke all brain processes could be non-conscious including psychological ones (§6a). The distinction between the two relies on the interpretation of the global clinical picture in terms primary and secondary organization of signs and symptoms (§6b) and how it can be tested (§6c). Kleist further elaborated this 3-levels theory in enriching the criteria regarding the limit between psychomotor and sensorimotor phenomena. The intentional appearance of the former was not always that simple to assure, e.g. at the beginning, chorea may have the same pseudo-expressive appearance than parakinesias. He reported that psychomotor phenomena were not experienced as self-alien, i.e. they remain self-syntonic, while sensorimotor ones are generally experienced as pathological, i.e. self-dystonic (Foucher et al., n.d.; Kleist, 1934).

§6h – WKL psychomotor and catatonic phenotypes

The differences between Kahlbaum, Kraepelin, ICD-DSM, and WKL concepts are too numerous to be reviewed (see table).

Table - WKL psychomotor phenotypes

	Kahlbaum	Kraepelin	WKL							
	Catatonia	<i>Dementia praecox</i>	Motility psychosis (not a catatonic phenotype)	Periodic catatonia	System catatonias					
					Parakinetic	Pseudo-compulsive	Proskinetik	Negativistic	Short-circuit-speech	Absent-minded
Course	Mostly favorable	Residuum	Relapsing-remitting	Relapsing-progressive	Progressive					
Catalepsy	X	X	X	X						
Waxy flexibility	X	X	X*	X						
Stupor	X*	X	X	X*		(X)			X	X
Agitation	X	X	X	X				X		X
Mutism	X	X	X	X		(X)				X
Negativism	X	X	X ^{ICD-DSM}	X				X		
Posturing	X	X		X				X ^{ICD-DSM}		
Mannerism	X	X		X		X ^{WKL}				
Stereotypies	X	X		X						
Grimacing	X	X		X	X					
Echolalia		X		(X)			(X)	(X)	(X)	
Echopraxia		X		(X)			(X)	(X)	(X)	
Other	Muscular signs (≈ parakinesias, <i>Gegenhalten</i>) * With staring and stiff facial expression		Hyperkinesia and akinesia, empty facial expression * May be hypotonia	Parakinesias, admixture of hyperkinesia and akinesia * With staring and stiff facial expression	Parakinesias, jumpiness of thoughts	Rituals, rigidity of posture and movements	Responsive grasping, <i>Mitgehen</i> , hyper-suggestibility, verbigeration	Aversion, PM ambitendency	Short-circuit speech, autism, no facial expression	Distracted, sluggish speech

Table: Symptoms reported in the various WKL-psychomotor phenotypes. We used the 12 symptoms checklist of the DSM-5 definition. X: when using DSM-5's understanding of the terms (see * in "other" for extended version). (X): when the manifestation is reasonably frequent but non-specific. X^{ICD-DSM}: when it is only true using ICD-DSM's definitions. X^{WKL}: when it is only true using WKL definition. It is likely that the majority of Kahlbaum's initial cases would match the WKL-phenotype of periodic catatonia.

§7 – Followers

At the beginning of the 20th century two major streams imprinted the way motor symptoms were interpreted. Both gave primacy to the mind and especially to that part which remained under the influence of consciousness.

§7a – Eugen Bleuler (1857-1939)

Bleuler completed the psychologizing turn initiated by Kraepelin and rejected the neuropsychiatric orientation of Wernicke. He went so far as to attribute a mental cause to motor symptoms of the catatonic form of schizophrenia which is illustrated in his account of the “snout spasm” (“Theories about symptoms” chapter).

Source: “*Dementia Praecox or the Group of Schizophrenias*” (“*Dementia praecox oder Gruppe der Schizophrenien*”), Bleuler, E., 1911. in: *Hanbuch Der Psychiatrie*. Aschaffenburg (Ed.), Franz Deuticke, Leipzig, p. 420.

4. motor symptoms.

Observations which compellingly pointed to an origin of motor symptoms in specifically altered motor cortical centers or at even subcortical locations have not yet been published. So far, the localization attempts remain inconclusive. As far as we know, all motor symptoms are dependent on psychic influences. Those which can be analyzed can often be completely explained by psychic means. [...] Choreatic, athetotic, tetanic phenomena are something quite different from the motor symptoms belonging to schizophrenia. Nor have I ever confused the movements of idiots with catatonic ones; they obviously have their purpose in themselves, they are an expression of the joy of movement inherent in all of us. – p.361.

If the Wernicke school speaks of such, it is only because it has made a concept of choreatic movements that goes beyond what is seen in the chorea forms. The localization into single muscle groups cannot be justified anatomically as plausibly as psychically, quite apart from the fact that in some individual cases the psychic genesis has to be proved. A spasm of the snout is more explicable as a sign of contempt than as a localized tonus of the prominent extensors of the lips, and the sudden fluctuations of intensity from zero to maximum under psychic influences are only understandable if at least the triggering of the symptoms is a psychic one. – p.362.

§7b – Karl Jaspers (1883-1969)

Jaspers did not discuss the nosological position of catatonia, it was out of the scope of a book focused on phenomenologically oriented psychopathology. Yet he addressed the question of “psychotic motor-phenomena”. Though criticizing Wernicke’s “psychic reflex arch”, he acknowledged that there should be something to fill the gap between the subjective awareness of Will and neurological systems translating it into motility. Yet to avoid the use of the theoretically imprinted term of “psychomotricity” he prudently talked about “motor action”. However, if on the one side he implicitly endorsed conscious awareness to differentiate between Will from “motor action” phenomena, he provided no cue on how to differentiate between “motor action” and “motility” disorders.

Source: “General psychopathology” (“*Allgemeine Psychopathologie*”) Jaspers, K., 1946., 4th ed. Springer, Berlin Heidelberg.). Retranslated.

4. Motor phenomena

From the point of view of the psychic reflex arc all psychic events merge at last into motor phenomena, which assist the final inner elaboration of stimuli into the external world. From the point of view of inner meaning, subjective awareness of Will translates itself into movement. This volitional act is associated with an extra-conscious motor mechanism, which gives this act of the will the ability to work.

We can, therefore, examine the many, often grotesque, movements of mental patients from two points of view. Either we try to acquaint ourselves with the disturbances of the motor mechanism itself, which can sometimes show disturbances independently of any psychic anomaly and this is the approach adopted by neurology. Or we try to get to know the abnormal psychic life and the patient’s volitional awareness, which these conspicuous movements exhibit. In so far as we know the meaningful connections, the movements become behavior we can understand, for instance, the delight in activity shown by manic patients in their exuberance, or the increased urge to move shown by patients who are desperately anxious. Somewhere between the neurological phenomena, seen as disturbances of the motor-apparatus, and the psychological phenomena, seen as sequelae of psychic abnormality with the motor-apparatus intact, lie the psychotic motor-phenomena, which we register without being able to comprehend them satisfactorily one way or the other. Neurological phenomena are termed disturbances of motility, the psychotic phenomena are termed disturbances of motor functions. Psychological phenomena are not conceived to be primary motor phenomena but are to be seen as actions and modes of expression which have to be understood. – p. 150-151.

§8 – Current views

§8a – English definitions

These are embracing the sensualistic understanding.

(The American Heritage Dictionary of the English Language, fourth edition, 2000)

[Psychomotor means] of or relating to movement or muscular activity associated with mental processes.

Collins dictionary (<https://www.collinsdictionary.com/dictionary/english/psychomotor>)

of, relating to, or characterizing movements of the body associated with mental activity.

§8b – German: “*Psychomotorik*”

Definitions

Most definitions embrace the sensualist understanding. It endorses two meanings. We were only able to find a “medical” definition for “psychomotricity” in German, fitting with our matter of interest. All sources agree to name “*psychomotorik*” all non-verbal motor outputs which can be interpreted as reflecting mental, intentional, affective, or emotional states of others. Instances of such readouts could be postures, gait, facial expressions, gestures, manners, tempo, dexterity, and gracefulness of movements. If the definition excludes the informational content of speech, it encompasses its vocal component, e.g., prosody, pitch, loudness etc.

Only a few sources would limit the concept to those states which are consciously experienced. Yet, this feature might be of interest as a criterion to discriminate psychomotricity from other kind of motor outputs, e.g., “neurological” phenomena.

Source: The German Wikipedia page (accessed the 01/01/2021):

<https://de.wikipedia.org/wiki/Psychomotorik>

Dorsch dictionary of psychology

<https://dorsch.hogrefe.com/stichwort/psychomotorik>

Duden dictionary

<https://www.duden.de/rechtschreibung/Psychomotorik>

The totality of all movements (e.g., walking, speaking, or facial expressions) that are voluntarily controlled, consciously experienced, and influenced by mental moments.

Examination of psychomotricity

The observation and testing of psychomotricity is said to be part of the general psychiatric examination (appearance, state of consciousness, orientation, thought process and content, mood, cognition...). Again, in this educational article is mostly apprehended from a sensualist perspective.

Source: “Brief Guide to Psychopathology: An Orientation Guide for Observation and Documentation of Mental Abnormalities” (“Kurzleitfaden Psychopathologie: Eine Orientierungshilfe bei Beobachtung und Dokumentation psychischer Auffälligkeiten”) - Richartz-Salzbürger E et al. Psychiatrie. 2006; 2:47-52).

Drive: active, energetic, buoyant, spontaneous, agitated, or apathetic, indecisive, inhibited, increased, reduced, listless, restless, impoverished, diminished.

Gait: elastic, energetic, springy, powerful, swinging, or exhausted, small-stepped, dragging, shuffling, tripping, unsteady, tense.

Expressive behavior: adequate, adapted, balanced, lively, loose, mimic modulated, resonating, sober, rounded or inexpressive, driving out, bizarre, angular, constricted, excited, expansive, flat, bound, locked, grimacing, inadequate, cool, mask-like, with tics or parakinesia, tired, mutistic, sparse, sparing, rigid, dull, apathetic, over-expressive, maladjusted, uncontrolled, unmodulated, pinched, cranky.

Voice: articulate, accented, clear, dialect, differentiated, high-pitched, clear, loud, whispering, artificial, breathy, hoarse, quiet, monotone, rumbling, unclear, unintelligible, washed out.

Manner and manners: empathetic, dexterous, polite, correct, natural, confident, impartial, apathetic, pushy, self-conscious, tomboyish, submissive, aloof, affected, indifferent, condescending, affable, perplexed, shy, stiff, insecure, submissive.

Will: impression of strength and dynamism (“sthénicité”), persistent, persevering, controlled, assertive, consistent, unbending, purposeful, indifferent, negativistic, indecisive, dogged, weak-willed, wormy.

Contact behavior, communication style, relationship building: maintaining eye contact, detailed, differentiated, colorful descriptions, willing to communicate, open, warm-hearted or reserved, rejecting, avoidant, hostile, help-seeking, cool, affable, distrustful, closed, sure, unsure, trustful, distrusting

§8c – Spanish: “*psicomotricidad*”

The definition of “*psicomotricidad*” has 3 meanings according to the dictionary of the Royal Academy (*Diccionario de la Real Academia*). The two first meanings are in agreement with sensualist school since motor domain is understood as origin, integration of mental, intentional, affective, or emotional states. The third meaning echoes the influence of the psychomotricity as discipline.

Source: Diccionario de la Real Academia (RAE)

<https://dle.rae.es/psicomotricidad>

Motility of psychic origin, integration of motor and psychic functions, and set of techniques that stimulate the coordination of motor and psychic functions.

According to the Spanish version of Wikipedia, the term “*psicomotricidad*” refers both to the interaction between the psychic and the motor domains and the discipline.

Source: Wikipedia

<https://es.wikipedia.org/wiki/Psicomotricidad>

The term psychomotor constitutes in itself, starting from its linguistic analysis, a dual construct that corresponds to the Cartesian mind-body duality. It reflects the ambiguity of the psychic (psycho) and the motor (motor), as well as the complex relationships between these two poles.

Psychomotricity is a discipline that, based on an integral conception of the subject, deals with the interaction that is established between knowledge, emotion, movement and its greater validity for the development of the person, their corporeity, as well as of his ability to express himself and relate to the world around him. His field of study is based on the body as a construction, and not on the organism in relation to the species.

§8d – French: “*psychomotricité*”

The definition remains in line with the sensualist tradition though under the influence of the psychometricians* it rehearses the reverse influence that the body and kinesthetic sensations may have on the psyche.

Source: Wikipedia

<https://fr.wikipedia.org/wiki/Psychomotricit%C3%A9>

Psychomotricity groups together motor functions that are related to thought, psychology and brain functions. It is a discipline that opposed to the dichotomy between the body and the psyche. It is rooted in a system of regulation, feedback and homeostasis that seeks to increase coherence between body and thoughts.

§9 – International classifications

The ICD and the DSM still embrace the a-theoretical stance. Here “psychomotor” is no more used as an *explanans*, i.e. the mechanisms of the phenomena, but as an *explanandum*, i.e. a descriptor.

§9a – DSM-5

The DSM-5 use it as a qualifier for “activity”, “agitation” and “retardation” (stimulant withdrawal and depression). Regarding catatonia, psychomotor “features”, “disturbances” as used as equivalent for “catatonic”; and in the criteria “activity” as readout. Here are the terms defined in the glossary.

* In Germany, France and Belgium, “Psychomotricity” is the name of an independent profession that has its own cursus and diploma.

Source: “DSM 5: diagnostic and statistical manual of mental disorders, fifth edition” American Psychiatric Association, 2013. American Psychiatric Publishing, Washington DC, USA.

Psychomotor agitation: Excessive motor activity associated with a feeling of inner tension. The activity is usually nonproductive and repetitious and consists of behaviors such as pacing, fidgeting, wringing of the hands, pulling of clothes, and inability to sit still.

Psychomotor retardation: Visible generalized slowing of movements and speech.

Catatonia: defined by the presence of 3 or more of 12 psychomotor features [...] that may involve decreased motor activity, decreased engagement during interview or physical examination, or excessive and peculiar motor activity. The clinical presentation of catatonia can be puzzling, as the psychomotor disturbance may range from marked unresponsiveness to marked agitation. Motoric immobility may be severe (stupor) or moderate (posturing, catalepsy and waxy flexibility). Similarly, decreased engagement may be severe (mutism) or moderate (negativism). Excessive and peculiar motor behaviors can be complex (stereotypy, mannerism, grimacing) or simple (agitation) and may include echolalia and echopraxia.

§9b – ICD-11

Source: WHO website.

<https://icd.who.int/browse11/>

Psychomotor agitation (MB23.M): Excessive motor activity, usually manifested by purposeless behaviors such as fidgeting, shifting, fiddling, inability to sit or stand still, wringing of the hands, etc.

Psychomotor retardation (MB23.N): A visible generalized slowing of movements and speech.

Psychomotor symptoms in primary psychotic disorders (6A25.4): Psychomotor symptoms in primary psychotic disorders include psychomotor agitation or excessive motor activity, usually manifested by purposeless behaviours such as fidgeting, shifting, fiddling, inability to sit or stand still, wringing of the hands, psychomotor retardation, or a visible generalised slowing of movements and speech, and catatonic symptoms such as excitement, posturing, waxy flexibility, negativism, mutism, or stupor. The rating should be made based on the severity of psychomotor symptoms during the past week.

Catatonia (associated with another mental disorder): is a syndrome of primarily psychomotor disturbances that is characterized by the simultaneous occurrence of several symptoms such as stupor; catalepsy; waxy flexibility; mutism; negativism; posturing; mannerisms; stereotypies; psychomotor agitation; grimacing; echolalia and echopraxia. Mental disorders associated with catatonia include Mood disorders, Schizophrenia or other primary psychotic disorder, and Neurodevelopmental disorders, especially autism spectrum disorder.

Remark: the two first (agitation and retardation) are defined in chapter 21 (Symptoms, signs or clinical findings, not elsewhere classified); group MB23 (Symptoms or signs involving appearance or behavior)

§10 – Current psychiatric literature

§10a – A-theoretical account: “psychomotor” to qualify the *explanandum*

As in the ICD and the DSM, “psychomotor” is used as an *explanandum*, i.e. the phenomena, rather than an *explanans*, i.e. their causes. Hence, “psychomotor symptoms” refer to every motor manifestation occurring in psychiatric patients. This very broad definition all-embrace any motor manifestations occurring within the course of a psychiatric disorder with the surprising exception of conversion phenomena (see concept map §13a).

Source: “Editorial: Psychomotor symptomatology in psychiatric illnesses”, Morrens H and Walther S in “Psychomotor symptomatology in psychiatric illnesses” (e-book) 2015. Front. Psychiatry.

<https://doi.org/10.3389/fpsy.2015.00081>

“Psychomotor symptoms are those symptoms that are characterized by deficits in the initiation, execution and monitoring of movements, such as psychomotor slowing, catatonia, neurological soft signs, reduction in motor activity or extrapyramidal symptoms”.

§10b – “Psychomotor” as an *explanandum* with psycho-physiological *explanans*

An elaborated, psychological-physiological cascade model of psychomotor abnormalities in psychiatric disorders has been proposed by Northoff and co-workers (see concept map §13e).

Source: “All roads lead to the motor cortex: psychomotor mechanisms and their biochemical modulation in psychiatric disorders”, Northoff G, Hirjak D, Wolf RC, Magioncalda P, and Martino M. Mol Psychiatry, 2021;26(1):92-102.

Definition: psychomotor = all mentally driven motor outputs

Psychomotricity remains conceptualized as a descriptor of part of the motor output that is accounted for by psychic activity.

Psychologically, psychomotor refers to bodily movements that result from mental activity and environment.

Psychological level: motor function modulated by cognitive and emotional functions

Psychomotricity does not refer to a specific function but to a generic property of many (if not all) psychological functions, i.e. their ability to modulate the “primary motor function”.

Psychomotor mechanism describes how primary motor function is modulated by non-motor function, i.e., cognition and emotion.

Physiological mechanisms: cortical and subcortical networks and different neurotransmitter systems

Yet, the core of the proposal is a (patho)physiological model explaining how mental processes could influence the motor function (see §13e).

Rather than recruiting specific and exclusive regions or pathways, these psychomotor mechanisms can be characterized as relationships or balances like the balance of

- (i) dopamine- and substantia nigra-based subcortical–cortical motor circuit by primarily non-motor subcortical raphe nucleus and serotonin via basal ganglia and thalamus (as well as by glutamate and GABA).*
- (ii) Cortical motor networks to other networks like default-mode and sensory networks.*
- (iii) Global cortical activity to motor cortex.*

Source: “Why is there symptom coupling of psychological and motor changes in psychomotor mechanisms? Insights from the brain’s topography”, Northoff G, Hirjak D, Wolf RC, Magioncalda P, and Martino M. Mol Psychiatry. <https://doi.org/10.1038/s41380-020-00945-7>.

Rather than recruiting specific and exclusive regions or pathways, these psychomotor mechanisms can be characterized as relationships or balances like the balance of

- (iv) *dopamine- and substantia nigra-based subcortical–cortical motor circuit by primarily non-motor subcortical raphe nucleus and serotonin via basal ganglia and thalamus (as well as by glutamate and GABA).*
- (v) *Cortical motor networks to other networks like default-mode and sensory networks.*
- (vi) *Global cortical activity to motor cortex.*

§10c – Psychomotor as a psychological *explanans*

One recent proposal aimed at providing Kraepelin’s understanding of psychomotricity from the reading of his textbook: it is supposed to be the physiological equivalent to conscious volition, but it is also changed by the affect \pm the sensorium. Here “psychomotor” explains clinical phenomena by supposing a functional defect. As opposed to the neuropsychiatric view, the *explanans* is the impairment of a specific “psychomotor function” rather than dedicated “psychomotor systems”.

Psychomotor = volition modulated by emotions (\pm sensorium)

Source: “Structure and neural mechanisms of catatonia”. Walther S, Stegmayer K, Wilson JE, and Heckers S. The Lancet Psychiatry. 2019;6(7):610-9.

Psychomotor is generally taken to refer to the volitional aspects and affective modulation of spontaneous or cued motor behavior. The term includes the will to act, and the planning and execution of a motor act, but also includes the modulation of motor behavior by sensorium and affect.

Psychomotor retardation = cognitive and motor execution slowing

Source: “Psychomotor slowing in Schizophrenia: Implications for endophenotype and biomarker development”. Osborne KJ, Walther S, Shankman SA, Mittal VA. Biomarkers in Neuropsychiatry. 2020; 2:100016.

[In psychomotor slowing there are] deficits across the cognitive (prefix “psycho”) and motor execution (root word “motor”) aspects of slowing, with cognitive processes such as planning and response selection being particularly affected.

§11 – Research domain criteria (RDoC) for sensorimotor constructs

Source: web site (accessed the 09/06/2021).

<https://www.nimh.nih.gov/research/research-funded-by-nimh/rdoc/constructs/>

The RDoC is a research framework for new approaches to investigating mental disorders, integrating many levels of information (from genomics and circuits to behavior and self-reports) to explore basic dimensions of functioning that span the full range of human behavior from normal to abnormal.

The sensorimotor domain was not part of the original proposal. Up to October 2021, it is the only domain that has been added to the RDoC matrix (Simmons, 2018), at the insistence of academics (Bernard and Mittal, 2015; Mittal et al., 2017). The contours of sensorimotor constructs and their

putative biological substrates were defined by consensus. Sensorimotor constructs are “involved in the control and execution of motor behaviors, and their refinement during learning and development”.

The sensorimotor domain is made of 4 constructs (see table 1) of which only 3 subconstructs of the “motor actions” construct are relevant for catatonic phenomena: “initiation”, “execution” and “inhibition and termination” (highlighted in bold blue in §11a, see §13d).

RDoC sensorimotor domain	
Constructs	Subconstructs
Motor actions	Planning and selection
	Sensorimotor dynamics
	Initiation
	Execution
	Inhibition and termination
Agency and ownership	
Habit	
Innate motor patterns	

Table 1: RDoC sensorimotor domain

§11a – Motor actions construct

Motor actions processes must be engaged during the planning and execution of a motor action in a context-appropriate manner. These processes will often be recruited in conjunction with motivational processes described in other domains, as when appetitive motivations drive approach behaviors.

- **Action planning and selection:** processes whereby an individual engages a plan for spatial and temporal components of possible purposeful movements, which match internal and external constraints to achieve a goal. This may also include cost-benefit calculations in the development and selection of motor plans.
 - Behavior: apraxia.
 - Regions: inferior and posterior parietal cortex, premotor cortex, supplementary motor area (SMA), superior temporal sulcus.
- **Sensorimotor dynamics:** specification and parameterization of an action plan and program based on integration of internal or external information, such as sensations and urges and modeling of body dynamics. This process is continuously and iteratively refined via sensory information and reward-reinforced information.
 - Behavior: developmental coordination disorder, hyposensitivity, weakness.
 - Regions: basal ganglia, cerebello-olivary-pontine complex, cerebellum, parietal cortex, somato-sensory cortex, substantia nigra, thalamus.
- **Initiation:** initiation of a selected action plan; this may include timing of movement onset.
 - Behavior: apathy, **catatonic stupor**, negative symptoms, **psychomotor retardation**, stuttering.
 - Regions: basal ganglia, dorsal cingulate, SMA.
- **Execution:** actualization and adaptation of the action implementation.
 - Regions: efferent and afferent spinal and peripheral pathways, motor cortex.
 - Physiology: *Bereitschaftspotential*, corticospinal tract excitability, Hoffman reflex, movement-related potentials, use-dependent plasticity.
 - Behavior: activity level, Ehlers-Danlos syndrome, **psychomotor retardation**.
 - Paradigms: motor evoked potential latency.
- **Inhibition and termination:** inhibition of motor plans, either before or after an action is initiated, and the sense that a motor plan has been successfully completed. The inhibition sub-construct is commonly operationalized as motor response inhibition and has conceptual overlaps with the “inhibition/suppression” subconstruct of the “cognitive control” construct (cognitive systems domain).
 - Molecules: dopamine, GABA, noradrenaline.
 - Cells: intracortical inhibitory interneurons, striatal interneurons.
 - Circuits: Basal ganglia, cerebellum, dorsolateral prefrontal cortex, frontal eye-fields, inferior frontal gyrus, inferior parietal cortex, lateral premotor cortex, supplementary motor area (SMA), pre-SMA, medial prefrontal cortex, mid-cingulate gyrus, posterior cingulate gyrus, superior parietal cortex.

- Physiology: measurements of cortical inhibition, oscillatory rhythms, prepulse inhibition.
- Behavior: activity level, **automatic obedience**, **catatonic immobility**, **catatonic rituals**, **negativism**, **perseveration**, **stereotypic behaviors**, tics, **utilization behavior**.
- Paradigms: stop-signal reaction time.

§11c – Agency and ownership construct

Agency is the sense that one is initiating, executing, and in control of one's volitional actions and their sensory consequences and the sense that one's body or body parts belong to oneself. This may include the comparison of the predicted and actual sensory consequences of one's action, awareness of the intention to move, temporal binding of self-generated action and their immediate effects, and attenuation of sensory consequences of self-generated actions.

- Cells: mirror neurons.
- Circuits: cerebellum, corpus callosum, inferior parietal cortex, SMA, pre-SMA, sensorimotor-thalamus, somatosensory cortex.
- Physiology: efference copy, readiness potential.
- Behavior: alien hand syndrome, functional movement disorders, neglect, perceptions of external control, stereotypic behaviors, tics.

§11c – Habit construct

Habits are learned stimulus-response mappings triggered by internal or external stimuli that are autonomous of the current value of the outcome or goal. Habits may include overlearned sequences. Habits are implicit and efficient, requiring few cognitive resources, but can also be maladaptive under novel circumstances. Habits are based on previous positively or negatively reinforced learning and commonly occur after extended learning. Both habit formation and expression are commonly operationalized within motor control systems. When habit formation is motivated by reward learning it overlaps with the habit-construct within the "positive valence domain".

- Molecules: dopamine, GABA, glutamate, serotonin.
- Circuits: parietal association cortex, sensorimotor-basal ganglia.
- Behavior: compulsive behaviors, stereotypic behaviors.
- Self-Report or rush video based: tic rating scale, Yale global tic severity scale
- Paradigms: 2-step task.

§11d – Innate motor patterns construct

Innate motor patterns are unlearned action plans that may be triggered by internal or external stimuli. This can include such behaviors as stereotyped expressions of affect, orientation to salience, innate approach and withdrawal phenomena, and startle responses.

- Circuits: brainstem, hypothalamus, motor cortex, oculomotor system.
- Physiology: eye-blink reflex.
- Behavior: disinhibition of early motor reflexes, incontinent affect, startle, stereotypic behaviors.

§12 – Authors' preferences

After contributing to the clarification of the different concepts, each author was asked to rank their preferences for the main approaches detailed in the paper:

- Psychomotor systems:
 - 1 - Sensualist psychomotor concept, e.g., Griesinger's account.
 - 2 - (Illenau's and) WKL psychomotor systems.
- Sensorimotor:

3 - Sensorimotor systems, like Kahlbaum and SyNoPsis.

5 - Sensorimotor constructs as in the RDoC.

- 5 - Other (to be specified) / None.

All authors expressed their opinions (n=32).

§12a - Psychomotor vs sensorimotor

Though there is a preference for the psychomotor framework at the first vote, psychomotor and sensorimotor approaches are considered equally as soon as the second vote (Figure 7).

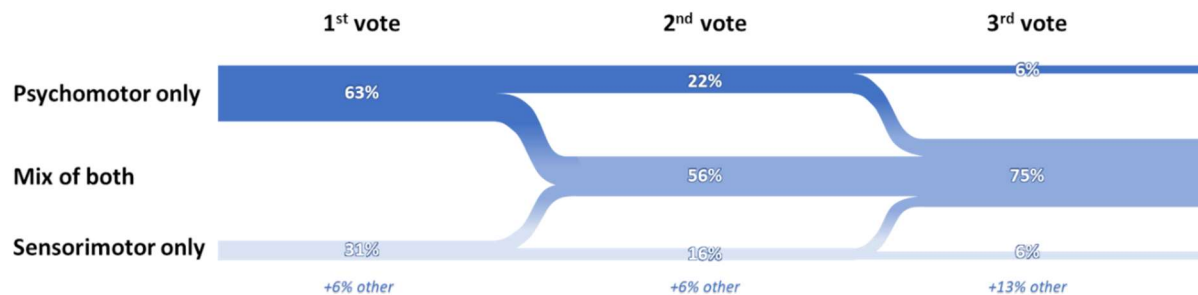


Figure 7: Evolution of the proportion of vote for psychomotor and sensorimotor approaches. Psychomotor only: proportion of the votes that exclude sensorimotor proposal. Note that as there were two of each, the 6% who remain in this category at the 3rd choice vote for the other/none item.

§12b - Systems vs constructs

The systemic approach was favored by most authors, though part of this preference might have been driven by the fact that there was only one proposal for the constructivist approach (Figure 8).

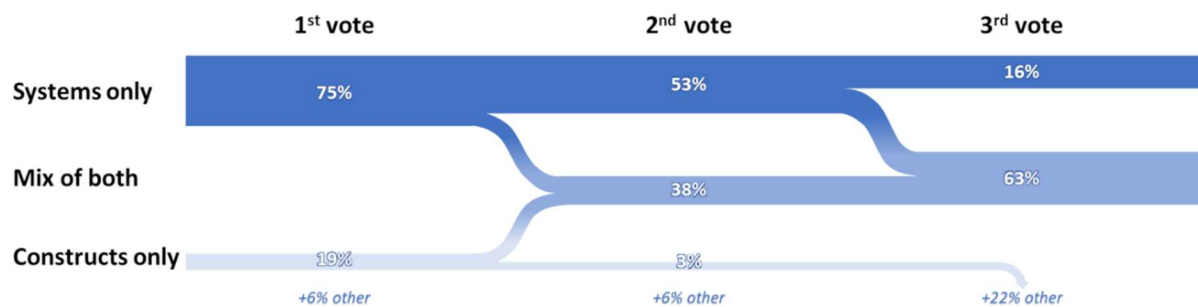


Figure 8: Evolution of the proportion of vote for systems vs constructs approaches.

§12c - Authors remarks

John Waddington

The “Wernicke-Kleist Leonhard psychomotor systems” would be choice 1 if systems referred to here can extend to concepts of cortical-striatal-thalamocortical neuronal network dysfunction.

Manuel Cuesta

Psychomotor disturbances (or Motor abnormalities) appear to represent a true transdiagnostic domain putatively sharing neurobiological mechanisms of neurodevelopmental, functional or neurodegenerative origin (Cuesta and Peralta, 2018).

§13 – Psychomotricity: different concept and their relationship with catatonia

The relationship between “psychomotor” and “catatonia” concepts varies according to the framework in which they are embedded (Figure 9 for a field map). As the a-theoretical framework aims to remain

purely descriptive both refer to purely clinical phenomena (§13a). Conversely, other approaches are making pathophysiological or psychopathological models.

Proponents of the neuropsychiatric tradition, e.g. Griesinger (§13b), WKL (§13c), elaborate pathophysiological models, according to which clinical phenomena are supposedly accounted for by the disfunction of systems. PM phenomena, i.e. the *explanandum*, are explained at the level of PM systems, i.e. the *explanans*. Hence, they mostly proceed from gathering clinical observations from which they infer the existence of specific systems, i.e. bottom-up.

Most current approaches remain in the psychological tradition according to which clinical phenomena are supposedly accounted for by the impairment of a mental function. Mental or cognitive functions are generally elaborated in a top-down manner, i.e. they are constructs. Here PM phenomena, i.e. the *explanandum*, are explained by the impairment of a PM function (or PM construct). In psychopathological models, the *explanans* is at the level of the function, not at the level of the system. This does not mean that the dysfunction cannot be accounted for by a brain system, but it implicitly assumes a one-to-one correspondence between the function and a specific system. The RDoC sensorimotor proposal will illustrate this view (§13d).

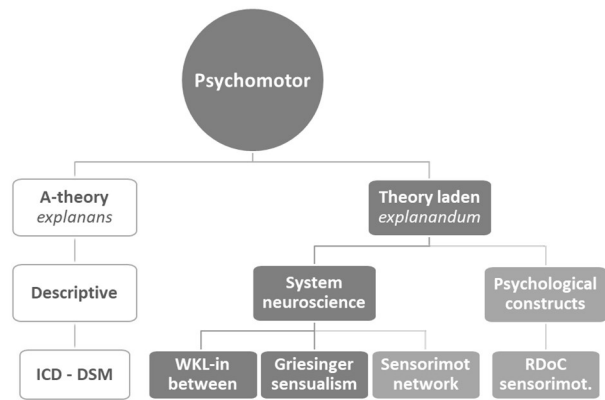


Figure 9: Field map of the different understanding of “psychomotor”.

Last a recent proposal stands out from the other proposals as it neither assume the existence of specific PM systems, nor the existence of a dedicated PM function. PM phenomena are supposed to be accounted for by a generic property of all mental activities to influence motor outputs (§13e).

§13a – A-theoretical understanding

The a-theoretical use of “psychomotricity” allows any understanding up to the extensive version (§10a) illustrated in Figure 10. According to this extensive version, the psychomotor domain (light blue) encompasses all motor manifestations occurring in psychiatric patients, including catatonic phenomena (dark blue). The sole exception however are conversion phenomena.

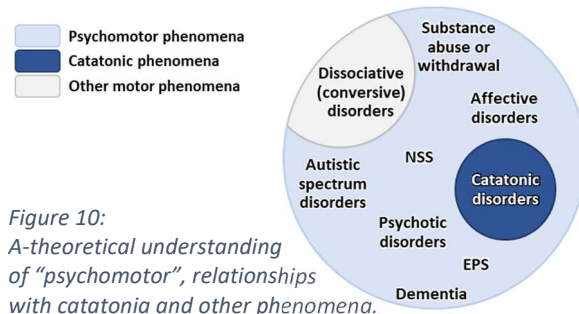


Figure 10: A-theoretical understanding of “psychomotor”, relationships with catatonia and other phenomena. EPS: extrapyramidal side effects; NSS: neurological soft signs

§13b – Griesinger’s sensualist model

Griesinger’s physiological model of the mind illustrated in the figure on the right closely matches sensualist views. The “psychic-motor” part of his loop accounted both for the will-contents and the will-processes in driving motor systems (Figure 11). The term “psychic-motor” was later replaced by “psychomotor” (PM) by Richard von Krafft-Ebing (§3a, 1b).

According to this neuropsychiatric view, “psychomotricity” belongs to the psychism (light blue cloud). PM systems are

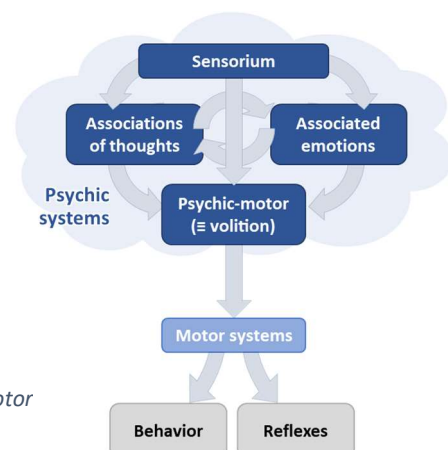
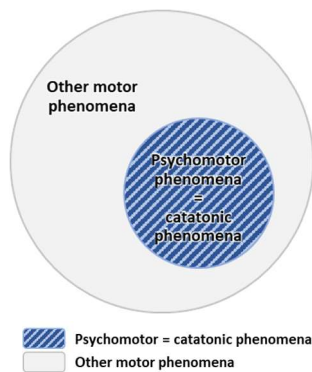


Figure 11: Concept map of Griesinger / sensualist model in which psychic-motor systems are within the psychological domain.

compulsory crossing point for psychic contents to influence motor outputs (behavior and reflexes – grey boxes) as they are the only ones to project directly to motor systems (out of the psychism).



As illustrated in Figure 12, the sensualist understanding supposes that “PM phenomena” and “catatonic phenomena” refer to the same thing. Catatonic phenomena are supposed to result from the impairment of PM systems.

The same concept map might apply to one of the current understandings of “psychomotricity” defined as “volition modulated by emotions ± the sensorium” (§10b). However, since the latter is an interpretation of Kraepelin’s views, it might not fit with the neuropsychiatric account in terms of systems’ failure.

Figure 12: Griesinger/sensualist understanding of “psychomotor” and its relationships with catatonia and other phenomena.

§13c – WKL model

Following Heinrich Schüle (§3b), PM systems are no more belonging to the psychism per se but stand in-between the intra-psychic and motor systems. As illustrated in Figure 13, PM systems are no more in the cloud as in the sensualist model (§13b).

Following Schüle, Carl Wernicke divided motor phenomena according to into three groups according to their supposed level of impairment:

- i) Neurological systems which are responsible for pyramidal, extrapyramidal or cerebellar symptoms for instance.
- ii) PM systems which are responsible for quantitative or qualitative changes looking like intentional, emotional or reactional actions but unrelated to any intrapsychic drive or content.
- iii) Abnormal intrapsychic process or content that secondarily result in abnormal motor or behavioral output through normally functioning PM and motor systems.

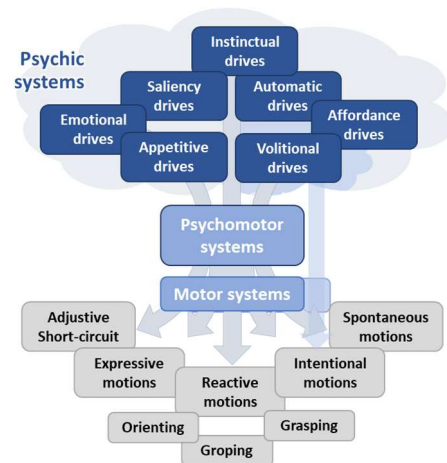


Figure 13: Concept map of WKL model.

PM systems are processing various inner drives, not only coming from volition, but also from many non-conscious processes. Though all these drives are intra-psychic, their content can be non-explicit (light grey part of the cloud) as opposed to the volitional drive which content is consciously experienced (small light blue part of the cloud).

Although PM systems contribute to make the behavior more coherent, many intra-psychic drives still have more or less selective effects on specific motor outputs (grey boxes). For instance, emotional drives mainly result in expressive movements, e.g. facial mimics, and body postures. This led Wernicke and Kleist to propose a typology of motor outputs, e.g. expressive, reactive, short-circuit (grey boxes), which makes it often possible to distinguish between primary vs secondary PM phenomena (§6b, 6c). PM systems still are supposed to be the compulsory crossing point for all automatic drives to influence motor systems (grey arrows), except for volitional drives (blue arrow). On specific occasions these can (effortfully) bypass PM systems. Primary PM phenomena prevail on expressive and reactive motor outputs while intentional actions can be relatively spared (§6b, 6c). In system catatonia phenotypes, the impairment can even be attributed to more specific PM system(s) (§6f).

As already stated, a catatonic stupor is supposed to result from a different pathophysiology than a melancholic stupor which results in different clinical expression, e.g. no or distorted facial expression in the former and painful or anxious mimic in the latter (§6c).

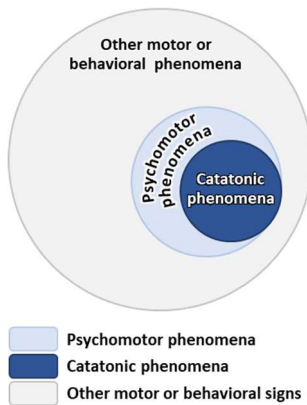


Figure 14: WKL understanding of “psychomotor” and its relationships with catatonia and other phenomena.

As schematized in Figure 14, according to the WKL neuropsychiatric perspective “catatonic phenomena” (dark blue) are no more equivalent to “PM phenomena” (light blue). The formers are only a subfraction of the latter. “PM phenomena” refer to all kinds of motor and behavioral outputs that are primarily accounted for by changes in the functioning of PM systems, either quantitative or qualitative. Conversely, a narrow understanding of “catatonic phenomena” would limit them to signs that can only be accounted for by qualitative changes in PM functioning, e.g. parakinesias, proskinesia, PM-negativism.

Indeed, while primary quantitative PM-changes, i.e. hyperkinesia or akinesia, can be observed in motility psychosis (a non-catatonic phenotype), qualitative PM-changes are quasi-pathognomonic for either form of catatonia. As stated in the main text, WKL-catatonia only refer to those phenotypes coming with a progressive buildup of PM residuum (§6e,f,g) (Pfulmann and Stöber, 2001).

From a pathophysiological perspective, only qualitative changes are indicative of the dysfunction of PM systems, whereas quantitative changes could also be accounted for by the impairment of regulatory systems rather than PM systems *per se*. Since qualitative PM-signs quite exclusively occur in the context of a catatonic phenotype, they are supposed to result from different kinds of self-limited neurodegenerative processes.

From a clinical perspective, qualitative changes or “catatonic phenomena” are of prognostic value.

§13d – RDoC: motor actions construct of the sensorimotor domain

The Figure 15 sketches the research domain criteria (RDoC) account of typical catatonic signs (dark grey boxes) and related phenomena (light grey, see §11). All are supposed to result from the impairment of the motor actions construct (light blue cloud) within the sensorimotor domain. This construct was split in 5 subconstructs (blue boxes) of which the inhibition and termination subconstruct is supposed to account for all but one of typical catatonic phenomena: catatonic stupor is supposed to be accounted for by the impairment of the execution subconstruct. Though it is not clearly stated, this implicitly supposes that catatonic stupor result from defective implementation of action while catatonic immobility would be accounted for by excessive inhibition. However, the RDoCs do not specify how to distinguish stupor and immobility clinically.

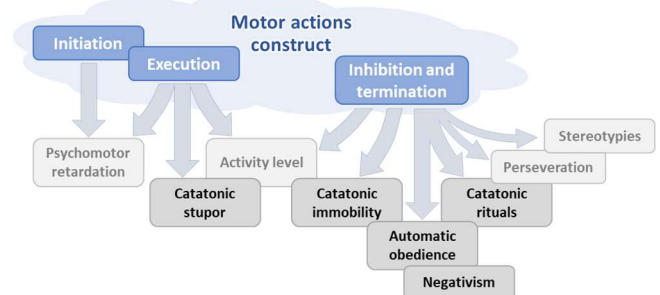


Figure 15: Concept map of the RDoC motor action construct (sensorimotor domain).

As far as we are aware of, the RDoC do not define “psychomotor” and rather use the terms is the same a-theoretical way than the international manual and is supposed to share their conceptual map (§13a). There is no statement on how cognitive and emotional constructs interact with the sensorimotor domain and find their way to motor outputs.

Remark: Systems vs constructs. By calling its domain sensorimotor “systems”, the RDoC let believe that construct and system’s approaches are the same. However, constructs are defined at the functional (mental/psychological) level whereas systems are described at the structural (somatic/neurological) level. Moreover, constructs are linear and sequential account of psychological functions, as illustrated by the decomposition of motor action in independent planning, selection, initiation, execution, inhibition, and

termination subconstructs (§11a, §13d). Conversely, systems are non-linear and adaptive by nature so that they rarely have a one-to-one relationship with linear psychological functions (Foucher et al., 2021b).

§13e – Functional integration model: psychomotricity as a property of all psychic systems

In this model, PM phenomena manifest themselves as motor outputs that can be accounted for by psychic/affective activity or content. The model does neither assume the existence of dedicated PM-systems nor the one of a specific PM-function. Here psychomotricity is a generic property that all mental/affective processes modulate motor outputs. This PM-property is intrinsic and indissociable from all psychic/affective processes.

Accordingly, PM phenomena, the *explanandum*, are not accounted for by specific PM-systems or PM-function which could be impaired on their own but by abnormal cognitive-emotional state, content, or process. Abnormal psychic functioning is supposed to result in the abnormal modulation of the motor function due to their PM-property (dark blue arrow in Figure 16).

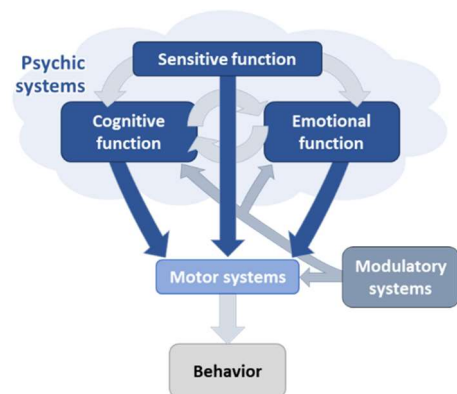
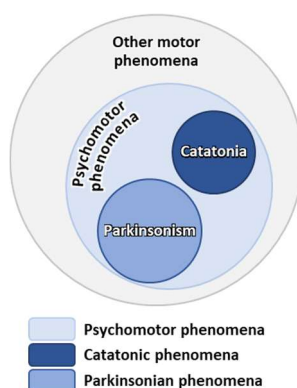


Figure 16: Concept map of the functional integration model.

The operationalization of the functional integration model relies heavily on resting state networks (Seitzman et al., 2019), equating the motor function with the sensorimotor network and psychic activity with the other resting networks, as the salience and the default mode networks (Northoff et al., 2021).

- i) Through the neuromodulation of the sensorimotor network as defined by resting state connectivity (\approx sensorimotor system). For instance and decrease of serotonin would reduce the GABA_A tone in the motor cortex (5HT_{2A} Rc). A second instance is the dopaminergic modulation of motor striatal loop.
- ii) The same modulatory systems could also change the activity of non-motor resting state networks subserving cognitive or emotional functions. These will interact with the sensorimotor network, either in one-to-one relation, e.g. with the default-mode network, or globally with the whole cortical activity. This interaction is modeled by functional connectivity.
- iii) Cerebellar loops were added to the model that could further modulate both motor and non-motor cortical networks (light grey) (Mittal et al., 2020; Northoff et al., 2020).

All three mechanisms operate in a dimensional and cross-nosological way (Northoff et al., 2021). Depending on their balance, they can lead to either psychomotor slowing, e.g. depression, akinetic catatonia, or psychomotor agitation, e.g. mania, hyperkinetic catatonia (Martino et al., 2020, 2016). This model proposes a neurobiological account for clinical overlaps and similarities between various sensorimotor and psychomotor phenomena, e.g. spontaneous and drug induced parkinsonism, parakinesias, and tardive dyskinesia.



According to the functional integration model, both parkinsonism (blue, Figure 17) and catatonia (dark blue) are PM phenomena or syndromes (light blue). Hence, as in WKL model, PM phenomena are not the same as catatonic phenomena. Yet, the frontier is set very differently. On the one hand parkinsonism is part of PM phenomena which is not supposed to be the case in neuropsychiatric models (§13b,c). On the other hand, there is no difference between a psychotic stupor and an affective one for instance. Catatonia is conceived trans-nosologically, i.e. it is supposed to have the same pathophysiology whatever it happens in the context of schizophrenia, bipolar or unipolar mood disorders as opposed to WKL for instance (§13c).

Figure 17: Relationships of psychomotor with catatonic phenomena according to the functional integration model.

Remark: A recent systematic review nicely illustrates the impact of prerequisites on the results of brain functional correlates (Hirjak et al., 2020). If catatonic patients are only assessed on their motor/behavioral phenomena, as it is the case with the Bush and Francis catatonia rating scale, scores mostly correlate with cortical and subcortical sensorimotor systems. Other studies started from the prerequisite that affective symptoms, intrinsic to catatonia could account for psychomotor manifestations. When affective symptoms were assessed together with motor and behavioral manifestations, i.e. with Northoff catatonia rating scale, scores correlated with a much wider cortical network encompassing medial and lateral orbitofrontal, prefrontal, and parietal cortices (§10b, §13e)(Northoff et al., 2021).

§14 – A digest of the historical analysis

The historical analysis, up to the a-theoretical era, has been presented in a symposium at the 8th European Conference on Schizophrenia Research the 24th of September 2021. The symposium was proposed by Prof. Fabrice Berna and was entitled “Toward a better understanding of catatonia: An historical, epistemological and system neuroscience approach”.

The first author presented it on the behalf of the ECSP (European Collaboration on movement and Sensorimotor & Psychomotor functioning in psychoses):

“A historical analysis of the catatonia and psychomotricity concepts” (17 min, HTML5 multiplatform format, click on the blue eye to see the presentation*)



* Link: http://www.cercle-d-excellence-psy.org/fileadmin/Cours/WKL/ECSR_2021/JF/index.html

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