A historical perspective of apraxia, from 1860 to 1935

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ABSTRACT

Introduction. Research into gestural disorders is typically associated with Liepmann. However, in the second half of the 19th century, Finkelnburg, Meynert, and Nothnagel also made significant contributions to this field. The aim of this study is to explore research conducted into gestural disorders during the period between 1860 and 1935.

Development. In 1870, Finkelnburg proposed the term asymbolia to refer to the loss of the ability to understand and intentionally use concepts by means of acquired linguistic or gestural symbols. The following decade, in 1887, Nothnagel, taking Meynert's associationist model of brain organisation as a reference, proposed that gestural disorders originate in what he describes as mind palsy. The term apraxia was used for the first time in Steinthal's book *Abriss der Sprachwissenschaft*, but Liepmann, Pick, and Kleist were the main authors to synthesise the clinical, anatomical, and psychopathological aspects of apraxia. In the 1920s, Morlaas and Grünbaum resumed the 19th-century line of reasoning associating gestural disorders with perceptual disorders.

Conclusions. Between 1860s and 1930s, the study of gestural disorders underwent extraordinary conceptual development, marked by the close association between perception and gestuality. The contributions of Liepmann and the school of Wernicke sought to organise a chaos of terminology and taxonomy.

KEYWORDS

Agnosia, apraxia, asymbolia, gestuality, mind palsy, perception

Introduction

The term "apraxia" (from Greek ἀπραξία [ἀ- "no," "without" and πρᾶξις "action"]) refers to the group of gestural disorders secondary to brain injury. It refers to the inability or difficulty of performing propositional (intentional) acts in individuals with no primary sensory or motor alterations and full knowledge of the action to be performed.[1,2]

The history of apraxia is usually considered to begin with the contributions of Liepmann at the beginning of the 20th century. However, the study of gestural disorders began decades earlier, at a time when the study of the functional organisation of the brain was moving from localisationist approaches to associationist models.

At a meeting of the Société d'Anthropologie de Paris, held on 18 April 1861, Paul Pierre Broca (1824-1880)

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presented the first empirical evidence of the correspondence between a cognitive process and a specific area of the cerebral cortex.[3,4] He questioned the equipotentiality of the cerebral cortex defended by Marie-Jean-Pierre Flourens (1794-1867). Nine years after Broca's communication, Eduard Hitzig (1838-1907) and Gustav Theodor Fritsch (1838-1927) provided new evidence supporting localisationist theories. In their article entitled "Electric excitability of the cerebrum,"[5] they provide the first experimental evidence of the link between the cerebral cortex and movement. This consolidated the interest in the localisation of functions in the cerebral cortex, marking the beginning of a historical period that approximately encompasses the years 1870-1890, known as the "Golden Era" of cerebral localisation.[6]

In parallel to the localisationist postulates, associationist proposals also emerged, based on the idea that mental processes are the result of collaborative (or associative) work involving different brain regions. This conception of mental architecture was mainly represented by Carl Wernicke (1848-1905), who was the master of a whole generation of clinicians and researchers, including Heinrich Lissauer (1861-1891), Karl Heilbronner (1869-1914), Hugo Liepmann (1863-1925), Otfrid Foerster (1873-1941), Karl Bonhoeffer (1868-1948), and Karl Kleist (1879-1960).

The aim of this study is to explore research conducted into gestural disorders during the period between 1860 and 1935. We did not aim to perform an exhaustive review including all the publications on the subject, but rather to highlight the historical milestones marking the conceptual evolution of these disorders.

Development

Non-verbal deficits of aphasia

The term aphasia refers to alterations affecting linguistic processes secondary to brain injury. In the 1860s, Broca linked the loss of speech (*l'usage de la parole*) to the presence of lesions to the left third frontal gyrus.[3] Later, in 1874, Wernicke[7] published his book "The aphasia symptom-complex: a psychological study on an anatomical basis." In this book, starting from the associationist model of brain organisation proposed by Theodor Hermann Meynert (1833-1892), Wernicke establishes the foundations of modern aphasiology and consolidates the basis of brain localisation.

Although the predominant movement in aphasiology held that aphasia is a disorder of language (with other cognitive processes being relatively preserved), such classic authors as John Hughlings Jackson[8] (1835-1911), Adolf Kussmaul[9] (1822-1902), and Pierre Marie (1853-1940) openly criticised this linguistic-localisationist approach. They considered aphasia as an alteration of thought and conceptual reasoning, which is not inevitably associated with a linguistic code. In 1866, Hughlings Jackson[10] reported that some patients with aphasia presented difficulty performing voluntary or deliberate acts. When they were asked to stick out their tongue, they understood the command, tried to open their mouth, and put their fingers to their mouth to pull out the tongue, but were unable to perform the action voluntarily. In contrast, they performed the act of licking their lips or licking postage stamps automatically. Such observations led him to suggest that this is due to a disorder in the formulation, expression, and understanding of symbols, whether verbal or non-verbal in nature. Years before, in 1861, Broca reported that the patient known as Monsieur Tan-Tan was "not able to express his ideas or his desires other than by the movement of his left hand, he often made incomprehensible gestures."[11(p236)]

Asymbolia: aphasia, agnosia, or apraxia?

In 1870, Ferdinand Carl Finkelnburg (1832-1896; Figure 1A) suggested that the language impairment observed in patients with aphasia is only one of several manifestations derived from the disruption of symbolic processes.[12] According to this author, asymbolia is characterised by the total or partial loss of the ability to understand and intentionally use concepts by means of acquired symbols (linguistic or gestural). To support his ideas, he described several patients with aphasia who showed non-verbal receptive and expressive symbolic alterations: *1)* a Catholic woman who, when making the sign of the cross, put her hand behind the ear or on the neck; *2)* a violin player who was unable to write or read musical notation; *3)* a salesman who could not count money because he was unable to identify the value of each coin; and *4)* a civil servant who was unable to understand social norms or the liturgical symbols used during religious acts.

Wernicke used the term asymbolia in his book *The aphasia symptom-complex*, but with a different meaning than that suggested by Finkelnburg. For Wernicke, asymbolia refers to the extinction of the optical memory of an object, or of any of the images of the memory of

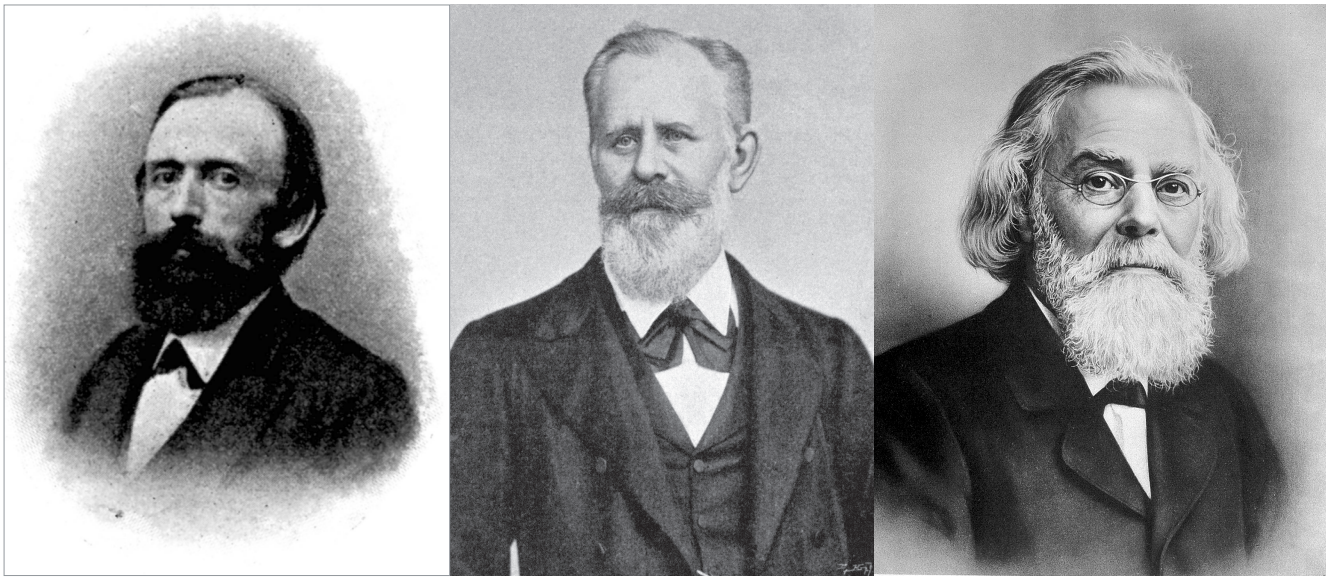

Figure 1. From left to right: Carl Ferdinand Finkelnburg (1832-1896), Hermann Nothnagel (1841-1905), and Heymann Steinthal (1823-1899).

an object that are essential for the concept. Therefore, according to Wernicke, this term refers only to distorted object recall, whereas aphasia involves an alteration to the identification of linguistic signs.

In 1890, Wernicke's mentor Theodor Hermann Meynert[13] (1833-1892) drew a distinction between sensory asymbolia (*sensorische Asymbolie*) and motor asymbolia (*motorische Asymbolie*). The first type refers to an alteration to object recognition; the second to the inability to use objects (in the absence of paralysis). Meynert's sensory asymbolia corresponds to Wernicke's asymbolia, whereas motor asymbolia bears similarities to the current concept of aphasia.

Sigmund Freud (1856-1939) attempted to bring clarity to the terminological confusion surrounding the concept of asymbolia. In his monograph *Zur Auffassung der Aphasien, eine kritische Studie* (On aphasia: a critical study; 1891), he proposed the term agnosia to refer to alterations to object recognition. His proposal was gradually adopted by clinicians, whereas the term (sensory) asymbolia was progressively left aside and forgotten.

Mind palsy and motor memory images

In 1876, Hermann Munk (1839-1912) showed that experimental lesions to the occipital lobes of dog brains led to changes in visual behaviour: the dogs could see but did not understand the meaning of what they saw. According to Munk,[14,15] the lesions caused impairment of the visual memories (*Erinnerungsbilder*) of previously perceived stimuli. Consequently, they were unable to relate current experience with past experience and, therefore, unable to understand the meaning of the stimuli perceived. He calls this alteration *Seelenblindheit* (mind blindness). As a result of the experimental lesions he made to the temporal lobe, Munk proposes the term *Seelentaubheit* (mind deafness) to describe a condition in which the animal's hearing is intact, but it is unable to

interpret sounds (due to the loss of memories of sounds). Similarly, in 1887, Hermann Nothnagel[16] (1841-1905; Figure 1B) suggests the term *Seelenlähmung* (mind palsy) to refer to the loss of motor memories.

The associationist model of brain organisation postulated by Meynert assumes that excitation in the cortical targets of nerve fibres does not fully disappear when peripheral stimulation stops. The remaining past sensations are stored as "memory images" in the areas surrounding the cortical targets. According to Meynert, mind blindness (*Seelenblindheit*) occurs when visual memory images are altered, and mind deafness is caused by impairment of auditory memory images. Similarly, Nothnagel proposes that movements create memory images of the executed movement: destruction of these memories leads to mind palsy (*Seelenlähmung*).

Nothnagel argues that movement memory images cannot be stored in the "motor centres" that transmit motor commands from the cortex to the peripheral nervous system, as while its destruction causes paralysis of the contralesional limb, the will to perform movements remains intact:

> The destruction of the motor cortical field causes permanent simple paralysis in the human being. The cortical field of motor memory images is found adjacent to this field, but spatially separated, in the parietal gyri. Its destruction causes mind palsy, in contrast with simple paralysis.[16(p24-5)]

The history of apraxia

The first appearance in print of the term apraxia is in the book *Abriss der Sprachwissenschaft* (A summary of linguistics; 1871) by Heymann Steinthal[17] (1823-1899; Figure 1C). This German linguist described a patient with aphasia who was unable to play his violin. Furthermore, when writing, he held the pen upside down. He also held spoons and forks as if he had never used them before. The patient could move his limbs freely but presented problems performing intentional acts associated with the use of objects. Steinthal, like Finkelnburg, considered aphasia to involve a general inability to understand signs, whether of verbal or gestural nature. Whereas Finkelburg uses the term asymbolia, Steinthal proposes the term asemia.

The word apraxia (*apraxie*) appears only once in *Abriss der Sprachwissenschaft*, on page 458. It is surprising that Steinthal introduces the term almost in passing, as though it were already commonly used. Such vagueness prevents us from determining whether the disorder affects object recognition or actions. In 1873, Ludwig Gogol (1847-?) used the term apraxia to refer to impairment of the ability to use objects. For Gogol,[18] apraxia is the result of a perceptual deficit. In the 1880s, such authors as Kussmaul[19] and Starr[20] again used the term apraxia, although with some differences from the current conception.

In the 1877 book *Die Störungen der Sprache* (Language disorders), Adolf Kussmaul states that some patients with aphasia lose not only the understanding of expression symbols but also the ability to use objects ("they confuse spoons with forks, and want to eat soup with a fork"[19(p181)]). The book also mentions that:

> [The patient] urinated in the sink, bit the soap, and did similar things, which may be related to a misinterpretation of the objects. […] We may observe how the misrecognition of objects, which is the foundation of apraxia, is much more severe than the misinterpretation of expression symbols.[19(p199)]

From this extract, we may conclude that Kussmaul believed that the incorrect use of objects was due to a perceptual failure in their recognition.

In 1888, Moses Allen Starr (1854-1932) published an article on apraxia and aphasia that defines apraxia as the inability to recognise the use of objects. He suggested that its assessment may only consist in presenting several objects to the patient and observing whether he or she displays any signs of recognition. His concept of apraxia is more similar to what we understand today as agnosia. Similarly, Morlaas and Grünbaum suggested the terms "apraxia of use" (1928) and apractoagnosia (1930), respectively (refer to the section "The close connection between apraxia and agnosia," below).

Apraxia: Liepmann and Wernicke's school

In 1900, Hugo Karl Liepmann (1863-1925; Figure 2A) reported the case of a patient (an imperial counsellor) who presented a disorder affecting the execution of learnt movements, including the ability to perform motor commands, by imitation or when the object is absent. Liepmann[21] also stresses that this is not due to an essential motor deficit or impairment of the basic ability to execute or coordinate motor activity. He uses the term apraxia to refer to this gestural disorder, asserting that it is not explained by a problem in the perception of an

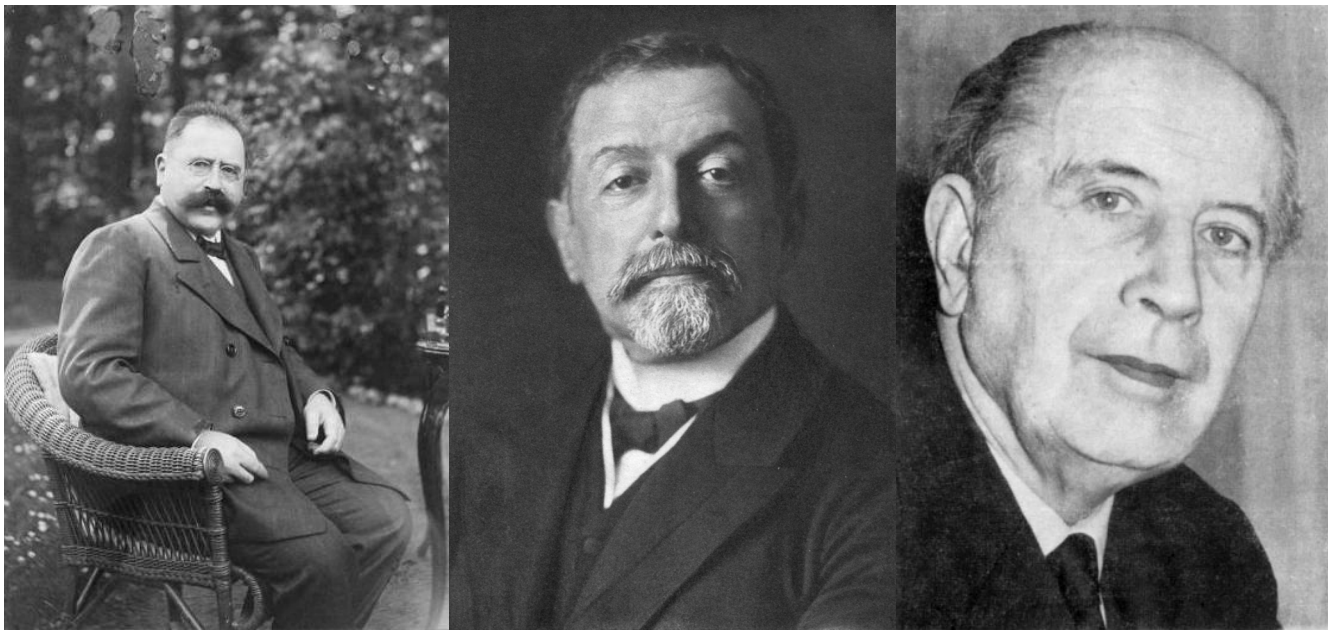

Figure 2. From left to right: Hugo Karl Liepmann (1863-1925), Arnold Pick (1851-1924), and Karl Kleist (1879-1960).

object, ignorance of its use, or to an overall alteration in symbol expression.

In 1899, one year before the publication of Liepmann's article, the Belgian physician David de Buck reached similar conclusions on the pathophysiological origin of apraxia after visiting a patient with postpartum neurological symptoms: "[…] she may conceive actions, but is unable to remember the corresponding kinetic images. There is a disconnection between movement regions and the ideation area."[22(p373)] This author locates the ideation area in the occipital and parietal gyri. De Buck uses the term parakinesia (*parakinésia*) to refer to this gestural disorder.

In *Ueber Störungen des Handelns bei Gehirnkranken* (On the alterations of action in patients with cerebral disease; 1905), Liepmann[23] suggests that there are two types of apraxia: motor (innervatory) apraxia (*motorische [innervatorische] Apraxie*) and ideational apraxia (*ideatorische Apraxie*). In motor apraxia, "movement does not correspond to the ideational process: the corticomuscular system works properly, but not to the service of the entire mental process."[23(p156-7)] Whereas in ideational apraxia, "movement corresponds to the ideational process, but this process is distorted in the section dedicated to the design of the series of movements, leading to the conversion of a main objective idea into a partial objective idea."[23(p157)] Liepmann repeatedly cites the contributions of Pick and Heilbronner[A] in *Ueber Störungen des Handelns bei Gehirnkranken*.

In 1905, several months before Liepmann's book was published, Arnold Pick[24] (1851-1924; Figure 2B) published *Studien über motorische Apraxie: und ihr nahestehende Erscheinungen: ihre Bedeutung in der Symptomatologie psychopathischer Symptomenkomplexe*

[A]Heilbronner and Liepmann both worked as assistants to Wernicke in Breslau (1894-1898 and 1895-1899, respectively).

(Studies on motor apraxia and associated phenomena: their relevance in the symptoms of psychopathic symptom complexes). This book, in which the term motor apraxia is used generically, includes four relevant sections for the subsequent definition of the concept and the different types of apraxia. The third section analyses motor apraxia in the context of focal brain lesions. It describes the use of several objects, such as a comb, a coffee grinder, a hair brush, etc. Pick gives the following description:

> He takes a match from the box, makes striking movements in the air over the candle and puts the match back into the box; when he is provided a lit match and asked to light the candle, he brings the match to the candle, but stops it close to the wick, which he blows out even though the candle is not lit, when the match is burnt […][24(p80)]

Pick suggests the name ideomotor apraxia (*ideomotorische Apraxie*) to refer to apraxia caused by a general attentional disorder, in an attempt to indicate that it is not due to an alteration in the perceptual recognition of objects or to a deficit in the motor execution of their use. Liepmann substitutes the name ideomotor apraxia with ideational apraxia, and agrees with Pick's conclusion: this variant of apraxia is generally caused by a mental alteration that manifests in the action domain, but it is rooted in deficits that are not specific to action. Despite separating ideomotor apraxia from any type of alteration in object recognition, Pick repeatedly asserts that parieto-occipital lesions may be particularly relevant for this type of apraxia, as they destroy the neural basis of the mental visual images of actions.

In the article "Zur Frage der motorischen Asymbolie (Apraxie)" (On the subject of motor asymbolia [apraxia]), published in 1905, Karl Heilbronner[25] classified apraxia into three subtypes: *1)* cortical apraxia (*kortikale Apraxie*): the sensorimotor system is impaired, with parakinetic phenomena being present in all forms of movement; *2)* transcortical apraxia (*transkortikale Apraxie*): the sensorimotor system is preserved but complex voluntary movements are impaired; patients may present parapraxic movements (movement elements are wrongly selected and incorrectly sequenced); and *3)* conduction apraxia (*Leitungsapraxie*): the expected movement is replaced by non-adaptive movements (eg, picking up the object located in front of the patient rather than the requested object) and the patient is unable to change position, maintaining the previous one (eg, the patient holds several objects in the hand after picking them up). In conduction apraxia, movement alterations are rare or non-existent. Although Heilbronner suggests that it is possible to differentiate between transcortical apraxia and conduction apraxia, he also acknowledges that "they cannot be strictly separated, not even schematically."[25(p190)]

In 1907, Karl Kleist[B] (Figure 2C) published the article "Kortikale (innervatorische) Apraxie" ("Cortical [innervatory] apraxia").[C] In this article, he describes a type of apraxia in which movements, especially movements of the hand, are scarce, slow, imprecise, and stiff, observing no sensory loss, ataxia, or changes in muscle tone. Although movements are slow or imprecise, they are executed in the correct sequence and oriented in space, and their ideation is correct. Kleist proposes the term innervatory apraxia.

Months after the publication of Kleist's article, Liepmann[26] published a summary of the clinical, anatomical, and psychopathological aspects of apraxia in the journal *Medizinische Klinik*. At the same time, in an attempt to diminish the terminological confusion surrounding gestural disorders, Liepmann proposes a distinction between three types of apraxia:

— Ideational apraxia (*ideatorische Apraxie*): caused by a disturbance of motor planning (ideation schemas). As a consequence, action elements are not correctly integrated according to their superordinate purpose.

— Ideokinetic apraxia (*ideokinetische Apraxie*): characterised by a disassociation between the ideation schema and its execution. According to Liepmann, difficulty imitating movements constitutes unequivocal proof of the separation between idea (ideation schema) and execution.

— Limb-kinetic apraxia (*gliedkinetische Apraxie*): specific loss of purely kinetic memory of the limbs (kinaesthetic and innervatory).

In the neuroanatomical field, following the associationist model of brain organisation proposed by Meynert

[B]Kleist worked as an assistant to Wernicke at the Neuropsychiatric Clinic at the University of Halle between 1903 and 1908.

[C]We could not find the original article, but the ideas it discusses are collected in Kleist's huge monograph *Gehirnpathologie* (Cerebral pathology; 1934).

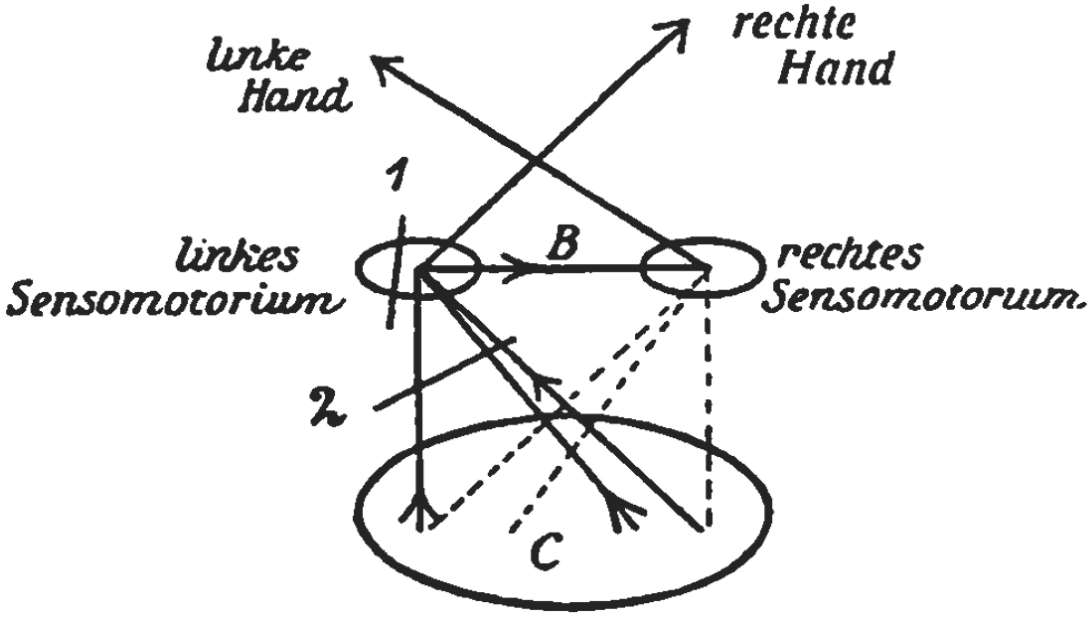

Figure 3. Liepmann's horizontal schema (1908) depicting the conversion of mental images into motor commands. The cortex (C) predominantly sends information to the left sensorimotor region (*linkes Sensomotorium*), responsible for controlling the right hand (*rechte Hand*). The left sensorimotor area controls the left hand (*linke Hand*) via the corpus callosum (B).

(and subsequently developed by Wernicke), Liepmann suggests that the idea or plan of a movement, in both its spatial and temporal forms, is stored (as a motor memory) in the left parietal lobe.[D] To perform a propositional motor action, the spatiotemporal plan may be retrieved and be associated, by means of cortical connections, with the innervatory scheme stored in the left sensorimotor region, which transmits information on formulas to the left primary motor areas. When the right limb executes the movement, the information is transmitted from the left to the right sensorimotor centre through the corpus callosum to activate the right motor cortex (Figure 3).

[D]In 1907, Friedrich Hartmann (1871-1937) suggested that apraxia may be caused by frontal lesions (Hartmann F. Beiträge zur Apraxielehre. Monatsschr Psychiatr Neurol. 1907;21:97-118, 248-70).

Liepmann's taxomony, together with his neuro-anatomical organisation, was embraced by most of his contemporaries.[27-29] However, he also had critics among those who disagreed with the associationist model, including Pierre Marie (1853-1940) and Constantin von Monakow (1853-1930).

Marie considers apraxia to be an intellectual disorder.[30] In 1914, he and his student Charles Foix (1882-1927) published an article bearing a true statement of intent as a title: "Phénomènes dits apraxiques, avec lésion du lobe pariétotemporal gauche" (Apraxic phenomena with a lesion to the left parietotemporal lobe).[31] They described two patients with Wernicke aphasia who presented gestural disorders, specifically ideational apraxia. They assert that it is not possible to confirm that the parietal lobe represents an ideational eupraxic centre or that ideational apraxia is a distinct entity. In any case, it may

be an indicator of an intellectual disorder "which, should we so wish, we may call ideational apraxia."[31(p276)]

The same year that Marie and Foix questioned the nature of apraxia, von Monakow writes in his monograph *Die Lokalisation im Grosshirn* (Brain localisation) that:

> The main characteristic of Liepmann's and his supporters' approach is the assumption that apraxia is the necessary and probably exclusive consequence of the anatomical interruption of the continuity of very specific and locally well-delimited pathways (long association and commissural fibres) in the brain […]. This idea, accepted long ago by the majority of authors, essentially represents a development of Wernicke's ideas, but in my opinion, can no longer be sustained according to the current knowledge on the physiology of the central nervous system. [32(p503-4)]

In 1916, Foix[33] published a work that resumes Liepmann's taxonomy, although he introduces two relevant changes. The first applies to terminology: he replaces the term ideokinetic apraxia (*ideokinetische Apraxie*) with ideomotor apraxia (*apraxie idéo-motrice*) (Table 1). The second applies to concepts. He agrees with Liepmann on the distinction between a mental and a motor element in action control. However, he disagrees regarding the existence of a mechanism that converts one element into another. According to Foix, there is no solution of continuity between both variants. In line with Marie's ideas, he postulates that ideational apraxia is a mental disorder and ideomotor apraxia a disease of the body, declaring the latter to be the only "true" apraxia.

"New" forms of apraxia: constructional apraxia

In 1909, Conrad Rieger[34] (1855-1939) described how some patients with lesions to posterior areas of the right hemisphere are not able to construct or deconstruct spatial models (as for example, constructing words with letters or models with sticks) due to an alteration to the cerebral spatial apparatus (*räumliche Hirnapparat*).[34] Several years later, in 1918, Walther Poppelreuter[35] (1886-1939) coined the term optic apraxia (*optische Apraxie*) to group the deficits that manifest in propositional acts requiring visual control (eg, drawing, using scissors, getting dressed, or navigating in a familiar setting). Optic apraxia includes alterations to grasping, pointing, and navigating in space, which were considered to be preserved in previous conceptions of apraxia. The excessively broad range of activities included under this conception led Kleist to reformulate the concept, renaming it constructional apraxia.

In 1934, Kleist published *Gehirn-Pathologie vornehmlich auf Grund der Kriegserfahrungen* (Cerebral pathology mainly due to war experiences).[36] This monumental work[E] includes a chapter entitled *Konstruktive (optische) Apraxie* (Constructional [optic] apraxia), in which Kleist proposes that this is a specific disorder "in which spatial action fails with no apraxia of the individual movements."[36(p483)] In fact, he excluded from constructional apraxia those disorders that may be explained in terms of visuoperceptive alterations or ideomotor apraxia. For Kleist, constructional apraxia is the result not of a perceptual deficit but of a failure in the integration (disconnection) between visuospatial processes and kinesthesic engrams due to lesions to the parieto-occipital region of the left hemisphere.

Constructional apraxia rapidly gave rise to multiple neuroanatomical and conceptual uncertainties. Johannes Lange (1891-1938) disagreed with the anatomical substrate postulated by Kleist. He believed the right hemisphere to play an essential role in visuospatial functions, and that lesions to this hemisphere may lead to visuoconstructive disorders (including constructional apraxia).[37] Benno Schlesinger[38] (1900-1983) considers constructional apraxia a special form of limb-kinetic apraxia due to an alteration of the optical control of movement. Other authors, such as Mikhail B. Kroll (1879-1979) go even further, questioning the existence of this type of apraxia. In 1933, Kroll and Stolbun wrote that "there is no sufficient evidence to consider constructional apraxia as a new and special type."[39(p142)] They consider that "this is no more than a new and valuable research methodology that is able to show new characteristics of apraxia […]. The assessment of constructional ability showed that the apraxia also includes an agnostic component."[39(p158)] In fact, Kroll is a strong supporter of the unified interaction between gnosis, praxis, and language.

[E]This work was initially intended be included in the fourth volume of *Handbuch der ärztlichen Erfahrungen im Weltkriege 1914/1918* (Manual of medical experiences in the World War of 1914-1918), published in 1922, which suggests that Kleist already had a definition for constructional apraxia in the early 1920s.

Table 1. Terminological development of taxonomies of apraxia.

Heilbronner (1905)	Pick (1905)	Liepmann (1905)	Liepmann (1908)	Foix (1916)
Cortical apraxia			Limb-kinetic apraxia	
Transcortical apraxia	Motor apraxia	Motor (innervatory) apraxia	Ideokinetic apraxia	Ideomotor apraxia
Conduction apraxia	Ideomotor apraxia	Ideational apraxia	Ideational apraxia	Ideational apraxia

The close connection between apraxia and agnosia

In the late 1920s, Joseph Morlaas (1895-1981) and Abraham Anton Grünbaum (1885-1932) revisited the 19th-century line of reasoning that relates gestural disorders with perceptual disorders.

Morlaas, a student of Charles Foix, published in 1928 his work *Contribution à l'étude de l'apraxie* (Contribution to the study of apraxia).[40] He suggests that ideomotor apraxia is the result of a disturbance of gesture, whereas ideational apraxia is explained by problems in the manipulation of real objects. An individual with ideomotor apraxia is able to recognise the symbolic form of the gesture he/she is not able to perform, whereas a subject with ideational apraxia may name and describe the use of objects he/she is not able to use. According to Morlaas, ideational apraxia originates in apraxia of use. Ideomotor apraxia is a disturbance of gesture in itself, caused by spatial dyskinesia (*dyskinésie spatiale*).

The spatial dyskinesia described by Morlaas refers to an incorrect movement leading to an erroneous final result of the gesture (eg, the hand over the eye in a military salute or a thumb in the mouth when the desired/requested gesture is to put the thumb to the nose); this result is due to the loss during the gesture of the intuitive notion of the relationship between the moving limb or hand, which tries to stop, and the bodily coordinates. Spatial dyskinesia is the equivalent, for gesture, to the planotopokinesis described by Marie, Bouttier, and Bailey.[41]

In 1922, these authors described a patient who failed to perform actions requiring the integrity of spatial representation:

> When the patient is asked to put on a shirt (...) he seems absolutely disoriented, rather than clumsy. He successively grabs each end, examining them, hesitating and turning the shirt in all directions, and only after a few minutes, he lifts the shirt to the level of his head; most times, he is not able to place it over his head and is blinded by the flapping fabric while he incoherently tries to get out of it.[41(p507-8)]

These authors propose the term planotopokinesis (*planotopokinésie*, from the Greek *plan-* "errant", "elusive"; and *topos* "place") to refer to a disorder of spatial representation in the execution of voluntary movements. In 1941, Walter Russell Brain[42] (1895-1966) described the inability to dress oneself as a separate entity within apraxia (apraxia for dressing).

In 1930, Grünbaum[43] developed a proposal that is conceptually opposed to the associationist theories of apraxia (with Liepmann being the main representative). The author stresses that action is not constructed from motor memory images; rather, images are activated based on action. Grünbaum asserts that the aim of the action is frequently derived from the recognition (memories) of possible motor interactions with objects. For example, the same piece of paper is recognised differently depending on the different behavioural moments: at one time, it may be something to write on, and at another, it may be used for wrapping. Therefore, the deficient use of objects

(or inability to use them) may be the consequence of erroneous recognition of qualities of the object that are linked to a certain action. Grünbaum deems it impossible to establish a clear distinction between the agnostic and apraxic aspects of actions, and suggests replacing the term apraxia with apractoagnosia (a term conceptually close to the apraxia of use defined by Morlaas).

Conclusions

The scientific study of gestural disorders underwent an immense conceptual evolution in a very short period of time: from the asymbolia described by Finkelnburg to the apraxia defined by Liepmann, by way of the mental palsy described by Nothnagel. Regarding the term apraxia, by the late 19th century, such authors as Kussmaul believed that difficulty using familiar objects is caused by a perceptual deficit. Apraxia gained its current meaning with Liepmann. In the early 20th century, several taxonomies of apraxia were proposed at the same time that the relationship between apraxia and agnosia was being analysed. Many authors questioned the existence of pure forms of apraxia and agnosia. In fact, in *Ueber Störungen des Handelns bei Gehirnkranken* (1905), Liepmann admits that "in my patient [the imperial counsellor], we may even identify a mixture of partial agnosia, which I have so far disregarded in order first to underscore the main contradictions."[23(p107)]

Limiting a study to a historical period (in this case from 1860 to 1935) necessarily leads to significant omissions of relevant contemporary contributions. In the second half of the 20th century, we should underscore the contributions of Critchley (1953),[44] Denny-Brown (1958),[2] de Ajuriaguerra and Hécaen (1964),[1] Geschwind (1965),[45] and Luria (1966),[46] without neglecting the cognitive models proposed by Roy and Square (1985),[47] or Rothi, Ochipa, and Heilman (1991).[48]

Conflicts of interest

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