Apraxia
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Apraxia
The Cognitive Side of Motor Control

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Preface

Apraxia is a fascinating syndrome. Clinical observations of patients who cannot decide whether a fork or a knife is the right instrument for slicing bread, who are unable to replicate the movement of cutting with scissors immediately after they have used them, or whose left hand withdraws objects from the right hand but gives them readily to other persons yield intriguing insights into the fragility of the deliberate control of human action. Apraxia is also a fascinating topic of research. Its scientific exploration has a history of some 140 years and continues to produce novel and exciting insights. The twists and controversies of this long history touch on core issues in our understanding of mind and brain.

The aim of this book is to provide a comprehensive review of history, clinical appearance, and scientific research on apraxia. The review is guided by the hypothesis that apraxia is a disturbance at the boundary between cognition and motor control. Its position on one or the other side of this boundary is a topic of controversy that runs as a central thread through conflicting theories of apraxia. The attraction of apraxia as a field of theorizing and research owes much to this ambiguous position that ultimately refers to a mind–body dichotomy.

I hope that the width of its scope will make the book attractive to readers with backgrounds ranging from therapeutic disciplines, medicine, neuropsychology, and neuroscience to history and philosophy. I tried to write understandably for all of them and to explain terms and facts that are evident for specialists but unfamiliar for readers from other disciplines. Some very basic notions of clinical neuroscience, for example, that lesions of one side of the brain cause motor impairments of the opposite side of the body should suffice for following the course of the arguments. French and German quotes from early work on apraxia have been translated by me. Keeping a balance between literal translation and comprehensibility was sometimes a challenge particularly for nineteenth-century German texts. When comprehensibility was endangered, I supported it by comments on the concepts that underlay the choice of words.

I have researched and published on aspects of apraxia for nearly 30 years. I do not claim exception to the long-standing scientific tradition of considering one’s own contributions as being exceptionally important and reliable, but I have made a serious effort to adhere also to the somewhat less universal tradition of giving due space and attention to the work of others. Out of 600 references cited in this book, 51 have been authored or co-authored by me. This is, I would say, a decent proportion. I have strained to discuss controversial results and theories in a fair and balanced way even if one of the controversial positions was my own.

There are many persons who helped me in writing this book. First of all, I want to thank the patients who consented not only to being videotaped but also to the use of these
records for cartoons illustrating their problems. For them, apraxia is less a source of fasci-
nation than an intriguing assault on lifelong established competency and autonomy. The
nurses and therapists of our department gave me precious insights into the consequences
of apraxia for daily living and the possibilities and limits of their therapy.

Armin Schnider encouraged me to propose this book to Oxford University Press where
a competent team accompanied me from the first synopsis to the final production of the
book, and Charlotte Green was always ready to answer my questions.

Paul Eling gave me critical feedback on the historical chapters. Joachim Hermsdörfer
and Wolfram Ziegler read single chapters and Joseph Spatt a first draft of the whole book.
Discussions with them were extremely helpful for clarification of my own position with
respect to the boundary between cognition and motor control. Philippe Peigneux and An-
dreas Marneros provided me with copies of influential nineteenth-century contributions
preceding Hugo Liepmann’s seminal first report of apraxia, and Ioanna Athanasoupoulou
shed light on the confusing nomenclature of the first modern accounts of intermanual
conflicts by Andrew Akelaitis.

Dani Goldenberg drew the cartoons for illustrations and Anna Goldenberg advised
me to make sentences short. Their affection is the solid ground on which my life and this
book rest.

Georg Goldenberg
Munich, February 2013
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Chapter 1

Apraxia before Liepmann: Mind-palsy, asymbolia, and apraxia

As a first approximation, apraxia can be defined as a disturbance of the mental control of deliberate motor actions. Apraxia is a clinical syndrome with a long history, the beginning of which is usually identified with the seminal writings of the German psychiatrist Hugo Karl Liepmann in the first decades of the twentieth century (Goldenberg, 2003a), but disturbed mental control of deliberate movements had been subject to clinical observation and theorizing before Liepmann. The clinical literature of the late nineteenth century recognized three syndromes characterized by wrong or awkward actions in spite of preserved motor strength and coordination: mind-palsy, asymbolia, and apraxia.

Mind-palsy

Usually, the recognition of new clinical syndromes starts with clinical observations which do not fit in established diagnostic categories. When the reliability of the observation has been established, the next step is a search for underlying mechanisms, possibly supported by experimental studies. Finally, explanations of the syndrome may lead to a revision of basic theoretical assumptions.

The syndrome of mind-palsy developed in the opposite direction. Its starting point was a general theory of localized brain function. The framework of the theory led to the expectation of a hitherto unknown syndrome which was then sought for and allegedly found in animal experiments. Only then were clinical observations adduced, which pointed to the existence of the syndrome in patients with brain damage. We will follow this course and discuss first the basic theoretical model, then the animal experiments, and finally the clinical observations.

Theoretical foundations

The concept of mind-palsy (“Seelenlähmung”) was based on an associationist\(^1\) model of brain organization (Figure 1.1), which had been elaborated by the Viennese psychiatrist

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\(^{1}\) This approach to brain functions has also been termed “connectionist” (Caplan, 1987; Eling, 2011). This designation has the advantage of emphasizing the distinction from the British version of associationism that culminated in the writing of John Hughlings Jackson (Young, 1990; see Chapter 3) but the disadvantage that it is still in use in modern cognitive science where it characterizes computerized network models of cognitive functions (McClelland et al., 1986; Fodor & Pylyshyn, 1988). I prefer “associationism” because it is not in common use any more and thus underlines the historical nature of the present discussion.
Figure 1.1 An associationist schema illustrating the motor reaction of the hand to the sight of a candle and the sensation of heat. The blue lines indicate centripetal, the red lines centrifugal, and the black lines association tracts. In this schema, neural processing beyond the incoming of sensation and outgoing of motor commands is limited to uninterrupted connections from the cortical end points of the sensation tracts (A and B) to the origin of the motor command (C). A: a point within the visual center; B: a point within the center for cutaneous sensations; C: a point within the territory of innervation sensations; ccO: occipital cortex; F: frontal cortex; 1: tract leading sensations from hand; 2: tract of movement of arm; 4C: tract for sensations of innervation; 5: centrifugal tract originating from C. Reproduced from Meynert, T. Klinische Vorlesungen über Psychiatrie auf wissenschaftlichen Grundlagen für Studirende und Aerzte, Juristen und Psychologen, p. 147 © 1889, Wilhelm Braumüller. (See Plate 1.)
and anatomist Th eodor Meynert (1874), and applied to the study of aphasia by his disciple Carl Wernicke (1874) (Figure 1.2).

The empirical background for their model of brain function was the discovery that nerves transmitting input from peripheral sense organs, as well as nerves transmitting motor commands to peripheral muscles, are rooted in circumscribed regions of the cerebral cortex. For example, the optic tract brings visual information from the eyes to the occipital cortex, and excitation of motor cortex located in the central cortex triggers movements of the limbs (Fritsch & Hitzig, 1870; Munk, 1881; Young, 1990). The associationist model of brain function assumed that excitation in the cortical end points of afferent nervous tracts does not completely vanish when peripheral stimulation ceases. The remnants of past sensations are stored as “memory images” in cortical areas surrounding the end points. Likewise, movements of the body or the limbs give rise to memory images of the executed movement which are stored near the cortical region where the motor commands are generated. Localization of cerebral function is confined to such simple memory images surrounding the anatomical end point of the nervous pathways carrying sensations to, or movement commands away from, the cortex. These memory images are, however, richly interconnected by fiber tracts. Due to these connections, memory images can be evoked and recombined also in the absence of peripheral stimulation or action. They thus form the substrate of “intrapsychic” processes which defy further reduction to localizable elements:
Memory images of sensations on the one hand, of movements of the own body on the other, are the elements provided by outer reality for constituting the contents of consciousness. Everything beyond these most simple functions, the combination of different sensations to a concept, thinking, consciousness, are an achievement of the masses of fibres which link the different sectors of the cerebral cortex among each other. (Wernicke, 1874, pp. 4, 9)

Incoming sensations must make connections with corresponding memory images of the same modality in order to be integrated into the multimodal network. If such integration fails, the sensation remains isolated and meaningless. There are thus two ways how cortical lesions can interfere with the perception and comprehension of external stimuli: destruction of the area where the pathways from the periphery reach the cortex would lead to “cortical” losses and destruction of the surrounding memory images to “mind” losses. Depending on the modality of the sensation whose memory images were lost, the theory predicted the existence of mind-blindness, mind-deafness, and mind-numbness (Munk, 1877; Lissauer, 1890). By analogy, a loss of motor memories should result in “mind-palsy.”

The search for clinical correlates of these theoretical predictions was conducted in animal experiments before it was applied to clinical observations in humans.

Animal experiments

Berlin around 1870 was a good place for scientists interested in the anatomy and physiology of the human brain but was a bad place for dogs. They were the preferred subjects of experimental studies exploring the effects of stimulation or destruction of circumscribed parts of the brain. They had to sustain stress and pain from surgical procedures, which in the beginning were carried out without anesthesia, and some of them died from bleeding or inflammation of the exposed brain (Fritsch et al., 1870; Munk, 1877). When the experiment was successful, the dogs remained mutilated for the rest of their lives.

2 Successful connection of incoming sensations to memory images of the same modality does not necessarily guarantee integration into the multimodal network of memory images, since there can be interruption between memory images of the same modality and associated images from other modalities. Heinrich Lissauer, a disciple of Wernicke, described this possibility for the visual modality and suggested naming it “associative” mind-blindness (Lissauer, 1890).

3 The German expressions were “Seelenblindheit,” “Seelentaubheit,” “Seelenfühllosigkeit,” and “Seelenlahmung.” In English literature they have sometimes been translated as “psychic blindness.” I prefer the combination with “mind” because “psychic paresis” has a connotation of paresis from non-organic causes like hysteria or conversion disorder. “Mind-palsy” was used by Wilson (1908) in a review of the current state of the art in apraxia. In German, the word “Seele” means both the mind and the immortal soul. Munk, who introduced the terms, addressed possible misunderstandings of “Seele” in a footnote regarding mind blindness (“Seelenblindheit”): “I choose this designation in 1877 after long reflection and I thought to have good reasons for preferring it to ‘image-blindness’ (’Vorstellungsblindheit’) or ‘memory-blindness’ (’Erinnerungsblindheit’). Since I made clear repeatedly that soul-blindness = absence of mental visual images, absence of memory images of visual perceptions, I felt legitimated to consider the use of the word ‘soul’ as harmless as if I had used α-blindness or β-blindness.” (Munk, 1881, p. 53)
In 1870, the anatomist Gustav Fritsch and the psychiatrist Eduard Hitzig attacked two contemporary beliefs about the cerebral cortex: that it could not be excited by electrical currents and that it had no direct access to motor actions of the limbs (Fritsch et al., 1870). They removed parts of a dog's skull and applied weak electrical currents to the bare surface of the brain. When such stimulation was administered to the anterior part of the brain it elicited contractions of muscles on the opposite side of the body, whereas no such reactions could be obtained by even much stronger currents applied to the posterior part. Further explorations of the effects of weak currents revealed specializations within the anterior part of the brain. There appeared to be fairly constant localizations where stimulation elicited motor twitches of the mouth, the neck, the foreleg, or the hind leg. Fritsch and Hitzig concluded that the cerebral cortex could send motor commands to the muscles, and that the cortical origins of these commands were laid out in a somatotopic map, so that the effects of local stimulation were body part specific.

While these observations had an enormous impact on brain research in Germany and beyond (Young, 1990; Finger, 2000), the results of subsequent excision studies are particularly relevant for the concept of mind-palsy. Fritsch and Hitzig opened the skull on the left side and excised a lentil-sized piece of cortex at the location where stimulation had elicited movements of the right foreleg. The excision did not result in a complete paralysis, but motor actions of this limb became somewhat awkward. During walking or standing the affected limb tended to slide away or to touch the ground with the dorsum instead of the sole. After partial recovery, one of the dogs showed a more spectacular symptom: When he was standing and the experimenter placed his right forelimb into an uncomfortable position, for example, amid the other three legs, the dog would neither protest nor try to bring the foreleg back into its natural position. When, however, the dog started to run, the leg was immediately brought back in its correct position and participated in running. Fritsch and Hitzig denied any deficiency of sensory aff erences, but nonetheless concluded that the dog “apparently had only defective awareness of the conditions of this limb. He has lost the ability to form a complete mental image of that limb.” (Fritsch et al., 1870, p. 331).

**Introducing the parietal lobes**

In Meynert’s and Wernicke’s version of associationism, memory images of sensations and of movements equally contributed to the “contents of consciousness.” In the further development of the concept of mind-palsy their equality was replaced by the assumption that only memories of sensations give rise to conscious mental images. According to the “ideo-motor principle,” voluntary movements had their origin in mental images of their sensory consequences (Prinz, 1987). The motor mechanisms that bring forward the intended consequences were believed to run automatically outside the realm of consciousness. Translated into anatomy, the “ideo-motor principle” shifted the possible source of mind-palsy from the motor cortex located in the frontal lobes to sensory regions located in the parietal lobe (see Figure 1.3). The further development of the concept of “mind-palsy” reflects this basic shift.
In 1878, the physiologist Hermann Munk (1878) replicated Fritsch and Hitzig’s experiments. After excision of only a few millimeters of left-sided cortex, he found impairment of the right forelimb almost identical to the previous description by Fritsch and Hitzig. Munk emphasized the contrast between the lack of isolated deliberate movements of the affected limb and its swift integration into global movement patterns involving all limbs, as, for example, in walking. He referred the leg’s immobility to the loss of limb-specific mental images of movements, but specified that these images are not equivalent to stored motor actions. They are sensory images of the tactile or kinesthetic feedback associated with a movement. These movement images elicit execution of the imagined movement, because “the generation of a movement image posits eo ipso the corresponding motor action” (Munk, 1878, p. 178). Therefore, the consequences of excisions depended on the affection of sensory areas:

Within the sensory area of each body part small excisions cause a partial loss of sensory images of that body part, larger excisions a complete loss: mind-palsy of that body part. (Munk, 1878, p. 176)

The assumption that the causal damage in mind-palsy affects kinesthetic memory images of the moving limb rather than the motor cortex directing the movement was foundational.
for the belief that the crucial lesions for mind-palsy affect the parietal lobes. It was further elaborated in the first descriptions of putative human analogs to the experimentally induced disturbances of motility in dogs.

**A human case of mind-palsy**

In 1887, the German internist Hermann Nothnagel reasoned that the memory images, whose destruction should give rise to mind-palsy, cannot be stored within or very close to the “motor centra” which transmit motor commands from the cortex to the periphery, because destruction of the motor-centra causes paralysis of the opposite limb but leaves intact the will to execute movements of the paralyzed part. He explained the preservation of the will to move by preservation of the conscious mental image of the intended motor action and concluded that this mental image must have a different neural substrate than the commands directing execution of the movement. Nothnagel suggested that “the field of motor memory images lies in the parietal gyrus. The motor neurons in the paracentral and central region only transmit the motor command” (Nothnagel, 1887, p. 214). Consequently, mind-palsy should result from parietal lesions.

Nothnagel did not support his conclusions with clinical observations of mind-palsy nor did he elaborate on the expected clinical features. The first detailed report of a presumed human case of mind-palsy was published 15 years after Munk’s creation of the syndrome, by the Swiss psychiatrist Eugen Bleuler (1893). Bleuler gave a very detailed and lengthy description of aphasia and other symptoms in a patient whose lesions analyzed post-mortem affected, among other regions, left supramarginal and bilateral anterior parietal regions. This patient had an incomplete paresis but a complete sensory loss of his right arm. He could move the shoulders and the upper arm when he was looking at them, but “when the patient does not see his right arm, he is not only unaware of the arm’s momentary position, but he is also completely unable to innervate any of its muscles” (Bleuler, 1893, p. 38). Bleuler reasoned that the inability to move the arm without visual control was due to the absence of kinesthetic motor memory images. Referring to Nothnagel, he classified the disturbance as mind-palsy.

**From sensory memory images to mental processes**

A few years later, but still three years ahead of Liepmann’s first paper on apraxia, Ludwig Bruns (1897) contributed a further case report together with an extensive discussion of mind-palsy. The patient was a luetic musician who suddenly developed aphasia, right-sided hemianopia, right-sided hemianesthesia, a mild paresis of the right leg, and a strange motor disorder of the right arm:

The patient never uses the right arm spontaneously; it lies beside him as if it were completely paralyzed. He offers the left hand for greeting, eats with the left hand, takes his pinch (of tobacco) with the left hand, and uses the left hand for blowing his nose. He can be prompted to use the right hand only by long verbal encouragement. It seems that at first he does not understand what he is expected to do and that this irritates him. If one wants him to raise the right hand to his nose, one must withhold his left hand and demonstrate the path of his right hand to the nose by passive movement.
Then, he will eventually execute the movement himself. In the same way it is possible to finally get him to give the right hand for greeting by withholding the left hand or refusing it repeatedly and asking for the right hand. Likewise, after long encouragement, he leads the spoon to the mouth with the right hand.

The patient is aware that something is wrong with his right hand. He frequently looks at it with astonishment and calls it: “you bastard.” (Bruns, 1897, p. 379)

Surprisingly, when actions of the right hand could be induced at all, movement strength was normal and dexterity only mildly reduced. Unlike Bleuler’s patient, this patient did not need to look at his hand in order to control it.

This peculiar disturbance of right-hand motor control recovered within a few days and only a slight awkwardness remained. The patient could even play the piano again, although his right hand sometimes missed the keys and hit the edge of the piano. In spite of this amelioration he died a few weeks later. Post-mortem examination revealed a left superior temporal lesion which extended parietally into the angular gyrus and the white matter underlying the supramarginal gyrus.

Bruns followed Bleuler in searching for the source of the problems on the sensory rather than the motor side. His emphasis on the importance of intact sensory representations for deliberate motor control went even further than Bleuler’s ideas. Whereas Bleuler had considered only kinesthetic sensations as being crucial for motor actions, Bruns reasoned that connections from all sensory modalities can elicit motor actions. He defended a radical response to the question whether mind-palsy was due to loss of motor or sensory memory images. He emphasized the importance of sensory and downplayed that of motor images:

Every “deliberate” movement has its source in a stimulus originating from a sensory centre. Intactness of these sensory centres and their connections is as necessary for deliberate movements as is the intactness of the so-called motor centres: After all, these motor centres are nothing more than the point where the sensory part of an intended movement turns into its motor part, and it is impossible to indicate exact borders between them. (Bruns, 1897, pp. 383–384)

Interruption of the connections from these centers deprives the motor centers of sensory stimulations and results in spontaneous disuse of the extremity, as had been observed in the case of the luetic musician.

To clarify the importance of the involvement of multiple sensory centers in the preparation of deliberate movements, Bruns compared them to simple reflex movements like the knee jerk. In these primitive reflexes, one specific sensory stimulus (tapping below the knee) always elicits the same specific reaction (extending the leg), and they are based on direct subcortical or (as in the case of the knee jerk) spinal connections between sensation and motor control. By contrast, for deliberate movements, the path from sensation to motor response travels through the cortex and is modulated by the inclusion of cortically stored memory images. The inclusion of memory images mitigates the tightness of the association between sensation and motor response. External sensory stimuli lose the power to firmly determine the nature of the motor response. They give way to mental processes mediating between stimulus and response:
Mental processes are based on associations between sensory centres distributed across the whole cortex—therefore mental processes cannot be localized in the same way as their single constitutive parts—they always demand the whole or a great portion of the brain. Mental processes express themselves by muscular actions. Via the association tracts they stimulate motor regions and evoke movements. If these tracts are interrupted the mind cannot influence movements any more—there is mind-palsy: Deliberate movements are absent, while reflexes in a narrow sense come to the fore without restriction. (Bruns, 1897, p. 387)

Since cortical sensory centers are located in the posterior part of the hemisphere and motor centers in the frontal region, the cortical path from sensation to motor action leads from posterior to anterior brain regions. It thus resembles the neural connections underlying the most primitive spinal reflexes, where the nerves carrying sensory stimulation enter the posterior part and those exciting the muscular response originate from the anterior part of the spinal cord.

The alleged dissociation between preserved reflex and defective deliberate movement transgressed the clinical evidence of Bleuler’s and Bruns’ case reports. No such dissociation had been noted by Bleuler. Bruns adduced as evidence that his patient used the right hand to scratch himself when the left one was restrained. However, restriction of the left hand could also bring forward less reflex-like movements, like greeting (see earlier extract). Arguably, the alleged dissociation between deliberate and reflex movements owed more to theoretical expectations than to clinical observations.4

Mind-palsy as a physiological concept

Although it bears the notion of mind and in German even the immortal soul in its name, mind-palsy is essentially a physiological rather than a psychological concept. “Memory images” are traces left by sensations in cortical areas and their connections. Both the generation and the destruction of memory images are completely determined by transformations or destructions of cells and fiber paths. Mental states are thought of as the product and not the cause of physiological changes. The lack of spontaneous movements of the right hand of Bruns’ patient was not attributed to deficient understanding or unwillingness or any other mental state, but to destruction of either sensory centers or fibers connecting them with motor centers. It is significant that the syndrome was described in dogs before it was searched for in human beings. While it is reasonable to assume that the physiology of sensation and motor control is similar in humans and other animals, it would seem harder to argue that the mental capacities of dogs are a good model for understanding human behavior.

An important argument in favor of the physiological nature of mind-palsy was its body part specificity. Mind-palsy affected only the limbs on the side opposite to the lesioned hemisphere, and the animal experiments even suggested that it could be restricted to only one part of the limb. The explanation for this body part specificity was sought in the

4 The opposition between propositional and automatic movements was central to the writings of John Hughlings Jackson. We will come back to his influence on the science of apraxia in Chapter 3.
somatotopy of the motor cortex rather than in differences between the mental processes associated with movements of different body parts.

The literature on the anatomical substrate of mind-palsy did not consider possible differences between the hemispheres. In the cases reported by Bleuler and by Bruns, the lesions happened to be in the left hemisphere and mind-palsy affected the right limbs, but in their presentations there is no hint of a suspicion that right-sided lesions would not cause the same kind of mind-palsy of the left limbs. The indifference to laterality is remarkable, because in the last decade of the nineteenth century the left hemisphere’s dominance for speech was already firmly established. It was, however, consistent with the associationist doctrine that the cortical end points of nerves leading to the periphery are the firm poles determining the extension of associative fiber networks. The layout of the cortical origins of sensory and motor nerves does not differ between hemispheres. This symmetry was considered to be more relevant for understanding functional divisions of the brain than the strikingly different effects of right- and left-hemisphere lesions on speech and language.

The legacy of mind-palsy

From the point of view of modern neuropsychology, Bleuler’s case would probably be classified as an instance of “kinesthetic ataxia” (alternative terms are “afferent apraxia,” “tactile apraxia,” and “parietal hand”; Luria, 1980; Freund, 1987; Goldenberg, 2003c), and Bruns’ case as motor neglect (Laplane & Degos, 1983; Coulthard et al., 2008). Possibly, they were the first detailed descriptions of these disorders, but their historical importance lies elsewhere. In Chapter 2, I will argue that Bruns’ interpretation of mind-palsy came as close as possible within a strictly associationist framework to Liepmann’s analysis of ideokinetik apraxia.

Before we leave mind-palsy let me briefly sum up features of this syndrome which recur in Liepmann’s elaboration of apraxia.

There is a stream of action control from posterior to anterior brain regions in which the parietal lobe plays a central role. Along this stream, sensory images of the intended actions are transferred into motor commands which produce a muscular expression of the sensory image. Interruption of the conversion of sensory images into motor commands causes a body part-specific inability to perform voluntary actions.

Asymbolia

In his seminal report of aphasia following a left frontal lesion, Paul Broca (1861) classified the patient’s disorder as a selective loss of articulated speech with preservation of other mental functions. He remarked, however, that “unable to manifest his ideas or his desires other than by movements of his left hand, he frequently made incomprehensible gestures,” and that “some questions to which a man with normal intelligence would have found a mean to respond by gesture remained unanswered.”
Nearly ten years later, the German psychiatrist Carl Maria Finkelnburg (1870) criticized the tenet that aphasic patients had a selective loss of speech and expanded on their defective production and comprehension of non-verbal conventional signs. He had observed an aphasic musician who could no longer read musical notes, a salesman who confused the values of different coins, and a government official who could not distinguish rank signs and who had forgotten how to behave during Mass. The problems were not confined to interaction with external signs or rules but also concerned the patients’ gestural expressions. Thus, an aphasic woman “who had been raised as a devout catholic never made the sign of the cross at the common grace. When asked by her surrounding to make it, she hesitantly reached sometimes behind the ear, sometimes to the neck until it was demonstrated to her. Then she imitated it correctly.” The salesman’s “mimic expression during speaking was exaggerated and gross, his gestures awkward and sometimes completely incongruent to what he wanted to express” and in another patient “mimic expression and gesticulation become gross and incomprehensible, and the comprehension for pantomimes made by other persons diminished.”

Finkelnburg concluded that the term “aphasia” was ill-chosen because the language disturbance was only one of several manifestations of a general “asymbolia,” that is, “a pathological disturbance of function where the ability to understand or express concepts by means of learned signs is partially or completely abolished.”

Finkelnburg invoked philosophy as support for the existence and importance of symbolic abilities:

The important and independent role of symbolic abilities for the reproduction and combination of mental images has long been acknowledged by philosophical schools of thought. Kant, for example, calls this ability, to which he dedicates a whole section of his *Anthropology*, as “facultas signatrix” and its accomplishments as “symbolic cognition.” (Finkelnburg, 1870, p. 461)

Concerning the cerebral substrate of asymbolia, Finkelnburg referred to Meynert’s anatomical findings and reasoned that the central part of the hemisphere, would be the most likely seat of responsible lesions, because of the plenitude of fibers connecting it with many different sectors of the cortex and multiplying their interactions.

Finkelnburg also discussed the laterality of lesions. Not surprisingly, all but one of his aphasic patients had left-sided lesions as manifested by their right-sided motor symptoms. Finkelnburg complained that the reasons for the asymmetry of lesions causing aphasia or, respectively, asymbolia had not yet been elucidated. He discussed but dismissed the possibility that due to asymmetry of vascular anatomy the left hemisphere is more likely to be the target of brain damage than the right, but he also refused the “paradoxical idea of French authors that as a rule the organ of language competence becomes functional only