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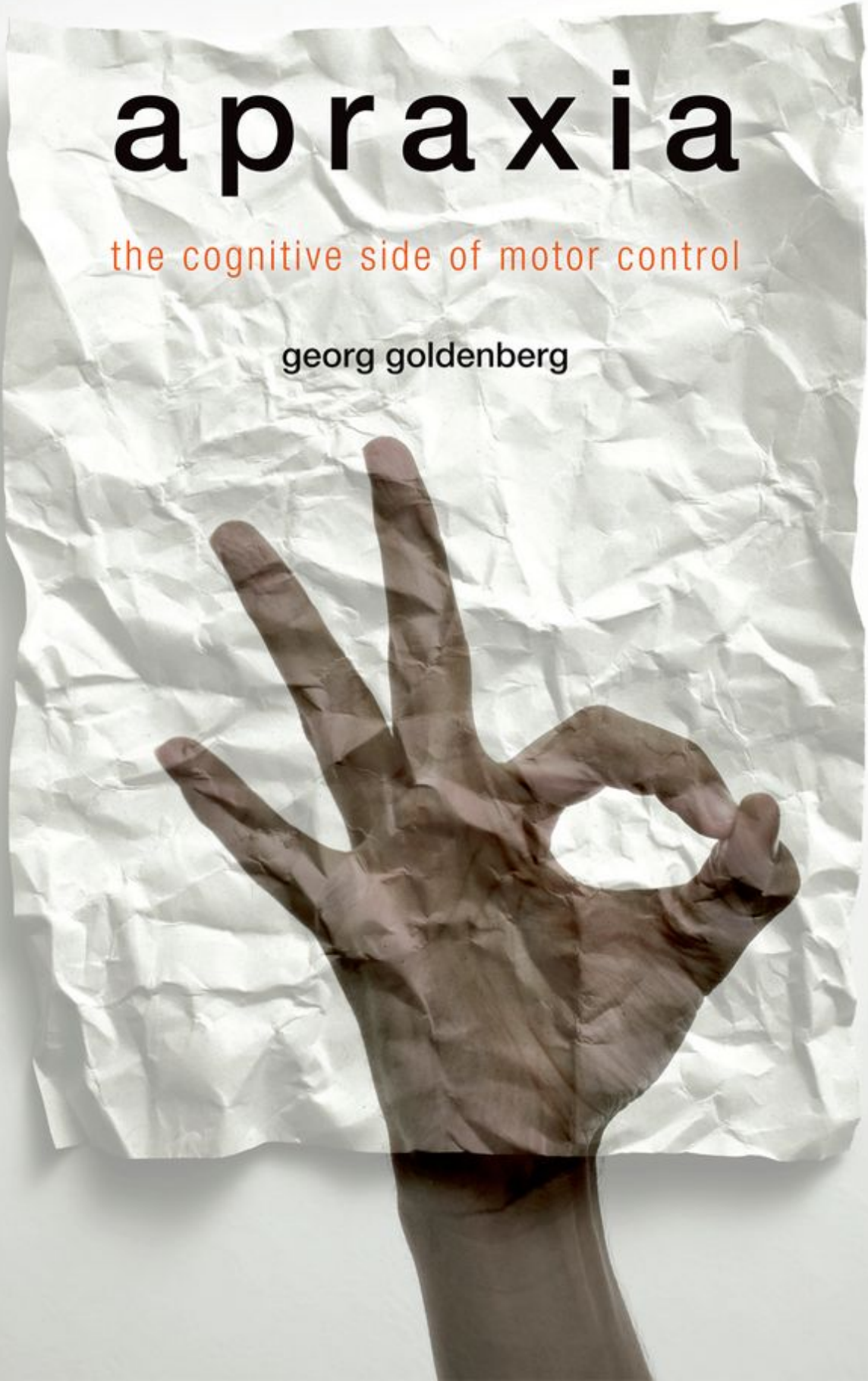
apraxia

the cognitive side of motor control

georg goldenberg



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The Cognitive Side of Motor Control

Georg Goldenberg

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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 fascination 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 Andreas 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

[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.

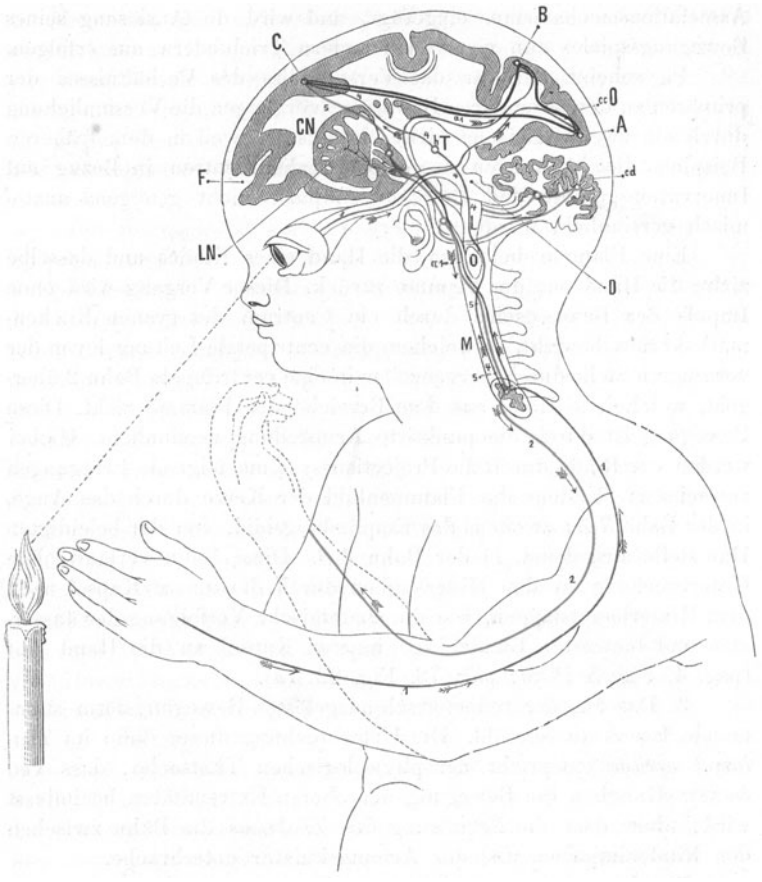

Figure 1.1 An associationist schema illustrating the motor reaction of the hand to the sight of a candle and the sensation of heat. The blue lines indicate centripetal, the red lines centrifugal, and the black lines association tracts. In this schema, neural processing beyond the incoming of sensation 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.)

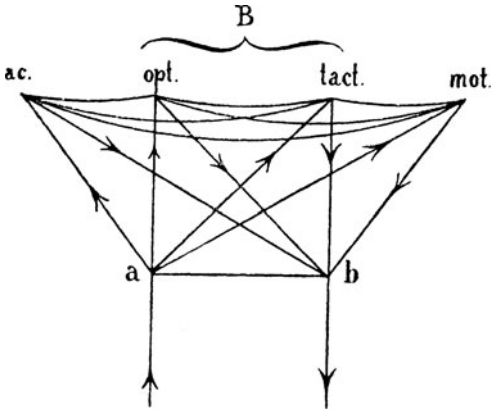

Figure 1.2 Wernicke's (1893, p. 100) schema is primarily concerned with understanding and production of words rather than with the control of limb movements. It is included here to illustrate the generation of psychic processes from associations between multimodal memory images. The elements of the schema are stored memory images. a: acoustic form of words; b: articulatory pattern of words. Ac, opt., tact., mot.: acoustic, optical, tactile, and motoric memory images associated with the perception and manipulation of objects. B: concepts of objects; although the parenthesis encloses only the optical and tactile memory image, the text leaves no doubts that the concept is composed of memory images from all modalities. The connections between the modality-specific memory images form a network which constitutes the neural basis of thinking and consciousness. Since these "intrapsychic" processes emerge from interactions across the whole network, they defy narrow localization of their neural substrate. Reproduced from C. Wernicke, Gesammelte Augsätze und kritische Referate zur Pathologie des Nervensystems ©1893, Verlag von Fischer's Medicinischer Buchhandlung.

and anatomist Theodor 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.[2] 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."[3]

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.

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[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 "Seelenlähmung." 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 afferences, 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.

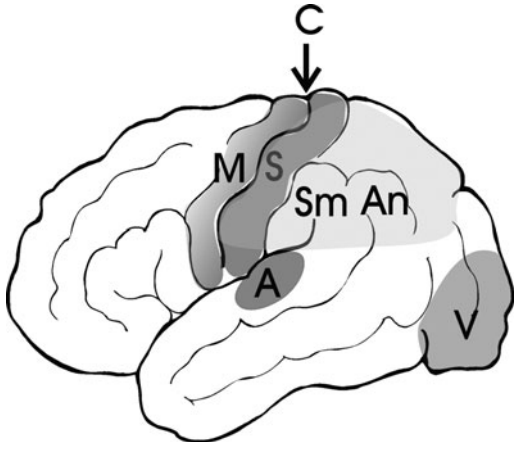

Figure 1.3 A schematic side view of the brain illustrating the anatomical considerations that underlay the discussions about the neural substrate of mind-palsy. According to the associationist model of brain function, memory images are stored close to the location where the original sensations have been received or the original motor commands have been sent out. Motor memories are thus stored in front of the central sulcus, and sensory memories behind it. The postulate that voluntary actions start with a mental image of the sensation of the completed movement and that this sensation is automatically transferred into motor action thus necessitating a stream of association from postcentral parietal to precentral motor areas constitutes a rudimentary form of a posterior to anterior stream of action control (see Chapter 2). A: acoustic cortex; C: central sulcus; M: motor cortex; S: somatosensory cortex; Sm: supramarginal gyrus; An: angular gyrus; V: visual cortex. Blue regions receive sensory afferences from the periphery, whereas the red region sends motor efferences to the periphery. Green denotes the extension of the parietal lobe. (See Plate 2.)

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 paralysed. 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 ideokinetic 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."

> Obviously the deficiency of word production represents only an aliquot—though the one interfering most with the living conditions and the most conspicuous for the surrounding—part of the total disturbance which extends more or less to all brain processes mediating the manifestation of conceptual ideas by learned sensory signs of any kind—symbols. (Finkelnburg, 1870, p. 461)

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

on the left side, analogue to the right hand, and that in left handed persons the right speech centre is filled with learned contents" (Finkelnburg, 1870, p. 461).[5]

Motor asymbolia

Finkelnburg's argument that lesions in the central part of the hemisphere are most likely to cause asymbolia because they interrupt the interactions between multiple regions paid lip service to cerebral localization of functions but implied that asymbolia cannot be ascribed to dysfunction of any single narrowly confined brain region. Asymbolia could be reconciled with Meynert's and Wernicke's anatomical schema at best by identifying it with a disturbance of the whole network of association fibers connecting the cortical end points of peripheral afferences and their surrounding memory images. However, these anatomical considerations were not constitutive for asymbolia. The concept of asymbolia was derived from the psychology of thought and language and not from the anatomy of its neural substrate.

Wernicke (1874) ignored this derivation from the physiological approach to mental functions and equated asymbolia with a loss of memory images. A logical consequence of this interpretation was that, in addition to a general asymbolia where all images are erased, there can be modality specific "asymbolia" which, in the end, is nothing else than the mind losses of functions conceptualized a few years later and without reference to asymbolia by Munk (1877, 1878).

In a series of lectures published in 1889, Theodor Meynert did not mention Finkelnburg but declared:

> Wernicke calls the manifestations of the combined loss of memory images as well in the optical as in the acoustical sense and in most cases also in the tactile sense, asymbolia. (Meynert, 1889, p. 224)

In a later lecture, he elaborates the effects of selective asymbolia affecting memory images of motor actions:

> Different areas of the cortex contribute distinguishing features for one and the same object, and the object becomes object of consciousness by their combined sensory stimulation. The loss of each of these distinguishing features is called asymbolia. With regard to the motor modality the distinguishing feature of object recognition is associated with the use of the object. Asymbolia will be revealed by the patient's inability to make proper use of the object. For motor asymbolia it suffices that a softening in the middle portion of the central regions makes the innervation images of the upper extremity inaccessible. (Meynert, 1889, p. 270)

The term "motor asymbolia" found little recognition and soon vanished from the literature, but it left a footprint in the history of apraxia: it figured as a parenthesis in the title of Liepmann's seminal first paper on apraxia (Liepmann, 1900). In later writings, Liepmann recognized "motor asymbolia" as a precursor of "limb-kinetic apraxia" (Liepmann, 1908).

[5] The formulation "paradoxical idea" for the refusal of French ideas sounds rather sharp for a scientific lecture. Finkelnburg's lecture was published in September 1871 and had probably been held shortly before. It may be relevant that the Franco-Prussian War had only ended in May 1871. Finkelnburg had participated in it and had returned "decorated with the iron cross" (Lent, 1896). Apparently the lecture demonstrates his patriotism as much as his clinical astuteness.

Asymbolia and mind-palsy

After the digression to Wernicke's attempt to reconcile asymbolia with his associationist model of brain function, we return to Finkelnburg's original concept of asymbolia. Although Finkelnburg briefly considered the possible anatomical substrate of asymbolia, the concept itself was grounded in psychological or even philosophical considerations rather than in the physiology of brain function.

In contrast to mind-palsy, asymbolia was not meant to be modality specific. It affected production and comprehension of visual and acoustic signs as well as motor actions. Aberrant, awkward, and incomprehensible gestures were symptoms of the general degradation of symbolic aptitudes. Deficient motor control did not contribute to their incomprehensibility. Consequently, the somatotopy of the motor cortex was irrelevant and there was no body part specificity of deficient motor actions. Finkelnburg did not consider the possibility that the woman who was unable to make the sign of the cross with the right hand would succeed when using the left hand.

Apraxia

The first printed appearance of the term "apraxia" was in a book published by the German linguist Chaim Steinthal (1871). Steinthal gave examples of the diversity of clinical manifestations of aphasia, emphasizing that it is not a unitary disorder but a combination of preserved and disturbed verbal and non-verbal capabilities. As an example for non-verbal impairment he reported the observation of an aphasic composer who wrote notes awkwardly, placing the head of quarter-notes to the right instead of the left side of the stem. He continued the discussion of this case:

> The patient had been aphasic and anarthric; yet he had remained intelligent. But when he was asked to write, he grasped the pen upside-down; he also took hold of spoon and fork as if he had never used them before. He asked for his violin, but gripped it so awkwardly that it was impossible to play on it. These symptoms are not equivalent to anarthria but to aphasia, specifically to the confusion of words; because it is not the movement of the limbs which is inhibited, but the relationship between the movements and the object used. The relationship between the mechanism and its purpose is disturbed.
>
> This apraxia is an obvious amplification of aphasia. In another direction aphasia extends to a general inability to comprehend sign, asemia. (Steinthal, 1881, p. 458)

Steinthal then listed examples of "asemia." They were taken from Finkelnburg's paper and made clear that Steinthal considered "asemia" as being synonymous with Finkelnburg's "asymbolia." He did not elaborate on the definition of apraxia. The term was introduced in passing as if it were already in common use. However, this passage is generally acknowledged as being the first printed appearance of the word "apraxia."[6]

[6] I am citing from a reprint of the second edition of Steinthal's book. This second edition was published in 1881, ten years after the first, but the preface of the editor of the reprint states that there were only minimal changes between the first and the second edition.

A psychological approach to brain functions

Steinthal defied the belief in the power of physiology for explaining mental functions (Hitzig et al., 1874; Jacyna, 1999; Eling, 2006). He stated: "Psychology is the indispensable prerequisite for a physiology of the brain" (Steinthal, 1881, p. 473) and accused the prevailing associationist model of cerebral function of overstretching the explanatory value of associations:

> Our medical doctors apparently have not yet realized the insufficiency of the category "association." Neither words, nor kneeling and making the sign of the cross, nor coins or insignia of military ranks are mere associations. Those are certainly present; but they are only the prerequisite for mental processes. And in the same way it is not just due to the inefficiency of associations if someone has forgotten how to use the pen or spoon and fork or to correctly position the bow upon the violin. (Steinthal, 1881, p. 469)

The first sentence of this quote points to another aspect of the debate between proponents of the physiological and the psychological approach to brain function. While physiology was a domain of medical doctors, psychological arguments were brought forward mainly by graduates of humanities (Hitzig et al., 1874; Jacyna, 1999). The division was not strict, however: Finkelnburg, whose proposal of asymbolia clearly belongs to the psychological camp, was a medical doctor. Nonetheless, it may be more than accidental coincidence that Hugo Liepmann, whose model of motor control united both approaches, had graduated in both fields (Goldenberg, 2003a).

Apraxia and asymbolia

Steinthal refused to relate the distinction between apraxia and asymbolia to the existence of different brain centers supporting the comprehension of signs and the use of objects. He wrote:

> The seat of symbolic action must not be separated from that of practical actions. To the machine the meaning of a movement is completely irrelevant. The meaning associated with a movement cannot make a difference to the physiological mechanism, and one and the same movement may sometimes be symbolic and sometimes practical. It cannot make a difference to the physiological mechanics whether I lift an arm for greeting or for working, whether I kneel in the service of god or for doing craftwork. (Steinthal, 1881, p. 468)

The argument that both aptitudes must have a common cerebral substrate because they exert commands of the same motor mechanisms is not necessarily compelling, but it illustrates Steinthal's refusal of cerebral localization as an explanation for dissociations between psychological entities. Instead, he offered a purely psychological explanation for dissociations between disturbed use of symbols and of objects. It started from the observation that disturbances of symbolic abilities are present in many patients and can be associated with only mild degrees of brain damage, whereas defective use of objects occurs only in severe cases in combination with aphasia. Furthermore, object use recovers earlier than symbolic aptitudes. If both abilities were supported by anatomically distinct substrates there would be no obvious reason why one of them should be more vulnerable to brain damage than

the other, and there should be cases where use of objects is affected more severely and recovers later than use of symbols. The assumption that the decisive difference concerns psychological mechanisms rather than cerebral locations can offer a better explanation:

> For a purely psychological theory, the milder brain damage and later recovery of symbolic function than of object use are easily comprehensible. Associations which are based on connections of arbitrary and immaterial, purely subjective, features (and symbols belong to this category) have only little power and get more easily into confusion than associations based on objective relationships. (Steinthal, 1881, p. 471)

It is somewhat confusing that in this quote Steinthal uses the very term "association" that he criticized as being insufficient for understanding mental processes, but there are obvious differences to its use in the associationist model of brain functions. Firstly, it refers only to relationships between mental entities and makes no reference to fiber connections between areas of the brain and, secondly, the coupling of associated entities is modulated by meaningful properties like, in the given example, the arbitrariness of their connection.

The first 30 years of apraxia

Nearly 30 years elapsed between the first edition of Steinthal's book and Liepmann's report of the imperial counselor (see Chapter 2). The term "apraxia" survived this gap although the number of publications referring to it seems to be rather limited. Those I could locate[7] (Kussmaul, 1885; Laquer, 1888; Starr, 1888; Lepine, 1897; Pick, 1898) used the term apraxia to denote wrong use of tools and objects and distinguished apraxia from general dementia, aphasia, and asymbolia, but none of them considered the possibility of a link between apraxia and mind-palsy or other motor disturbances. The source of errors was sought in recognition of tools and objects rather than in motor execution of their use. For example, Kussmaul described an aphasic patient whose speech was characterized by semantic paraphasias and paragrammatism and who:

> calculated correctly, was polite, greeted, and knew the sign of the cross. But he urinated into the washbasin, bit into the soap and did more of such actions which must be referred to misrecognition of objects. He made wrong actions and suffered from what is usually designated as apraxia. It is clearly to see, that in this case the misrecognition of objects which underlies apraxia was much more severe than the misrecognition of expressive signs. (Kussmaul, 1885, p. 199)

In this quote, Kussmaul described examples of misuse that are inextricably linked to the body part involved. One can bite only with the mouth. However, the accusation of defective recognition as being the source of errors left no place for the anatomy of the motor cortex to play a decisive role for the genesis of symptoms which would manifest itself by body part specificity of errors. Provided that the patient was not hemiplegic (Kussmaul did

[7] In 1876, Hughlings Jackson described a woman with a large right-sided tumor who "Now and then would do odd things, she would put sugar in the tea two or three times over, she made mistakes in dressing herself; put her things on wrong side before, and did little things of that kind." He did not, however, use the term "apraxia" for these disturbances (Jackson, 1873/1932c). Jackson's influence on theories of apraxia will be discussed in Chapter 3.

not comment on this aspect of the case), it seems very likely that he carried the soap to the mouth, regardless of whether he had grasped it with the right or the left hand.

Parakinesia

One year before Liepmann's seminal first paper on apraxia, the Belgian neurologist David de Buck published a paper on "parakinesies" (de Buck, 1899). It started with a description of "synkinesies," that is, involuntary movements of one body part accompanying either deliberate or reflex movement of other body parts. This phenomenon is quite common in hemiplegic patients. For example, the paralyzed arm raises involuntarily when the sound arm is raised or when the patient yawns or sneezes. The major part of the paper, however, was devoted to a more exceptional observation which de Buck classified as an instance of "parakinesia." He presented the case of a 40-year-old woman who, half a day after her fifth childbirth, suddenly had the sensation that "something wanted to get out of her genitals." She retained no memory of the subsequent events but her family reported that she fell into a coma which lasted for three weeks. During that time she was nursed by her family, and de Buck remarks that he could not obtain medical reports because she was rarely, if at all, seen by medical doctors. The coma cannot have been complete as she must have been capable of swallowing food and fluid.

When she awoke, her whole body was flaccidly paralyzed and insensitive to pain, and she did not speak. Within two months mobility and speech returned but they remained abnormal. Speech was fluent and well articulated but distorted by repetitions of letters, syllables, and sometimes whole words, and she did not always find the right word to express her ideas. The most remarkable symptoms concerned the posture and mobility of her body and her limbs. De Buck described her appearance:

> Stout woman with the appearance of good physical health. But what's striking the examiner is her posture. Regardless of whether she is sitting or standing, she never displays the usual posture of rest with both upper limbs hanging down or resting on the knees. They are in demi-flexion crossed one over the other before her chest. Her gaze is directed downwards and her face expresses melancholy. The posture frequently has a cataleptic appearance. (de Buck, 1899, p. 366)

She seemed to be unable to perform even the simplest actions and replaced them by rather bizarre movements which were performed with much apparent effort. For example:

> When asked to raise her right arm, she makes energetic efforts, the right hand crosses the trunk and is placed in the left armpit and the left hand extends backwards. Then the left hand tugs with effort her skirts. (de Buck, 1899, p. 366)

Generally, her movements deteriorated when she was asked to pay attention to them. The dependence of the severity of disturbance from the context of examination went even further:

> when she believes that she is not observed but left on her own in solitude, she executes swiftly quite complex movements like scratching her face or the hair. If she is asked shortly afterwards to execute the same movements, she is totally unable to make them but replaces them by series of substitute movements. (de Buck, 1899, p. 372)

Clinical wisdom says that a disorder which vanishes when the patient believes they are unobserved does not, or at least not only, have an organic cause. But rather than embarking on a psychiatric diagnosis, de Buck proposed an explanation in terms of motor control. He postulated that:

> our patient has the idea of her actions, but does not arrive at evoking the corresponding kinetic images. There is a rupture between the centres of movement and the area of ideation . . . The perturbation that gives rise to the parakinesie takes place in the transmission from the mental sphere to the sphere of motor images. (de Buck, 1899, p. 373)

This explanation leaves open the question why the transition from the idea to the execution of voluntary actions depends on whether the patient believes herself to be observed. Nonetheless, it merits David de Buck's admission into the hall of fame of apraxia. He had arrived at almost exactly the same ideas, and used nearly identical formulations for describing them, as Liepmann in his seminal first paper on apraxia which appeared only one year later.

Chapter 2

Hugo Karl Liepmann

Since the beginning of the twentieth century, clinical diagnosis and research of apraxia has been dominated by the writings of the German neuropsychiatrist Hugo Karl Liepmann (Goldenberg, 2003a) (Figure 2.1). Liepmann was born in Berlin in 1863, the son of a cultured and wealthy Jewish family.[1] He studied philosophy and acquired a PhD with a thesis on the mechanism of Leukipp–Democrit's atoms, but then entered university again to study medicine and acquired his MD in 1895. In the same year he started as an assistant to Wernicke in Breslau where he stayed until 1899. He then returned to Berlin and took a post at the municipal welfare for the mentally ill. He was first assistant, then consultant in the psychiatric hospital of Dalldorf (known today as the Karl Bonhoeffer Psychiatric Clinic, Berlin-Lichtenau) and from 1915 director of the psychiatric hospital in Herzberge (today Berlin-Lichtenberg).

The imperial counselor

In Liepmann's times, a substantial portion of patients in psychiatric hospitals were afflicted by dementia caused by general paralysis of the insane, a late manifestation of syphilis infection. On February 10, 1900, one such patient was admitted to the psychiatric hospital of Dalldorf where Liepmann had only recently started to work. The patient was a 48-year-old engineer who had worked in the imperial patent office. Since he was employed as an official he was qualified to bear the proud title of an "imperial counselor," but he had almost certainly never given advice to the German Emperor. He had acquired syphilis some ten years ago. His present illness had begun suddenly two months ago with a state of confusion accompanied by aphasia and agitated depression.

Liepmann described his first encounter with the patient, one week after his admission:

> I saw the patient for the first time on February 17. He was asked to point to certain objects and to carry out certain hand movements. He failed in almost everything, handling objects quite absurdly. At first sight it appeared as if the patient did not understand—that he was cortically deaf, possibly also cortically blind. However, I noted certain bizarre and distorted movements which he made during the course of the examination; they were confined to the right upper extremity which the patient used exclusively during the period of observation. This peculiar motor behaviour made me wonder if his incorrect responses reflected a basic lack of comprehension or, rather, faulty

[1] A substantial portion of the German clinicians and scientists contributing to the early development of apraxia were Jews, although partly converted to the Christian religion. I have indicated only their civic nationality because I think that this corresponds better to their own attitude versus religion and nation.

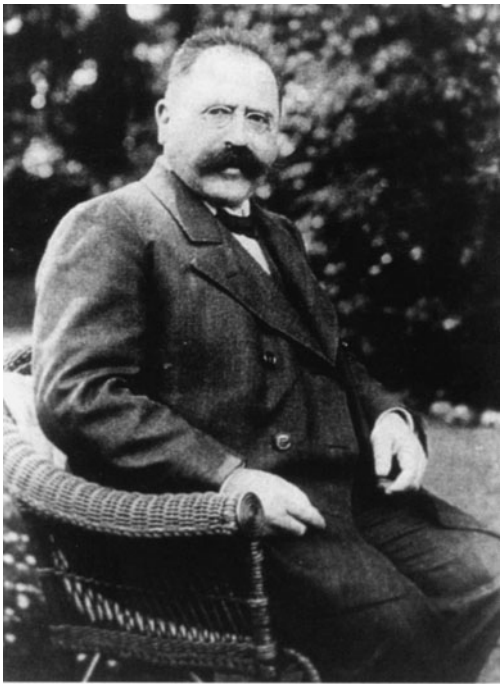

Figure 2.1 Hugo Karl Liepmann.

motor execution . . . To resolve this question I held on to the patient's right hand and forced him to use his left hand. Now, all of a sudden, the picture changed. With his left hand he immediately selected the card that was asked for from among five cards laid out in front of him. The same test repeated with the right hand led in general to faulty responses. I then established that the situation was the same as regards his lower extremities. The patient could imitate movements of my foot with his left foot but failed altogether with his right foot. Thus, it was established that the patient had neither word-deafness nor mind-blindness. Reproduced from Liepmann, H., Das Krankheitsbild der Apraxie (motorische Asymbolie) auf Grund eines Falles von einseitiger Apraxie. Monatschrift für Psychiatrie und Neurologie, 8, p. 19 © 1900, Karger, with permission.

This quote is from the fourth page of the first paper devoted to the "imperial counselor." Others were to follow. In the end, Liepmann's reports of this single case had given rise to 137 printed pages distributed on a tripartite paper (Liepmann, 1900) and another two-part paper (Liepmann, 1905a, 1906) reporting the subsequent clinical course, death, and post-mortem of the patient.

I will try to briefly summarize the main findings of Liepmann's extensive and methodically ingenious examinations:

Spontaneous speech was restricted to a small repertoire of exclamations like "yes" "oh God," "no," and he was unable to repeat any words. Comprehension of spoken commands varied according to which body part was addressed. He promptly followed commands for moving the whole body like standing up or going to a window, but was unable to follow commands for even simple movements of the head, the mouth, or the tongue. Whereas verbal commands were executed promptly using the left hand and named objects could be selected from an array, the right hand acted as if he could not understand anything at all.

The same pattern of preserved and disturbed compliance applied to written commands and also to imitation: he correctly imitated movements with the left leg and the left hand, but not with the right limbs, the head, mouth, and tongue.

Writing to dictation with the right hand produced a regular sequence of up and down strokes interspersed with single recognizable letters which had no relationship to the target word. On first sight, his left-handed writing looked completely aberrant, but on close inspection it turned out that the left hand produced mirror writing. The letters were drawn awkwardly and irregularly, and some of them were completely wrong, but the intended words were always recognizable. The left hand could also compose words out of single anagram letters whereas this task gave rise only to meaningless letter sequences when tried by the right hand.

When he was asked to point blindfolded with one hand to the location where the other hand had been touched, he failed in both directions though with somewhat different kinds of errors. The right hand did not follow the command at all, whereas the left hand tried to comply but made gross spatial errors. These errors were particularly impressive when single fingers of the right hand had been pricked. Then the left hand would search for the location of the prick on the forearm, while the right hand made small movements with the pricked finger as if it wanted to help the other hand finding the right place.

Unimanual use of objects was normal with the left hand. By contrast, the right hand committed impressive errors. For example, when given a comb the right hand stuck it behind the ear like a pen. The right hand used a toothbrush like a pen on one occasion and put the handle into the mouth on another. On a third trial the right hand took the toothbrush like a spoon, shoveled with it, and finally put it into the mouth.

Generally right-hand performance was better when the required actions were embedded in their natural context rather than being asked for in an examination, and there were a few activities in which the right hand nearly always succeeded. One such activity was smoking a cigar and another, that Liepmann considered particularly important for his interpretation of the imperial counselor's apraxia, was buttoning:

> The patient is always capable to button and unbutton. However, this happens virtually never after a first request, and sometimes much exhortation is necessary to get him to begin the action. But once the fingers have touched the button, the remainder of the action is performed with considerable deftness, even when the eyes are closed. (Liepmann, 1900, p. 32)

Bimanual coordination varied across different tasks. Seated before a piano, he placed both hands correctly and played simple recognizable melodies, though with errors. By contrast, he was unable to cooperate with both hands to spread butter on a slice of bread or to make a knot in a scarf. Sometimes the right hand interfered with unimanual left-hand activities:

> He is asked to pour water from a jug into a glass. The left hand takes the jug and wants to pour, but at the same time the right hand leads the empty glass to the mouth. If one holds onto the glass and thus enables the left hand to pour, it succeeds without further problems. (Liepmann, 1900, p. 35)

Figure 2.2 shows a snapshot of this episode and a similar observation made some 80 years later (Goldenberg et al., 1985).