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Citation	Phelps, Scott Douglas. 2014. Blind to Their Blindness: A History of the Denial of Illness. Doctoral dissertation, Harvard University.
Accessed	April 17, 2018 4:56:39 PM EDT
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Blind to Their Blindness: A History of the Denial of Illness

Abstract

For many historians, sociologists, and anthropologists of medicine, “disease” and “illness” are not equivalent. Whereas “disease” denotes the physician’s ostensibly objective criteria, “illness” emphasizes the patient’s subjective experience. This dissertation examines that distinction precisely at a point where it breaks down, in the history of a diagnosis called “anosognosia,” also known as the denial of illness.

In the 1890s, Austrian psychiatrist Gabriel Anton observed a number of patients who denied being blind, deaf, or paralyzed. “That is just how it is,” a patient demurred, “one sees better in youth.” The question to Anton was, were such patients truly unaware of their loss or did they sense it on some level, if only to deny it?

The history of Anton’s syndrome, later named “anosognosia” by the French neurologist Joseph Babinski, spans more than a century and a half across two continents and through both world wars. I treat its history as a special type of lens to focus on some of the broader intellectual and professional differences between neurology and psychiatry. I argue that the clinical perception and portrayal of this apparent loss of the patient’s experience depended on historical patterns of thinking about the distinction between conscious and unconscious perception as well as categories of health and disease. To think about the denial of illness was to think about the very meaning of illness and awareness, and the difference, if any, between the mind and the brain.

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Acknowledgements

Thank you. There is no way to say it adequately. There is no way to convey the kind of gratitude I feel for those whom I am about to “acknowledge” in these ephemeral pages.

To Joanna, first and foremost, we made it! You have been there for me in ways I did not know another person could be present for another. For years and years on end, sometimes seemingly to no end, you patiently waited for this project to take shape and come out from under my skin into the light of day. Thank you for your quiet faith. And thank you for reminding me there are always more adventures waiting around the corner.

To my primary advisor, Anne Harrington, who never turned her back on me, even if I was lost on a train to Wales, even despite shoddy internet connections in Berlin, and even when the difference in time zones was more than a little inconvenient, I thank you. You introduced me to the history of science. You kept me focused and more than once saved me from my own tangle of thoughts. It has been an honor calling myself one of your students. And it will always be. To my advisor, Charles Rosenberg, thank you for making yourself consistently available to edit my writing and help me incubate my ideas. I hope it is already obvious by now how much my project was inspired by your vast scholarship. To my advisor, Hisa Kuriyama, I am fortunate that our paths crossed in Paris four years ago. You offered me a kind of distraction of the highest order with all the wonderful books and films you recommended over the years. Your sense of play and depth of imagination breathed new life into my graduate experience and helped me keep not only my head but also my heart in the game.

To Philipp Lehmann, thank you for all your help over the years with my translations of arcane German medical jargon and your encouragement through many early drafts. To Andrew Parker, thank you for giving me the chance to fly to far-flung places each year and prodding me to keep up my French. To Beau Bellenfant, thank you for all the afternoon porch chats and helping me stay grounded. To David Smith, thank you for being my first friend in Cambridge and all the late talks. To Justin Valentine, thank you for keeping me in shape. To Greg Phelps, I am lucky to have always had such a friend and mentor in a big brother from day one.

I want to thank a number of folks at Harvard for being patient listeners and generously offering their time: Steve Shapin, Rebecca Lemov, David Jones, Sean Kelly, Katja Guenther (especially for sharing the copies of Schilder’s papers and reading an early draft of my first chapter), Chris Phillips, Jeremy Blatter, Peter Gordon, Jimena Canales, Stefan Sperling, Steve Hyman, and Nasser Zakariya. Thanks also for the support from Robert Martensen (especially in the early stages of my prospectus), Constantin Tranulis (pour les randonnées), Claude and Armelle Debru (pour un séjour très mémorable à Paris), Alena and Frank Lehmann (meine liebe deutsche Eltern), Romain and Pauline Schneckenburger, Jacob Rooksby, and Steven Benyas. I am grateful to the Beinecke Foundation, Rotary Foundation, and the National Science Foundation for generously funding my research.

Finally, to my parents, Doug and Peggy Phelps, *this one’s for y’all*.

Introduction

Blind to Their Blindness: A History of the Denial of Illness

“[N]o one is healthy who knows that he is so.”¹

In 1899, the year that Freud finished *The Interpretation of Dreams*, Austrian psychiatrist Gabriel Anton described three patients with a very peculiar type of behavior. All three had suffered a form of brain disease, and as a result, two were left completely deaf and the other blind. When the first patient checked into Anton's clinic, she complained of headaches and back pain. Within a month, she completely lost her vision. And yet, she never seemed to realize it. True, she admitted, her eyesight was not as good as it used to be. “That is just how it is,” she demurred, “one sees better in youth.” But she denied being *blind*. According to Anton, such patients lacked not only their eyesight but also “illness-insight.” They were, he wrote, “mentally blind to their blindness.”²

A decade and a half later, in the summer of 1914, French neurologist Joseph Babinski reported to the *Société de Neurologie* in Paris two patients with the most remarkable “mental trouble.” After suffering what appeared to have been a stroke, they were paralyzed on the left side of their body. Otherwise, they still recognized their

¹ Georges Canguilhem, *The Normal and the Pathological* (New York: Zone Books, 1991), 243.

² Gabriel Anton, “Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit,” *Archiven für Psychiatrie und Nervenkrankheiten* 32 (1899): 86–127; Gabriel Anton, “Blindheit nach beiderseitiger Gehirnerkrankung mit Verlust der Orientierung im Raume,” *Mittheilungen des Vereines der Aerzte in Steiermark* 33, no. 3 (January 27, 1896): 41–46; Gabriel Anton, “Beiträge zu klinischen Beurtheilung und zur Localisation der Muskelsinnstörungen im Grosshirne,” *Zeitschrift für Heilkunde* 14 (1893): 317–319, 323–325.

friends and family and could carry on normal conversations. There was just one glaring problem. Whenever Babinski asked them to try to raise the paralyzed arm, they completely ignored him. After repeated requests, one patient finally replied, “There, it’s done,” although the arm lay motionless by her side.³

By most accounts, this marked the beginning of the history of “anosognosia,”⁴ known also as the Anton-Babinski syndrome.⁵ Strictly speaking, it is true. Anton was the first doctor to describe the loss of “self-perception of focal brain disease” as a discrete disorder. And Babinski was the first to use the term “*anosognosie*” from the ancient Greek, meaning the “*lack of awareness of illness.*”⁶ But there is more to its history.⁷

³ Joseph Babinski, “Contribution à l’étude des troubles mentaux dans l’hémiplégie organique (anosognosie),” *Revue neurologique* 27 (1914): 845–848.

⁴ Pronounced *ă-nō’sog-nō’sē-ă*.

⁵ George P. Prigatano, “Historical Observations Relevant to the Study of Anosognosia,” in *The Study of Anosognosia*, ed. George P. Prigatano (Oxford: Oxford University Press, 2010); Ivana Marková, *Insight in Psychiatry* (Cambridge University Press, 2005); Constanza Papagno and Giuseppe Vallar, “Anosognosia for Left Hemiplegia: Babinski’s (1914) Cases,” in *Classic Cases in Neuropsychology*, ed. Christopher Code, vol. 2, 2 vols. (Hove, East Sussex: Psychology Press, 2001), 171–90; Emer M.E. Forde and Claus-W. Wallesch, “‘Mind-Blind for Blindness’: A Psychological Review of Anton’s Syndrome,” in *Classic Cases in Neuropsychology*, ed. Christopher Code, vol. 2, 2 vols. (Hove, East Sussex: Psychology Press, 2001), 199–222; George P. Prigatano and Daniel L. Schacter, *Awareness of Deficit after Brain Injury: Clinical and Theoretical Issues* (Oxford: Oxford University Press, 1991); Edwin A. Weinstein, “Anosognosia and Denial of Illness,” in *Awareness of Deficit after Brain Injury: Clinical and Theoretical Issues.*, ed. George P. Prigatano and Daniel L. Schacter (Oxford: Oxford University Press, 1991), 240–57; Xavier Francisco Amador and Anthony S. David, *Insight and Psychosis: Awareness of Illness in Schizophrenia and Related Disorders* (Oxford: Oxford University Press, 2004).

⁶ “a” = *lack*; “nosos” = *disease*; “gnosis” = *knowledge*.

⁷ The few neurologists, psychiatrists, and psychologists who have recently written brief histories of anosognosia provide very useful bibliographies, but all too often their accounts are skewed to anticipating the “right” and “wrong” theories according to contemporary consensus. Generally speaking, they fail to consider the larger history and the social and philosophical implications that arise from the study of such “borderland disorders” as anosognosia. For the earliest references to “borderland disorders,” see Charles D. Aring, “Observations on Multiple Sclerosis and Conversion Hysteria,” *Brain* 88, no. 4 (1965): 663–74; Z. J. Lipowski and R. Z. Kiriakos, “Borderlands between Neurology and Psychiatry: Observations in a Neurological Hospital,” *The International Journal of Psychiatry in Medicine* 3, no. 2 (1972): 131–47; Norman Geschwind, “The Borderland of Neurology and Psychiatry: Some Common Misconceptions,” in

My dissertation begins in the late 1860s in Austria and France and closes in the early 1980s in the United States. Spanning more than a century, over two continents and two world wars, the history of anosognosia wends its way through a series of important cultural debates, at once medical and philosophical. Some pertain to the organization of clinical knowledge and practice, particularly around the specialties we now call neurology and psychiatry. Others concern metaphysical questions about the nature of consciousness, the limits of perception, and the definition of illness. Over several decades, these debates helped to shape the identity of the denial of illness. In turn, the study of the denial of illness helped shape these larger debates, supplying them with new ideas and new reason to reflect on the boundaries between neurology and psychiatry, sickness and health, and ultimately, the body and soul.⁸

I treat the history of this diagnosis, therefore, as a special lens. On one level, I use it to bring into better focus the social, professional history of neurology and psychiatry.⁹ I show how it straddled their borders as well as helped to re-articulate

Psychiatric Aspects of Neurologic Disease, ed. Dietrich Blumer and D. Frank Benson, vol. 1 (New York: Grune & Stratton, 1975), 1–8.

⁸ As I hope to make clear, I do not submit that the shifting historical boundaries between the specialties we call today "neurology" and "psychiatry" always mirrored the historical divisions between the brain and mind, or between the nosological and etiological distinctions between organic brain disease and mental illness.

⁹ There are, as of yet, few comprehensive histories of neurology and psychiatry, particularly which critically reflect on the instability of their ostensibly individual identities. For some traces of this historiographical discussion, see Jack D. Pressman, *Last Resort: Psychosurgery and the Limits of Medicine* (Cambridge University Press, 2002); N. Tomes, "Beyond the 'Two Psychiatries': Jack Pressman's Last Resort and the History of Twentieth-Century American Psychiatry: Introduction," *Bulletin of the History of Medicine* 74, no. 4 (2000): 773–77; Eric J. Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice* (Cornell University Press, 2003); E. J. Engstrom and V. Roelcke, *Psychiatrie im 19. Jahrhundert: Forschungen zur Geschichte von psychiatrischen Institutionen, Debatten und Praktiken im deutschen Sprachraum*, vol. 13 (Schwabe, 2003); Christopher G. Goetz, Michel Bonduelle, and Toby Gelfand, *Charcot: Constructing Neurology* (Oxford: Oxford University Press, 1995); D. Gavirus, "Men of

them. On another level, I use the history of anosognosia to hone the philosophic, intellectual history of ideas about perception and illness. I show how it upset conventional definitions of health and precipitated new understanding of the patient experience.¹⁰ My aim is to elaborate these connections between the social history of

Dreams and Men of Action: Neurologists, Neurosurgeons, and the Performance of Professional Identity, 1920-1950," *Bulletin of the History of Medicine* 85, no. 1 (2011): 57–92; Charles E. Rosenberg, "The Crisis in Psychiatric Legitimacy: Reflections on Psychiatry, Medicine, and Public Policy," ed. George Kriegman, Robert D. Gardner, and D. Wilfred Abse, *American Psychiatry, Past, Present, and Future: Papers Presented on the Occasion of the 200th Anniversary of the Establishment of the First State-Supported Mental Hospital in America*, 1975, 135–48; Heinz Schott and Rainer Tölle, *Geschichte der Psychiatrie: Krankheitslehren, Irrwege, Behandlungsformen* (München: C.H. Beck, 2006). Hess, Volker, and Benoît Majerus. "Writing the History of Psychiatry in the 20th Century." *History of Psychiatry* 22, no. 2 (2011): 139–45.

¹⁰ The historiography of the so-called "patient's view" is now quite extensive, but only more recently have medical historians begun to broach the conflict between the attempted reconstructions of historical patient experience and the skeptics' deconstructions of it. For recent critical reflections on the problems of writing about historical patient experience and, correlatively, historical ideas of health, see Michael Stolberg, *Experiencing Illness and the Sick Body in Early Modern Europe* (Basingstoke, Hampshire: Palgrave Macmillan, 2011); Flurin Condrau, "The Patient's View Meets the Clinical Gaze," *Social History of Medicine* 20, no. 3 (December 1, 2007): 525–540; Roger Cooter, "Neuropatients in Historyland," *The Neurological Patient in History* 20 (2012): 215–22; Roger Cooter, "Re-Presenting the Future of Medicine's Past: Towards a Politics of Survival," *Medical History* 55, no. 03 (2011): 289–94; Ian Hacking, "Making Up People," in *Reconstructing Individualism*, ed. T. Heller (Stanford, CA: Stanford University Press, 1986), 222–36; N.D. Jewson, "The Disappearance of the Sick-Man from Medical Cosmology, 1770-1870," *Sociology* 10, no. 2 (1976): 225; Charles E. Rosenberg, "The Therapeutic Revolution: Medicine, Meaning and Social Change in Nineteenth-Century America," *Perspectives in Biology and Medicine* 20, no. 4 (1977): 485–506; David Armstrong, "The Patient's View," *Social Science & Medicine* 18, no. 9 (1984): 737–44; Roy Porter, "The Patient's View: Doing Medical History from below," *Theory and Society* 14, no. 2 (March 1, 1985): 175–98; G.B. Risse and J.H. Warner, "Reconstructing Clinical Activities: Patient Records in Medical History," *Social History of Medicine* 5, no. 2 (1992): 183; Jonathan Metzl and Anna Rutherford Kirkland, eds., *Against Health: How Health Became the New Morality* (New York: New York University Press, 2010); Ian Robert Dowbiggin, *The Quest for Mental Health: A Tale of Science, Medicine, Scandal, Sorrow, and Mass Society*, Cambridge Essential Histories (New York: Cambridge University Press, 2011); Joseph Dumit, *Drugs for Life: How Pharmaceutical Companies Define Our Health*, Experimental Futures (Durham, NC: Duke University Press, 2012); H. G. Gadamer, *The Enigma of Health: The Art of Healing in a Scientific Age*, trans. N. Walker and J. Gaiger (Stanford, CA: Stanford University Press, 1996); P. Laín Entralgo, "Being Healthy Down Through History," *Sudhoffs Archiv; Zeitschrift für Wissenschaftsgeschichte. Beihefte*, no. 24 (1984): 65–72; G. Danzer et al., "On the Theory of Individual Health," *Journal of Medical Ethics* 28, no. 1 (February 1, 2002): 17–19; C. R. Burns, "Diseases versus Healths: Some Legacies in the Philosophies of Modern Medical Science," *Evaluation and Explanation in the Biomedical Sciences*, 1975, 29–47.

medicine and the intellectual history of perceptual experience with a focus on theories of anosognosia.¹¹

That is not to say the diagnosis was pervasive. The opposite comes closer to the truth. But it represented a critical anomaly in the history of medical thought.¹² I argue

¹¹ This method has many precedents. In particular, I have found the scholarship of Ian Hacking, Jan Goldstein, Stephen Jacyna, and David Healy helpful models for navigating my own use of a diagnostic entity to draw into focus larger professional and philosophical questions in the history of medicine. David Healy, *Mania: A Short History of Bipolar Disorder*, 1st ed. (The Johns Hopkins University Press, 2008); Charles E. Rosenberg, "Introduction: The History of Our Present Complaint," in *Our Present Complaint: American Medicine, Then and Now* (Baltimore: Johns Hopkins University Press, 2007), 1–11; Charles E. Rosenberg, "Contested Boundaries: Psychiatry, Disease, and Diagnosis," *Perspectives in Biology and Medicine* 49, no. 3 (2006): 407–24; Jan Goldstein, *Console and Classify: The French Psychiatric Profession in the Nineteenth Century: With a New Afterword*, 2nd ed. (Cambridge: Cambridge University Press, 1987; Chicago: University of Chicago Press, 2001); Pressman, *Last Resort*; Ian Hacking, *Mad Travelers: Reflections on the Reality of Transient Mental Illnesses* (Harvard University Press, 2002); L. S. Jacyna, *Lost Words: Narratives of Language and the Brain, 1825-1926* (Princeton University Press, 2000); Allan Young, *The Harmony of Illusions* (Princeton, NJ: Princeton University Press, 1997); Ian Hacking, *Rewriting the Soul: Multiple Personality and the Sciences of Memory* (Princeton, NJ: Princeton University Press, 1995); Mark S. Micale, *Approaching Hysteria: Disease and Its Interpretations* (Princeton: Princeton University Press, 1995); Janet Oppenheim, *"Shattered Nerves": Doctors, Patients, and Depression in Victorian England* (New York: Oxford University Press, 1991); B. Sicherman, "The Uses of a Diagnosis: Doctors, Patients, and Neurasthenia," *Journal of the History of Medicine and Allied Sciences* 32, no. 1 (1977): 33–54; Charles E. Rosenberg, *The Cholera Years: The United States in 1832, 1849, and 1866* (Chicago: University of Chicago Press, 1962).

¹² On the power of using anomalous cases in the study of history, see especially Carlo Ginzburg, *Threads and Traces: True, False, Fictive* (Berkeley: University of California Press, 2012); Carlo Ginzburg, "Our Words, and Theirs: A Reflection on the Historian's Craft, Today," *Institute for Advanced Study Video Lectures*, October 3, 2011, <http://video.ias.edu/ginzburg>; Angela N.H. Creager, Elizabeth Lunbeck, and M. Norton Wise, *Science without Laws: Model Systems, Cases, Exemplary Narratives* (Duke University Press Books, 2007); John Forrester, "On Kuhn's Case: Psychoanalysis and the Paradigm," *Critical Inquiry* 33 (Summer 2007): 782–819; J. Forrester, "If P, Then What? Thinking in Cases," *History of the Human Sciences* 9, no. 3 (1996): 1–25; Perry Anderson, "The Force of the Anomaly," *London Review of Books*, April 26, 2012; German E. Berrios, "Phenomenology, Psychopathology and Jaspers: A Conceptual History," *History of Psychiatry* 3, no. 11 (September 1992): 303–27; German E. Berrios, *The History of Mental Symptoms: Descriptive Psychopathology Since the Nineteenth Century* (Cambridge: Cambridge University Press, 1996); Roger Smith, *Inhibition: History and Meaning in the Sciences of Mind and Brain* (University of California Press, 1992); Thierry Haustgen and M.-L. Bourgeois, "L'évolution du concept de mythomanie dans l'histoire de la psychiatrie," *Annales médico-psychologiques, revue psychiatrique* 165 (2007): 334–44; Gaston Bachelard, *The Psychoanalysis of Fire*, Second printing, November 1971 edition (Beacon Press, 1987); Georges Canguilhem, "La santé: concept vulgaire et question philosophique," *Cahiers du Séminaire de Philosophie*, no. 8 (1988): 119–33; Georges Canguilhem, *Essai sur quelques problèmes concernant le normal et le pathologique*, Publications de la faculté des lettres de l'université de Strasbourg, fasc. 100 (Clermont-Ferrand: La Montagne, 1943); Georges Canguilhem, *Études d'histoire et de philosophie des sciences*, 2. éd (Paris: Librairie Philosophique J. Vrin, 1970).

that it was never just a clinical diagnosis but a critical concept that could be wielded to delineate or dissolve the boundaries between neurology and psychiatry and to re-define the meaning of health and self-awareness.¹³ As such, its conceptual coherence relied on certain philosophical arguments about the relationship between the mind and body. It also depended on the intelligibility of conventional categories used to describe human behavior, such as “conscious” or “unconscious” and “normal” or “pathological.” In turn, it often helped destabilize these very categories.¹⁴

No one could say with certainty whether the patient was truly unaware of illness, indeed, what “unawareness” really entailed. Although Anton and Babinski initially believed it resulted strictly from an organic brain disease, other doctors later described it in terms of “unconscious urges” and “existential threats.”¹⁵ Still others argued the

¹³ There are other medical anomalies with arguably similar cachet. For the sake of anomaly alone, I might have chosen instead to write a history of depression, which is far more prevalent and similarly upsets the supposed division between mental illness and brain disease, and between behavioral norms of society and scientific facts of chemistry. Alternatively, I might have chosen more specific, “neuropsychiatric” disorders of consciousness, such as “prosopagnosia” or “Capgras’ syndrome.” They, too, capture the problems inherent in discriminating apparent motivated avoidance and cognitive-affective deficits. However, only anosognosia reflexively threatens its own intelligibility as an illness that entails never being known, not by the patient. In that sense, hypochondria approximates this paradoxical character from the other end. It, too, would seem to undermine its own plausibility. But the diagnosis of hypochondria seems rather to be self-confirming, whereas anosognosia tends toward the self-negating. Anosognosia denotes the illness that exists only by virtue of never being suffered or experienced as such. It appears only on the basis of a putative loss of awareness, or the kind of subjective loss which can only be inferred by those looking on but unable really to “look in.” In this way, above all, it joins together, definitionally, the ultimate questions of what really is “normal” behavior in response to personal illness and what really is the absolute “absence of mind” or loss of consciousness.

¹⁴ For similar studies of a destabilizing concept, see Smith, *Inhibition*; Canguilhem, *The Normal and the Pathological*; Maurice Merleau-Ponty, *The Structure of Behavior*, trans. Alden L. Fisher (Boston: Beacon Press, 1963).

¹⁵ Kurt Goldstein, *Der Aufbau des Organismus. Einführung in die Biologie unter besonderer Berücksichtigung der Erfahrungen am kranken Menschen* (Haag: M. Nijhoff, 1934); Paul Schilder, *The Image and Appearance of the Human Body: Studies in the Constructive Energies of the Psyche* (London: K. Paul, Trench, Trubner & co. Ltd, 1935). See Chapter 3 for further discussion.

denial of illness was caused by “interpersonal factors” and “psychic drives.”¹⁶ Patients were “in denial” about their painful situation, but on some level they recognized it enough to “repress” it.¹⁷ Every theory of anosognosia, therefore, relied on different interpretations of behavior and definitions of the mind. Moreover, each could be manipulated to challenge and revise other interpretations and definitions of mind and behavior.¹⁸

An essential question also remained as to whether anosognosia was actually pathological or whether it was a normal reaction. Some doctors argued that it was not so much a disorder in itself but a way of adapting to and dealing with disorder. They also urged for a more “holistic” view of medicine, one which did not rush to the assignment of disease without taking into account the individual's need to find personal meaning in his illness. In this respect, the re-interpretation of anosognosia helped blur the

¹⁶ Edwin A. Weinstein and Robert L. Kahn, *Denial of Illness: Symbolic and Physiological Aspects* (Springfield, Ill: Thomas, 1955). See Chapter 4 for more discussion.

¹⁷ Language is especially important here. I have chosen predominantly to use the term “denial of illness,” although the word denial is trailed with ambiguity, particularly thanks to Freud's school. On the one hand, denial refers generally to a speech act without necessary premeditation, as in one who merely disagrees with another's verbal account. On the other hand, it implies concealment and guilt, as one who is “in denial” of an offense he has committed. Indeed, some doctors described their effort to persuade patients about their illness in terms of “accusations” and the patients' acknowledgement as “confessions.” See Emil Redlich and Guilio Bonvicini, “Über mangelnde Wahrnehmung (Autoanästhesie) der Blindheit bei cerebralen Erkrankungen,” *Neurologisches Centralblatt* 29 (1907): 945–51; Ludwik Fleck, *Genesis and Development of a Scientific Fact* (1934; repr., Chicago: University of Chicago Press, 1979). The emphasis on denial often countered the implication that behavior was determined, involuntary, impersonal, and passive. Even so, it left open room for both interpretations among those who were ambivalent as to whether behavior was biochemically determined or dynamically motivated, or some interactive aggregate. The ambiguity allowed them to continue their research under the ideological radar, deflecting suspicions of partisanship, and preserving multiple, if at times paradoxical, intellectual and professional affiliations (as we will see in Chapter 3).

¹⁸ “Whoever controls the definition of mind controls the definition of humankind itself, and culture, and history.” Marilynne Robinson, *Absence of Mind: The Dispelling of Inwardness from the Modern Myth of the Self* (New Haven: Yale University Press, 2010), 32.

categories of health and illness, similar to the way it blurred the distinction between consciousness and the unconscious.¹⁹ Ultimately, it denoted a deeply ambiguous behavior. It looked as if it encompassed both motives and mechanisms, personality and brain, hovering somewhere between the normal and the pathological.

Thus, it is difficult to chart the history of a diagnosis so unstable as the denial of illness. To propose it even has a history presupposes that it was actually static enough to call it "it." But can we honestly know this about the history of anosognosia?²⁰ Can

¹⁹ On the history of self-styled "holistic" medicine, see Anne Harrington, *Reenchanted Science: Holism in German Culture from Wilhelm II to Hitler* (Princeton University Press, 1996); Anne Harrington, "A Feeling for the Whole: The Holistic Reaction in Neurology from the Fin de Siècle to the Interwar Years," in *Fin de Siècle and Its Legacy*, ed. Mikuláš Teich and Roy Porter (Cambridge: Cambridge University Press, 1990), 254–78; Christopher Lawrence and George Weisz, *Greater than the Parts: Holism in Biomedicine, 1920–1950*, 1st ed. (Oxford: Oxford University Press, 1998); Mitchell G. Ash, "Weimar Psychology: Holistic Visions and Trained Intuition," in *Weimar Thought: A Contested Legacy*, ed. Peter E. Gordon and John P. McCormick (Princeton University Press, 2013), 35–54; Kurt Goldstein, *The Organism: A Holistic Approach to Biology Derived from Pathological Data in Man* (New York: Zone Books, 1995); M. Hau, "The Holistic Gaze in German Medicine, 1890–1930," *Bulletin of the History of Medicine* 74, no. 3 (2000): 495–524; Charles E. Rosenberg, "The Tyranny of Diagnosis: Specific Entities and Individual Experience," *The Milbank Quarterly* 80 (2002): 237–60; Arthur Kleinman, *Patients and Healers in the Context of Culture: An Exploration of the Borderland between Anthropology, Medicine, and Psychiatry*, Comparative Studies of Health Systems and Medical Care ; No. 3 (Berkeley: University of California Press, 1980); Arthur Kleinman, *The Illness Narratives: Suffering, Healing, And The Human Condition* (New York: Basic Books, 1989).

²⁰ T.E. Feinberg et al., "The Neuroanatomy of Asomatognosia and Somatoparaphrenia," *Journal of Neurology, Neurosurgery & Psychiatry* 81, no. 3 (2010): 276; T. E. Feinberg and D. M. Roane, "Anosognosia, Completion and Confabulation: The Neutral-Personal Dichotomy," *Neurocase* 3, no. 1 (1997): 73–85; K. M. Heilman, R. T. Watson, and E. Valenstein, "Neglect and Related Disorders," *Clinical Neuropsychology* 3 (1993): 279–336; Edoardo Bisiach and Anna Berti, "Dyschiria: An Attempt at Its Systemic Explanation," in *Neurophysiological and Neuropsychological Aspects of Spatial Neglect*, ed. Marc Jeannerod, vol. 45, *Advances in Psychology* (Amsterdam: North-Holland, 1987); Edoardo Bisiach, Claudio Luzzatti, and Daniela Perani, "Unilateral Neglect, Representational Schema and Consciousness.," *Brain: A Journal of Neurology* 102, no. 3 (1979): 609–18; R.P. Friedland and E.A. Weinstein, "Hemi-Inattention and Hemisphere Specialization: Introduction and Historical Review," *Advances in Neurology* 18 (1977): 1–31; A. Heilporn, "Reflections on the Consciousness of Disability and Somatognosia in Cases of Acute Spinal Injuries," *Paraplegia* 6 (1968); Edwin A. Weinstein, Robert L. Kahn, and Walter H. Slote, "Withdrawal, Inattention, and Pain Asymbolia," *Archives of Neurology & Psychiatry* 74 (1955): 235–48; Macdonald Critchley, *The Parietal Lobes* (London: E. Arnold, 1953); Morris B Bender, *Disorders in Perception; with Particular Reference to the Phenomena of Extinction and Displacement* (Springfield, Ill: Thomas, 1952); Josef Gerstmann, "Problem of Imperception of Disease and of Impaired Body Territories with Organic Lesions: Relation to Body Scheme and Its Disorders," *Archives of Neurology and Psychiatry* 48, no. 6 (December 1, 1942): 890–913; Hermann Zingerle, "Über Störungen der Wahrnehmung des eigenen

anyone say for certain it is real?²¹ In fact, that was Babinski's question a hundred years ago. He tentatively affirmed it was.²² But my question is different. I mean, is it real enough to call it "it," to render as an *historical* object? I think so, but it was *made* real because it was applied by real doctors to real patients. It was real because it was used, modified, and disputed. What I mean by "it" is the diagnostic entity, anosognosia, which include the language, people, and practices that helped formulate it and lend it power.²³

Körpers bei organischen Gehirnerkrankungen," *Monatsschrift für Psychiatrie und Neurologie* 34 (1913): 13–36.

²¹ Mark Micale provides an excellent perspective on the difficulty of trying to chronicle the history of a volatile and elusive diagnosis such as hysteria. "In short, creating a remote and historical heritage for a disease entity...implies the universality of the disorder, establishes the validity of the diagnostic category, and bolsters the scientific status of psychiatric medicine itself." Micale, *Approaching Hysteria*, 46; Charles E. Rosenberg, "Framing Disease: Illness, Society, and History (Introduction)," in *Framing Disease: Studies in Cultural History*, ed. Charles E. Rosenberg and Janet Golden (Rutgers University Press, 1992), xiii–xxvi; Randall M Packard, *The Making of a Tropical Disease: A Short History of Malaria*, Johns Hopkins Biographies of Disease (Baltimore: Johns Hopkins University Press, 2007); Healy, *Mania*.

²² See Chapter 2 for further discussion. Babinski believed that the "reality" of anosognosia entailed both the question of aetiological specificity and the patient's sincerity. For him, it was "real" because it was a specific kind of brain disease. Many doctors today agree. V.S. Ramachandran, *The Tell-Tale Brain: A Neuroscientist's Quest for What Makes Us Human* (WW Norton & Co Inc, 2011); George P. Prigatano, *Advances in the Study of Anosognosia* (Oxford: Oxford University Press, 2010); E. Fuller Torrey, *The Insanity Offense: How America's Failure to Treat the Seriously Mentally Ill Endangers Its Citizens* (W. W. Norton & Company, 2008); Anthony J Marcel, Richard Tegnér, and Ian Nimmo-Smith, "Anosognosia for Plegia: Specificity, Extension, Partiality and Disunity of Bodily Unawareness," *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior* 40, no. 1 (February 2004): 19–40; Antonio R. Damasio, *The Feeling of What Happens: Body and Emotion in the Making of Consciousness* (Houghton Mifflin Harcourt, 2000); V.S. Ramachandran and S. Blakeslee, *Phantoms in the Brain: Probing the Mysteries of the Human Mind* (Harper Perennial, 1999); K. M. Heilman, A. M. Barrett, and J. C. Adair, "Possible Mechanisms of Anosognosia: A Defect in Self-Awareness.," *Philosophical Transactions of the Royal Society B: Biological Sciences* 353, no. 1377 (November 29, 1998): 1903; Bisiach and Berti, "Dyschiria: An Attempt at Its Systemic Explanation"; Todd E. Feinberg, *From Axons to Identity: Neurological Explorations of the Nature of the Self* (WW Norton & Company, 2009); Todd E. Feinberg and Julian Paul Keenan, *The Lost Self: Pathologies of the Brain and Identity* (Oxford University Press New York, NY, 2005). The question of "reality" remains relevant today in different guises. As prospects grow for new diagnostic methods such as genetic testing and bio-imaging technologies, uncertainty also grows over what to make of the so-called "pre-symptomatic" diseases such methods purport to reveal. While this dissertation will not treat these current problems directly, it gestures at them through the history of anosognosia, which is fundamentally a history of how concepts of illness and experience are defined against the other.

²³ Even if someone wanted to argue that the diagnosis and its network of related diagnoses only existed in the pages of medical journals, not real patients, I would still argue it is real. It is real if for no other reason

Apart from questions whether it really entailed the loss of awareness of illness—even whether it really counted as an illness—my dissertation is more concerned with what we might glean from the history of ideas about it and those particular individuals who studied it. For, as I argued above, its history was never just about the diagnosis alone.²⁴ To think with it required engaging much larger questions about the limits of science and medicine and the shifting definitions of consciousness and health.

Writing the history of a concept like anosognosia means recognizing its fundamental instability and being willing to veer into unexpected places which might seem to be, by definition, where it does not belong.²⁵ To give a better idea of its range and plasticity, I have selected seven cases from over a century and a half of clinical literature. Not all of the following vignettes are traditionally presented by physicians as part of the history of anosognosia, but for reasons I hope to make clear, I think they are.

In 1864, a French psychiatrist described a young woman who was brought to him in a trance-like state of sleep called “catalepsy.” She lay rigid and motionless on her back, her entire body numb. When suddenly she woke from the trance, her legs and

than the fact it is an historical artifact of medical literature. I think it is much more than that, but that it most definitely was and is. Young and Hacking furnish incisive critiques of this question. Young, *The Harmony of Illusions*; Ian Hacking, *The Social Construction of What?* (Cambridge, Mass: Harvard University Press, 1999); Hacking, *Rewriting the Soul: Multiple Personality and the Sciences of Memory*.

²⁴ For an excellent defense of the mutual indispensability of intellectual and institutional histories, see Charles E. Rosenberg, “Toward an Ecology of Knowledge: On Discipline, Context, and History,” ed. Alexandra Oleson and John Voss, *The Organization of Knowledge in Modern America, 1860-1920*, 1979, 440–55.

²⁵ While I have tended toward a historical-philosophical analysis of this illness-concept, principally modeled on the work by Canguilhem, Hacking, and Gadamer, I have also benefited tremendously from the historical-sociological analyses by Rosenberg, Stolberg, and Cooter.

arms remained insensible. Despite every attempt to arouse feeling in them by tickling, pinching, and pricking, she never seemed to notice or care what was done to her body. For some reason, the sensations that coursed through her limbs did not break through to her consciousness. According to the doctor, Charles Lasègue, the cause of her insensitivity was “a sort of laziness.” She was too “distracted,” he claimed, to notice her numbness.²⁶

In the winter of 1894, a fifty-six year-old woman checked herself into a clinic in Austria, complaining of intense headaches and dizziness. She also showed some trouble speaking and remembering words, occasionally substituting them with nonsensical sounds. Within a month, her vision gradually diminished until she was totally blind. And yet, she insisted she could see. According to her doctor, Gabriel Anton, this particular lack of “self-perception” was caused by “focal lesions” in the occipital lobes in the back of the brain.²⁷

In 1914, just weeks before the start of World War I, Joseph Babinski described two patients who had no idea that they were partially paralyzed on the left side of their body. Babinski believed that their loss of awareness had an organic basis, resulting from damage to the right cerebral hemisphere of the brain. Incidentally, the patients' families

²⁶ Charles Lasègue, “De l’anesthésie et de l’ataxie hystériques,” *Archives générales de médecine*, 1864, 385–400; Jan Goldstein, *The Post-Revolutionary Self: Politics and Psyche in France, 1750-1850* (Harvard University Press, 2005); Juan Rigoli, *Lire le délire. Aliénisme, rhétorique et littérature en France au XIXe siècle* (Paris: Fayard, 2001).

²⁷ Anton, “Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit”; Gabriel Anton, “Über Herderkrankungen des Gehirnes, welche vom Patienten selbst nicht wahrgenommen werden,” *Mittheilungen des Vereines der Aerzte in Steiermark* 35 (1898): 10–13; Anton, “Blindheit nach beiderseitiger Gehirnerkrankung mit Verlust der Orientierung im Raume.”

begged him not to try to “cure” them because they considered the unawareness “providential” for protecting their loved one from the painful realization of being paralyzed.²⁸

In the spring of 1924, the Viennese psychiatrist Otto Pötzl reported a case similar to Babinski's, with a slight twist. He described a patient who refused even to *look* in the direction of his paralyzed left side. When Pötzl lifted the limp left arm in front of the man's face, he claimed not to recognize it as his own. “I don't know where it came from,” he said. “It looks so long, so lifeless and dead like a snake.” According to Pötzl, this was not just the result of “focal lesions” in the man's brain. After all, he did not *want* to see. He refused to look. He tried to avoid having to acknowledge his disability. Pötzl suspected there was some unconscious motive at play as well.²⁹

In 1955, American neuropsychiatrist Edwin Weinstein and psychologist Robert Kahn argued that the denial of illness was caused by a combination of neurological and psychological factors. One of their patients was a thirty-eight year-old woman who denied her left arm was paralyzed. “What's the difference!” she exclaimed when she raised her leg instead. “Some people call it an arm; some a leg.” She even denied having had surgery. “It's ridiculous,” she complained, “Why are people bothering me about a nonexistent operation?” According to Weinstein and Kahn, she had always been afraid

²⁸ Babinski, “Contribution à l'étude des troubles mentaux dans l'hémiplégie organique (anosognosie)”; Joseph Babinski, “Anosognosie,” *Revue neurologique* 34, no. 11–12 (1918): 365–67.

²⁹ Otto Pötzl, “Über Störungen der Selbstwahrnehmung bei linkseitiger Hemiplegie,” *Zeitschrift für die gesamte Neurologie und Psychiatrie* 93 (1924): 117–68.

of illness and associated it with the “loss of prestige in the eyes of others.” They argued that she had a “denial personality” that led to her anosognosia.³⁰

In the winter of 1961, neurologist Norman Geschwind and psychologist Edith Kaplan identified the first “human split-brain syndrome.”³¹ One of their patients, who had had a brain tumor removed, was given a hammer. With his eyes closed, he was told to pantomime its function with his left hand. He made the correct hammering motion, but then he said, “I would use this to comb my hair.”³² He had no idea of the disconnect between his words and actions. He had “no insight into his illness.”³³ But his personality had nothing to do with it. According to Geschwind and Kaplan, anosognosia did not entail any personal, instinctual, or existential motives. It was due only to a “callosal lesion,” or focal disease in the corpus callosum which connected the right and left cerebral hemispheres.³⁴

In 1984, Oliver Sacks, a well-known writer and neuropsychologist, wrote about his own experience of illness which he compared to anosognosia. While hiking in Norway, he fell on his left leg, tearing the quadriceps muscles from his knee. After

³⁰ Weinstein and Kahn, *Denial of Illness: Symbolic and Physiological Aspects*.

³¹ Anonymous, “Random Reports: Human Split-Brain Syndromes,” *New England Journal of Medicine* 266, no. 19 (1962): 1013; Norman Geschwind, “Random Reports: Human Split-Brain Syndromes,” in *Selected Papers on Language and the Brain*, Boston Studies in the Philosophy of Science 16 (Netherlands: Springer, 1974), 18–21.

³² Norman Geschwind and Edith Kaplan, “A Human Cerebral Deconnection Syndrome,” *Neurology* 12, no. 10 (October 1, 1962): 678.

³³ *Ibid.*, 676.

³⁴ Geschwind and Kaplan, “A Human Cerebral Deconnection Syndrome.”

surgery, he wrote, "*I knew not my leg.*"³⁵ He described himself as "an 'internal' amputee."³⁶ Although Sacks knew he could not have anosognosia and still be able to write about it, the paradox was tempting. In the book, *A Leg To Stand On*, he reflected on his experience of illness, alternating between his roles as both doctor and patient, in order to overcome the limits inherent to one's outside, objective knowledge about another's subjective, inner life.

Notwithstanding the differences in these representations of anosognosia, I am less interested in deciding which are right and which are wrong. Rather, I am guided more by the opportunity to analyze the social and philosophical *uses* of these representations and explanations. For, they always entailed more than that which strictly pertained to the disorder itself. They reflected the fitful expansion and consolidation of medical knowledge and the shifting divisions in clinical, academic, "specialized" expertise. In particular, they reflected the different ways of thinking about the relationship between the mind and brain, differences which often, but not always, mirrored divisions between, and within, psychiatry and neurology.³⁷

³⁵ Oliver Sacks, *A Leg to Stand On* (New York: Summit Books, 1984; New York: Touchstone, 1998), 51.

³⁶ *Ibid.*, 53.

³⁷ I do not mean to imply here that the so-called "Cartesian" division between the body and mind mapped seamlessly onto the presumably stark division between neurology and psychiatry. Rather, I wish to stress that there were multiple ways of construing the relationship between mind and body, and that these multiple schema reflected, and in part helped legitimate, specific demarcations among both clinical experts and their respective patient populations within and between psychiatry and neurology. My argument, therefore, departs from those who portray a more static dualism in the history between psychiatry and neurology. Anne Harrington, *Medicine, Mind, and the Double Brain: A Study in Nineteenth-Century Thought* (Princeton, N.J.: Princeton University Press, 1987), 250; Sander L. Gilman, ed., *Hysteria*

It is important to remember that psychiatry and neurology did not always mean what they do today. Indeed, they were constantly changing.³⁸ When they began to form in the last decades of the nineteenth century, neurology and psychiatry referred to very different clinical practices, standards of research, and institutional affiliations. In Austria-Hungary, for example, psychiatry and neurology were combined. In France, they remained separate. Most Austrian clinical psychiatrists routinely dissected the brains of their patients, earning them the posthumous title of “brain psychiatrists.”³⁹ French neurologists, by contrast, rarely could afford to practice only “neurology” *per se*. They were typically practitioners of internal medicine who saw many other types of patients

beyond Freud (Berkeley: University of California Press, 1993), xiii–xiv; William F. Bynum, “Rationales for Therapy in British Psychiatry: 1780-1835.,” *Medical History* 18, no. 4 (1974): 317.

³⁸ George Weisz, *Divide and Conquer: A Comparative History of Medical Specialization* (Oxford: Oxford University Press, 2006), xv–xvi; Rosenberg, “The Crisis in Psychiatric Legitimacy,” 141; Tomes, “Beyond the Two Psychiatries,” 776; John C. Burnham, “Jack Pressman and the Future of the History of Psychiatry,” *Bulletin of the History of Medicine* 74, no. 4 (2000): 785.

³⁹ Erwin Ackerknecht first used the term *Hirnpsychiater* to describe the clinical, academic psychiatrists of German-speaking regions of Europe in the latter half of the nineteenth century. I question the historical accuracy of this term in Chapter 1. It was first used by doctors themselves only starting in the 1910s in the United States (see Chapter 4). I propose that both the terms “brain psychiatry” and “neuro-psychiatry” require special caveat if applied to historical actors any earlier, lest we gloss over the forgotten but fruitful ambiguities in the name they gave themselves, simply “psychiatry.” Erwin H. Ackerknecht, *Kurze Geschichte der Psychiatrie* (Stuttgart: F. Enke, 1957); Brigitta Keintzel and Eberhard Gabriel, eds., *Gründe der Seele: Die Wiener Psychiatrie im 20. Jahrhundert* (Wien: Picus, 1999); Katja Guenther, “Recasting Neuropsychiatry: Freud’s ‘Critical Introduction’ and the Convergence of French and German Brain Science,” *Psychoanalysis and History* 14, no. 2 (2012): 203–26; German E. Berrios and Ivana S. Marková, “The Concept of Neuropsychiatry: A Historical Review,” *Journal of Psychosomatic Research* 53 (2002): 629–38; Edward Shorter, *A History of Psychiatry: From the Era of the Asylum to the Age of Prozac* (John Wiley and Sons, 1997); Anne Harrington, “Alter Wein in Jungen Schläuchen? Historische Bemerkungen Über Aktuelle Tendenzen in Der Neuropsychiatrie,” in *Psychiatrie Des Rechten Und Linken Gehirns: Neuropsychologische Ansätze Zum Verständnis Von Persönlichkeit, Depression Und Schizophrenie*, ed. Godehard Oepen (Köln: Deutscher Ärzte-Verlag, 1988), 17–25.

in addition to those with nerve- or brain-related disorders.⁴⁰ Similarly, the distinction between American neurology and psychiatry was never static. While some “neuropsychiatrists” of the 1930s paired psychoanalysis with the injection of barbiturates, others preferred lobotomies and mental-hygiene tactics.⁴¹ The logic of diagnosis and treatment was always changing with the definitions of expertise and institutional support. By using the history of anosognosia, one can closely investigate some of these important changes.

Above all, the denial of illness involved a problem of knowledge. Those who observed it and tried to explain it had to rely on historical assumptions about human behavior and infer from it about another person's subjective experience.⁴² Fundamentally, it forced doctors, who might otherwise have been less interested in philosophical speculation, to reflect on the limits of self-knowledge and the illusion of self-transparency. At first, it reinforced their claims to objectivity by pointing out the blatant gap of self-awareness in a disorder like anosognosia.⁴³ It showed that patients

⁴⁰ George Weisz, “Regulating Specialties in France during the First Half of the Twentieth Century,” *Social History of Medicine* 15, no. 3 (2002): 457–80; Jacques Philippon and Jacques Poirier, *Joseph Babinski: A Biography* (Oxford: Oxford University Press, 2008); Goetz, Bonduelle, and Gelfand, *Charcot*.

⁴¹ John C. Burnham, ed., *After Freud Left: A Century of Psychoanalysis in America* (Chicago: The University of Chicago Press, 2012); Gavrus, “Men of Dreams and Men of Action”; Pressman, *Last Resort*; Nathan G. Hale, *The Rise and Crisis of Psychoanalysis in the United States: Freud and the Americans, 1917-1985* (Oxford: Oxford University Press, 1995); Joel Braslow, *Mental Ills and Bodily Cures: Psychiatric Treatment in the First Half of the Twentieth Century* (Berkeley: University of California Press, 1997).

⁴² Jacyna offers some illuminating discussion on such techniques of inference and attempts to read for subjective experience, although he rather summarily dispatches the problem of experience. Jacyna, *Lost Words: Narratives of Language and the Brain, 1825-1926*, 6.

⁴³ Along with a number of historians of science and medicine, I am interested in how the historical distinctions were drawn between objectivity and subjectivity, but I am especially interested in showing how these distinctions break down in unique ways when scholars try to argue that the history of neurology and psychiatry presupposes a certain clinical Cartesianism. Lorraine Daston and Peter Galison,

generally possessed no reliable insight or powers of introspection into the nature of their illness and the failures of their own body.⁴⁴ Doctors knew best the bodies of others. But by the same token, anosognosia confounded their pretensions to objectivity. Essentially, they did not know what went on in the heads of their patients. They were left to speculate over the actual limits of another person's awareness. In this way, anosognosia exposed the inherent problems of knowing other minds as well as one's own.

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Chapter 1, “On a Certain Blindness: The Making of Anton’s Syndrome,” begins with the question of what made it possible, and persuasive, to imagine a disorder like the unawareness of illness. It focuses on the career of a particular Austro-Hungarian psychiatrist, Gabriel Anton, who, just before the turn of the twentieth century, described the “the loss of self-perception of focal brain disease.”⁴⁵ The notion of “focal brain disease” relied on ideas of disease specificity and the doctrine of cerebral

Objectivity (New York: Zone Books, 2007); Gerald N. Grob, “The Ambivalent Character of American Psychiatry,” *Journal of Health Politics, Policy and Law* 29, no. 3 (2004): 515–25; Tomes, “Beyond the ‘Two Psychiatries’”; Burnham, “Jack Pressman and the Future of the History of Psychiatry”; Gilman, *Hysteria beyond Freud*; Bynum, “Rationales for Therapy in British Psychiatry.”

⁴⁴ Kurt Danziger, *Constructing the Subject: Historical Origins of Psychological Research* (Cambridge: Cambridge University Press, 1994); Kurt Danziger, “Introspection: History of the Concept,” in *International Encyclopedia of the Social & Behavioral Sciences*, ed. Neil J. Smelser and Paul B. Baltes (Oxford: Pergamon, 2001), 7888–91; Kurt Danziger, “The History of Introspection Reconsidered,” *Journal of the History of the Behavioral Sciences* 16 (1980): 241–62.

⁴⁵ Anton, “Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit.”

localization,⁴⁶ which helped contribute to the rise, and rationalization, of academic psychiatry, even as it bred critics within its own ranks. I examine the unique predicament of self-described “scientific psychiatrists,” such as Anton, whose interest in disorders of consciousness served both to display their ambitions for neuroanatomy and scientific credibility, yet at the same time, endangered their own success by proximity to fraught debates on the soul.⁴⁷

Chapter 2, “The Revolution of Indifference, or How One Half of Hysteria Became Two New Brain Diseases,” takes up a similar question of what “made” a diagnosis like Anton’s syndrome, but the focus is on France and specifically on the transformation of a single symptom called *indifférence*. Once an integral part of the diagnosis of hysteria, the psychologist Pierre Janet used the concept of *indifférence* to defend certain philosophical convictions about the unity of the self.⁴⁸ Later, the neurologist Joseph

⁴⁶ Anne Harrington, “Beyond Phrenology: Localization Theory in the Modern Era,” in *The Enchanted Loom: Chapters in the History of Neuroscience* (New York: Oxford University Press, 1991), 207–39; Susan Leigh Star, *Regions of the Mind: Brain Research and the Quest for Scientific Certainty* (Stanford University Press, 1989).

⁴⁷ For other histories of German-speaking psychiatry, see Helmut Gröger, “Zur Entwicklung der Psychiatrie in der Wiener Medizinischen Schule,” in *Gründe der Seele: Die Wiener Psychiatrie im 20. Jahrhundert*, ed. Brigitta Keintzel and Eberhard Gabriel (Wien: Picus, 1999), 30–48; Schott and Tölle, *Geschichte der Psychiatrie*; Katja Guenther, “A Body Made of Nerves: Reflexes, Body Maps and the Limits of the Self in Modern German Medicine” (Harvard University, 2009); Michael Hagner, “Lokalisation, Funktion, Cytoarchitektonik: Wege zur Modellierung des Gehirns,” in *Objekte, Differenzen, und Konjunkturen: Experimentalsysteme im historischen Kontext*, ed. Michael Hagner, Bettina Wahrig-Schmidt, and Hans-Jörg Rheinberger (Berlin: Akademie Verlag, 1994); Michael Hagner, “Hirnforschung Und Materialismus,” *Der Materialismus-Streit*, 2007, 204–22; Michael Hagner, *Homo cerebrialis: der Wandel vom Seelenorgan zum Gehirn* (Berlin: Berlin Verlag, 1997).

⁴⁸ Elisabeth Roudinesco, *La bataille de cent ans: histoire de la psychanalyse en france* (Paris: Editions Ramsay, 1982); Philippon and Poirier, *Joseph Babinski*; Jacqueline Carroy and Régine Plas, “How Pierre Janet Used Pathological Psychology to Save the Philosophical Self,” *Journal of the History of the Behavioral Sciences* 36, no. 3 (2000): 231–40; Micale, *Approaching Hysteria*; Goetz, Bonduelle, and Gelfand, *Charcot*; Henri F. Ellenberger, *The Discovery of the Unconscious* (Basic Books, 1970).

Babinski “dismembered” the diagnosis of hysteria and converted the symptom of *indifférence* into two new types of brain disease, which he called “anosognosia” and “anosodiaphoria.” At the end of the chapter, I question Babinski’s narrative of the dismemberment and reconsider anosognosia in closer historical continuity with hysteria.

Chapter 3, “The Paradox of Health: Wartime Lessons from the Brain-Injured,” focuses on the work of Austrian psychiatrist Paul Schilder and German neurologist Kurt Goldstein. I discuss how their clinical encounters from the first world war convinced them to re-examine the definition of anosognosia and, ultimately, the meaning of health. They each critiqued the tendency of isolating symptoms and diseases narrowly based on the divisions between specialties and argued instead that each doctor endeavor to treat the “whole patient.” In this chapter, I explore the rise of such “holistic” patterns of medical thought which were inspired by an assorted mix of psychoanalysis, Gestalt psychology, phenomenology, and existentialist philosophy.⁴⁹ At the end, I discuss the wide reach of Schilder's and Goldstein's theories of anosognosia in their reciprocal influence on philosophy, in particular, on the French philosopher Maurice Merleau-Ponty, who used their analyses of anosognosia to illustrate what he called the “paradox of all perception” and his phenomenology of the body.⁵⁰

⁴⁹ Harrington, *Reenchanted Science: Holism in German Culture from Wilhelm II to Hitler*; Mitchell G. Ash, *Gestalt Psychology in German Culture, 1890-1967: Holism and the Quest for Objectivity*, Cambridge Studies in the History of Psychology (Cambridge: Cambridge University Press, 1995); Herbert Spiegelberg, *Phenomenology in Psychology and Psychiatry: A Historical Introduction* (Northwestern University Press, 1972).

⁵⁰ Maurice Merleau-Ponty, *Phenomenology of Perception*, trans. Colin Smith (1962; repr., New York: Routledge, 2002), 67, 95.

Chapter 4, “Filling in the Patient’s View: Denial Syndromes and American Neuropsychiatry of the Mid-Twentieth Century,” follows Schilder and Goldstein to the United States where they helped shift the study of anosognosia at the height of American psychoanalysis.⁵¹ The central protagonist of this chapter is Edwin Weinstein, whose long career spanned a variety of specialties and subspecialties, many of which have since been at supposed odds with one another. It explores the role of so-called “culture and personality” theorists like Edward Sapir and Harry Stack Sullivan in Weinstein’s studies of anosognosia, along with his formative experience as a combat neuropsychiatrist during World War II.⁵² At the end of the war, Weinstein teamed up with the psychologist Robert Kahn with whom he later wrote the first monograph ever dedicated to research on anosognosia called *Denial of Illness: Symbolic and Physiological Aspects*. By discussing their work from the late 1940s to the early 1960s, I show how their interpretation of “denial syndromes” drew from a wide variety of methodologies

⁵¹ Burnham, *After Freud Left*; Mitchell G. Ash and Alfons Söllner, *Forced Migration and Scientific Change: Emigré German-Speaking Scientists and Scholars After 1933* (Cambridge University Press, 2002); Hale, *The Rise and Crisis of Psychoanalysis in the United States*.

⁵² Hale, *The Rise and Crisis of Psychoanalysis in the United States*; Gerald N Grob, *From Asylum to Community: Mental Health Policy in Modern America* (Princeton, N.J: Princeton University Press, 1991); R.G. Kvarnes, “The Washington School of Psychiatry--Past Dreams and Present Realities,” *Psychiatry* 27 (August 1964): 295–300; Susan Lamb, “Pathologist of the Mind: Adolf Meyer, Psychobiology and the Phipps Psychiatric Clinic at the Johns Hopkins Hospital, 1908-1917” (Johns Hopkins University, 2010); Roy W Menninger and John C Nemiah, eds., *American Psychiatry After World War II (1944-1994)*, 1st ed (Washington, DC: American Psychiatric Press, 2000); Mark S. Micale and Roy Porter, *Discovering the History of Psychiatry*, 1st ed. (Oxford University Press, USA, 1994); Mical Raz, “Between the Ego and the Icepick: Psychosurgery, Psychoanalysis, and Psychiatric Discourse,” *Bulletin of the History of Medicine* 82, no. 2 (2008): 387–420; Ben Shephard, *A War of Nerves: Soldiers and Psychiatrists in the Twentieth Century* (Cambridge: Harvard University Press, 2003); John C. Burnham, “The Founding of the Archives of Neurology and Psychiatry; Or, What Was Wrong with the Journal of Nervous and Mental Disease?,” *Journal of the History of Medicine and Allied Sciences* 36, no. 3 (July 1981): 310–24.

and therapies, including “narcoanalysis” and “interpersonal” psychiatric interviewing.⁵³

The end of the chapter considers the challenges of writing histories of postwar American neurology and psychiatry, which were never as sharply defined as once imagined.⁵⁴

Chapter 5, “From Denial to Disconnection: Norman Geschwind and the Rise of Behavioral Neurology,” traces the history of anosognosia in the work of American neurologist Norman Geschwind, a contemporary of Weinstein who debated him on several occasions about their respective theories of anosognosia.⁵⁵ I use their “friendly disagreement” as a focal point to examine the decline of Weinstein’s version of neuropsychiatry alongside the steady rise of what Geschwind called “behavioral

⁵³ Edwin A. Weinstein, Louis Linn, and Robert L. Kahn, “Psychosis During Electroshock Therapy: Its Relation to the Theory of Shock Therapy,” *The American Journal of Psychiatry* 109, no. 1 (July 1952): 22–26; E.A. Weinstein, “The Relationship to Dreams of Symbolic Patterns Following Brain Injury,” in *Dreams in Contemporary Psychoanalysis* (New York: Society of Medical Psychoanalysis, 1963); Edwin A. Weinstein, Robert L. Kahn, and Sidney Tarachow, “Denial of Illness in Brain Tumor,” *Transactions of the American Neurological Association* 74 (1949): 67–69; Edwin A. Weinstein and Sidney Malitz, “Changes in Symbolic Expression with Amytal Sodium,” *American Journal of Psychiatry* 111, no. 3 (September 1, 1954): 198–206; Edwin A. Weinstein, “Symbolic Neurology and Psychoanalysis,” in *Modern Psychoanalysis: New Directions & Perspectives*, ed. Judd Marmor (Piscataway, NJ: Transaction Publishers, 1968), 225–50; Alison Winter, “The Making of ‘Truth Serum,’ 1920-1940,” *Bulletin of the History of Medicine* 79, no. 3 (2005): 500–533.

⁵⁴ While I agree with Nancy Tomes’ assessment that it is hard to “maintain a clean story line” when twentieth-century doctors so often alternated their claims to professional identity, from psychiatrist to neurologist and then from neurologist to neurosurgeon, I do not share her assessment that the twentieth century was “harder” than the nineteenth century: “The rapid rise of medical specialism produced a complex array of individuals identifying themselves as psychiatrists, neurologists, or some hybrid of the two...compared to the nineteenth century, in which generalizations about ‘asylum doctors’ versus ‘neurologists’ could be made with comparative security, the complexity of twentieth-century actors claiming the professional identity of psychiatrist, neurologist, or neuropsychiatrist makes it much harder to maintain a clean story line.” Tomes, “Beyond the ‘Two Psychiatries,’” 776.

⁵⁵ Davis Howes and Norman Geschwind, “Quantitative Studies of Aphasic Language,” in *Disorders of Communication, Proceedings of Association for Research in Nervous and Mental Diseases, December 7 and 8, 1962, New York, NY*, ed. Edwin Weinstein and David McK. Rioch, vol. 42 (Baltimore: Williams and Williams, 1964), 229–44; E. A. Weinstein, M. Cole, and M. S. Mitchell, “Anosognosia and Aphasia,” *Transactions of the American Neurological Association* 88 (1963): 172–75.

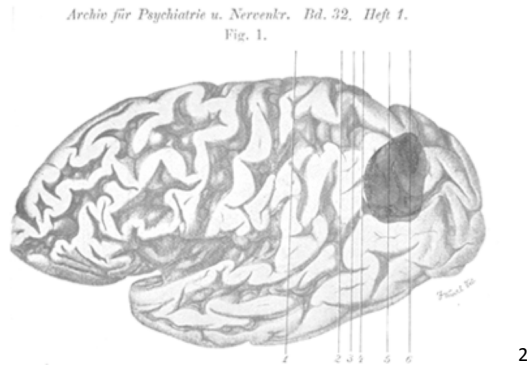
neurology.” I am particularly concerned with understanding the ramifications of Geschwind’s view of anosognosia as a “disconnection syndrome” and, therefore, as clinical evidence of the disunity of consciousness—indeed, the unintelligibility of “the patient’s experience.”⁵⁶ In closing, I propose that the historical study of anosognosia can give one a new appreciation for the medical historiography of “the patient’s view,”⁵⁷ as it grew out of this period and has shaped how the history of medicine is written today.

⁵⁶ Marco Catani and Dominic H ffytche, “The Rises and Falls of Disconnection Syndromes,” *Brain* 128, no. 10 (2005): 2224–39; John R. Absher and D. Frank Benson, “Disconnection Syndromes: An Overview of Geschwind’s Contributions,” *Neurology* 43, no. 5 (1993): 862–67; Norman Geschwind, “Disconnexion Syndromes in Animals and Man. I,” *Brain: A Journal of Neurology* 88, no. 2 (June 1965): 237–94; Norman Geschwind, “Disconnexion Syndromes in Animals and Man. II,” *Brain: A Journal of Neurology* 88, no. 3 (September 1965): 585–644; Geschwind and Kaplan, “A Human Cerebral Deconnection Syndrome.”

⁵⁷ Stolberg, *Experiencing Illness and the Sick Body in Early Modern Europe*; Condrau, “The Patient’s View Meets the Clinical Gaze”; Porter, “The Patient’s View”; Armstrong, “The Patient’s View.”

Chapter 1

On a Certain Blindness:¹ The Making of Anton's Syndrome



*"'Tis the blot upon the brain
That will show itself without."³*

On November 30, 1894, fifty-six year-old seamstress Ursula Mercz entered a clinic in Graz, Austria, complaining of headaches, back pain, and dizziness. During the admission exam, she struggled with speaking, sometimes inventing fake words, and begged not to be bothered by any more testing. After nearly a month in the clinic, her vision began to deteriorate. She could no longer recognize objects like a triangle or a red

¹ Although only indirectly related to Anton's syndrome, an essay by William James with a similar title, published the same year, inspired this chapter's title. On the opening page, James writes of a blindness not confined to any particular patient but inherent in every person confronted with the presence of mind of another: "Now the blindness in human beings, of which this discourse will treat, is the blindness with which we all are afflicted in regard to the feelings of creatures and people different from ourselves." William James, "On a Certain Blindness in Human Beings (1899)," in *Writings, 1878-1899* (New York: Library of America, 1992), 841.

² FIG. 1: Anton's re-touched photograph of Ursula Mercz's brain. Anton, "Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit"; Sarah de Rijcke, "Light Tries the Expert Eye: The Introduction of Photography in Nineteenth Century Macroscopic Neuroanatomy," *Journal of the History of the Neurosciences* 17 (2008): 349–66.

³ Alfred Lord Tennyson, "Maud," in *The Harvard Classics*, ed. Charles William Eliot, vol. 42 (P.F. Collier & son, 1910).

ribbon placed on the table in front of her. By January, her sight was completely gone.

However, she never knew it.⁴

How was this possible? Mercz was not delirious. She knew where she was, who she was, and to some extent at least, why she was in the clinic. The only aspect about herself that she was confused about, indeed oblivious to, was the fact that she could no longer see. If asked to name what object was held in front of her face, she would guess with a blank, fixed stare in the wrong direction. When asked about her eyesight, she casually dismissed it, saying, "That is just how it is. One sees better in youth."⁵ After only a few months in the clinic, Mercz's health declined considerably. The left side of her body grew weak and then paralyzed, and her pulse in the right carotid artery of her neck was barely detectable. She fell into a coma that lasted little more than a month when she died on May 29, 1895.⁶

Despite her death, Ursula Mercz's story was not yet over, however. On January 27, 1896, her physician Gabriel Anton (1858-1933) reported the case of "Ursula M." at a regional meeting of physicians. As he described Mercz's symptoms, including her blindness, back pain, dizziness, and speech disorder, he concentrated on one symptom

⁴ Gabriel Anton, "Über Herderkrankungen des Gehirns die vom Patientin selbst nicht wahrgenommen werden," *Wiener klinische Wochenschrift* 11 (March 10, 1898): 227–229; Anton, "Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit," 92–94.

⁵ Anton, "Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit," 93.

⁶ *Ibid.*, 94.

in particular. Merz had been not only blind. She had become “mentally blind to her blindness” [*seelenblind für ihre Erblindung*].⁷

A few months after Ursula Merz passed away, Anton encountered another patient with similar symptoms. On October 2, 1895, sixty-nine year-old Juliane Hochrieser arrived at Anton's clinic in a weak and confused state. She could not understand any of his questions. She never complained, indeed showed no reaction, to loud noises around her. It turned out she was completely deaf. Since she could still read, Anton asked her in writing, “Frau Hochrieser, do you hear well?” Deflecting the question, she replied, “Yes, that is what I am called, that is my name.”⁸ Despite his efforts, Anton could not persuade her to admit to being deaf.

Within the year, Hochrieser died from severe bronchitis. Anton performed an autopsy to look for damaged areas of brain tissue that might account for her “cortical deafness” and corresponding unawareness. Like Ursula Merz, the story of Juliane Hochrieser did not exactly end with her death.⁹ On December 20, 1897, Anton reported her case history to fellow physicians in Graz and concluded, “The patient Hochrieser *knew nothing of her total deafness* and took no notice of it. She became, as it were,

⁷ Anton, “Blindheit nach beiderseitiger Gehirnerkrankung mit Verlust der Orientierung im Raume,” 45.

⁸ Anton, “Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit,” 107.

⁹ Jacyna writes that the patient's death was treated as “merely prologue” while the autopsy was the “climax.” Whether or not one ascribes to this strong reading of the case narrative genre, it does seem compelling to say that the story of such patients' lives and identities continued to be written even after their lives were over. Jacyna, *Lost Words: Narratives of Language and the Brain, 1825-1926*, 42, 115.

mentally deaf to her deafness.”¹⁰ Now there were two individual cases, each featuring a kind of mental blindness (or deafness), which suggested the existence of a new kind of disease entity, specifically, a disease that prevented the feeling of illness.¹¹

Anton published his clinical and anatomical examinations of Merz and Hochrieser just before the turn of the twentieth century, in 1899. The title of his paper was “On the Self-Perception of the Focal Diseases of the Brain in Patients with Cortical Blindness and Cortical Deafness.” It appeared in the leading German journal of psychiatry, the *Archive for Psychiatry and Nervous Diseases* [*Archiv für Psychiatrie und Nervenkrankheiten*].¹² Beyond that, there was little fanfare to his publication. The paper read much like the others it appeared alongside in the journal: a sober, systematic discussion of case histories and autopsy results with a careful review of related medical literature. Read in this way, it may seem that Anton's paper was in fact merely medical,

¹⁰ Anton, “Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit,” 119; Anton, “Über Herderkrankungen des Gehirnes, welche vom Patienten selbst nicht wahrgenommen werden,” 12–13.

¹¹ Many scholars have taken to distinguishing the terms “disease” and “illness.” Typically “illness” denotes the personal, subjective aspect of feeling sick, the experience of the so-called sick man or sufferer (not “patient”), while “disease” denotes the anonymized abstraction, the statistical index, the invading germ, or the decaying tissue. In other words, the lab tests and diagnoses do not always (or ever?) capture the patient's experience. Though this distinction is far from hard and fast, it can be useful to build upon, if only to complicate, as in the case of Anton's syndrome. Rosenberg provides a nice concise distinction of illness and disease: “This is the characteristic split screen...the tension between illness in the individual patient and disease as crystallized and socially real in the laboratory's and the epidemiologist's outputs.” Rosenberg, “Introduction: The History of Our Present Complaint,” 6; Martin Dinges, “Social History of Medicine in Germany and France in the Late Twentieth Century; From the History of Medicine toward a History of Health,” *Locating Medical History*, 2004, 208; Kleinman, *The Illness Narratives*, 4.

¹² The Berlin psychiatrist Wilhelm Griesinger founded the journal shortly before his death in 1868. His successor in Berlin, Carl Westphal (1833-1890), took over as editor until his death in 1890. Gabriel Anton himself was an editor later in his career. Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice*, 126; H.H. Eulner, *Die Entwicklung der medizinischen Spezialfächer an den Universitäten des deutschen Sprachgebietes*, vol. 4 (Stuttgart: F. Enke, 1970), 281; Friedrich Hartmann, “Gabriel Anton zum 70. Geburtstag,” *Münchener medizinische Wochenschrift* 75 (1928): 1507.

just one more contribution to the growing corpus of psychiatric research. And perhaps it was, or he meant for it to be, at least in that form. But in terms of its content, the particular subject matter, Anton struck out into uncharted territory. This paper was not just about a new brain disease. It was about the anatomical origins of the experience of illness. Ultimately, it was about the nature of consciousness and the existence of the soul.¹³

This chapter examines the early history of a disorder which was eventually called “Anton’s syndrome.” It explores how it was “made”—that is, how it could have been first imagined as a disease, then recognized in a particular individual, and finally used as a diagnosis. In turn, I also use the diagnosis as a base from which to analyze the relationship of larger themes and problems in the history of medicine, including concepts of disease specificity, medical specialization, and the debates over scientific materialism. The focus of the chapter is then split between the trajectory of Anton’s career as an Austro-Hungarian psychiatrist of the late nineteenth century,¹⁴ and the

¹³ While it is certainly true that many physicians and physiologists were very interested in questions about the soul and consciousness long before Anton appeared on the scene, he was the first to describe explicitly specific parts of the brain that potentially allowed for the personal awareness of being sick or limited in some way like lacking sight or being paralyzed. In 1885, the Swiss psychiatrist Constantin von Monakow briefly alluded to this possibility, but Anton exploited it and launched the first sustained discussion. Constantin von Monakow, “Experimentelle und pathologisch-anatomische Untersuchungen über die Beziehungen der sogenannten Sehsphäre zu den infracorticalen Opticuscentren und zum N. opticus,” *Archiv für Psychiatrie und Nervenkrankheiten* 16 (1885): 166–167.

¹⁴ Names are important to historians, some of whom have taken to calling the style of psychiatry that Anton practiced “neuro-psychiatry” or “brain psychiatry” [*Hirnpsychiatrie, Gehirnpsychiatrie, or Neuropsychiatrie*]. In fact, no one described it in these terms in the nineteenth century. The label is an historical artifact first applied almost a century later in 1957 by the medical historian Erwin Ackerknecht. Since Ackerknecht, others have used similar terms such as “neurological psychiatry” and “biological psychiatry.” But none of these were explicitly used at the time neither by Anton or his German-speaking

impact of localization theory on the changing ideal of both scientific and psychiatric identity at the end of the century. At the end of the chapter, I consider whether Anton's syndrome may be creatively read as a "symptom" of the pervasive preoccupation with scientific indeterminacy, social detachment, and personal dissolution in turn-of-the-century Central Europe.

Out of Confusion and Into Awareness

Ideas about disease have always been tightly linked to, if not synonymous with, the identity of medicine. These ideas have been instrumental in connecting the individual patient's experience to a larger body of knowledge aimed at making sense of that experience, to explain it in generalizable terms, and to treat it. Ideas about disease, then, help translate the idiosyncrasies of personal experience, not just of the patient

colleagues. They described themselves simply as "psychiatrists." This is not to imply that they did not try to distinguish themselves as psychiatrists. It is precisely that they *tried*, but their efforts were not neatly subsumed under a monolithic label that the hyphenated prefix "neuro-" would seem to suggest. Certainly, they expressed strong opinions about the proper direction psychiatry should proceed, but they often shared the same name with other psychiatrists who did not share the same opinions. For this reason, it is anachronistic to call them "neuro-psychiatrists," much less "neurologists." This "nominal" confusion becomes all the more critical in subsequent chapters which examine various specialists' revisionist histories of psychiatry and neurology in the latter half of the twentieth century. For now, I wish simply to emphasize that names do matter and that the distinction of medical specialties, particularly dealing with the mind, was anything but tidy and neat. Ackerknecht, *Kurze Geschichte der Psychiatrie*, 69, 77; Erwin H. Ackerknecht, "Gudden, Huguenin, Hitzig. Hirnpsychiatrie im Burghölzli 1869-1879," *Gesnerus* 35, no. 1-2 (1978): 73; Guenther, "Recasting Neuropsychiatry"; Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice*, 60; Shorter, *A History of Psychiatry*, 69; Andrew Kertesz and Pavel Kalvach, "Arnold Pick and German Neuropsychiatry in Prague," *Archives of Neurology* 53, no. 9 (1996): 935-38; Berrios and Marková, "The Concept of Neuropsychiatry: A Historical Review"; E. Kumbier, K. Haack, and S. Herpertz, "Überlegungen zum Wirken des Neuropsychiaters Gabriel Anton (1858-1933)," *Nervenarzt* 76, no. 9 (2005): 1132-40; E.D. Caine and R.J. Joynt, "Neuropsychiatry . . . Again," *Archives of Neurology* 43, no. 4 (April 1986): 325-27; Georg Northoff, "Neuropsychiatry. An Old Discipline in a New Gestalt Bridging Biological Psychiatry, Neuropsychology, and Cognitive Neurology," *European Archives of Psychiatry and Clinical Neuroscience* 258, no. 4 (June 2008): 226-38; W. Schmitt, "Das Modell der Naturwissenschaft in der Psychiatrie im Übergang vom 19. zum 20. Jahrhundert," *Berichte zur Wissenschaftsgeschichte* 6, no. 1-4 (1983): 89-101; Klaus Wiese, "Vom hirnpsychiatrischen Paradigma zu einer humanwissenschaftlichen Psychiatrie," *Wissenschaftliche Zeitschrift der Karl-Marx-Universität Leipzig, Mathematisch-Naturwissenschaftliche Reihe* 31 (1982): 139-49.

who suffers but of all who witness that suffering and want to give it a name, into the generalities of what gets to be called “disease.” In this way, ideas about disease are not just about disease. They connect up to other ideas and assumptions, specific to every historical moment and cultural context, about what sorts of behaviors pass as correct and healthy and desirable, or at least acceptable, and what do not.¹⁵ They even connect up to ideas about what counts as *experience*, what is agreed upon and can be expected to look like *perception*, and ultimately what gets to be called *consciousness*.

Many scholars have written about the “making” of diseases, from malaria to mania.¹⁶ Among them probably the most original and influential was a Polish doctor named Ludwik Fleck (1896-1961), who in 1934 wrote the *Genesis and Development of a Scientific Fact*.¹⁷ The “fact” Fleck had in mind was syphilis. He wanted to probe how this particular disease came to be, not so much in terms of incidence rates or causes of contagion, nor the individual factors of diagnosis, but its conception and intelligibility as a disease, the very idea of its existence as “*scientific fact*.” Fundamentally, Fleck was interested in the role of perceptual experience, specifically, how it was constrained by historical “styles of thought” [*Denkstille*] and accumulated to form consensus, then gradually crystallized into fact.¹⁸ The subtle but critical shift for Fleck was away from

¹⁵ Rosenberg, “The Tyranny of Diagnosis,” 241.

¹⁶ Healy, *Mania*; Packard, *The Making of a Tropical Disease*; Robert A. Aronowitz, *Making Sense of Illness: Science, Society, and Disease*, Cambridge History of Medicine (Cambridge, U.K. ; New York, NY: Cambridge University Press, 1998).

¹⁷ Fleck, *Genesis and Development of a Scientific Fact*.

¹⁸ *Ibid.*, 95.

thinking about facts as passively perceived to thinking about facts as actively “directed perceptions.”¹⁹ He wanted to emphasize the cognitive and experiential, even phenomenological,²⁰ side to science and medicine, and their constructive nature as a set of group exercises in making knowledge, if only to emphasize that this knowledge was always incomplete and under continual formation. Fleck's stress on perception, therefore, was integral to his argument that scientific and medical facts were generated and constructed through a collection of minds, relying on specific parts of the body, and rooted in historical habits of social organization.²¹

And so with Anton. It is difficult to say when, where, or even who exactly “made” what became the disease entity known as “Anton's syndrome.”²² After all, he himself never referred to it as such. But someone did at some point. The earliest record dates to 1918, when a former student proposed to honor Anton by naming it: “Anton's

¹⁹ *Ibid.*, 99.

²⁰ *Ibid.*, xxviii.

²¹ “[E]xperience must be understood as a complex state of intellectual training based upon the interaction involving the knower, that which he already knows, and that which he has yet to know. The acquisition of physical and psychological skills, the amassing of a certain number of observations and experiments, the ability to mold concepts, however, introduce all kinds of factors that cannot be regulated by formal logic. Indeed, such interactions as those mentioned prohibit any systematic treatment of the cognitive process.” *Ibid.*, 10–11.

²² George P. Prigatano and Thomas R. Wolf, “Anton’s Syndrome and Unawareness of Partial or Complete Blindness,” in *The Study of Anosognosia*, ed. George P. Prigatano (Oxford University Press, 2010), 455–68; Ekkehardt Kumbier, Kathleen Haack, and Sabine C. Herpertz, “Der Fall der Juliane Hochriehser [sic]: Eine klassische Fall von Anosognosie,” in *Würzburger medizinhistorische Mitteilungen*, ed. Michael Stolberg et al., vol. 26 (Königshausen und Neumann, 2007), 53–74; Forde and Wallesch, “‘Mind-Blind for Blindness’: A Psychological Review of Anton’s Syndrome”; Hans Förstl, Adrian M. Owen, and Anthony S. David, “Gabriel Anton and ‘Anton’s Symptom’: On Focal Diseases of the Brain Which Are Not Perceived by the Patient (1898),” *Cognitive and Behavioral Neurology* 6, no. 1 (1993): 1–8; A. David, A.M. Owen, and H. Förstl, “An Annotated Summary and Translation of ‘On the Self-Awareness of Focal Brain Diseases by the Patient in Cortical Blindness and Cortical Deafness’ by Gabriel Anton (1899),” *Cognitive Neuropsychology* 10, no. 3 (1993): 263–72; B. E. Swartz and J. C. M. Brust, “Anton’s Syndrome Accompanying Withdrawal Hallucinosis in a Blind Alcoholic,” *Neurology* 34, no. 7 (1984): 969–969.

symptom.”²³ Never mind the awkward implication that, given the definition of “symptom” as the patient's subjective experience of disease, “Anton's symptom” would actually denote the lack of itself.²⁴ In any case, Anton's symptom, or syndrome, or at least what it signified, emerged initially through a series of Anton's own publications in the last decade of the nineteenth century. That it *could*, however, begs for a different sort of inquiry from what it *was*.

The possibility that there could be a syndrome without symptoms, and without any sign of the patient's subjective awareness of it, relied on a very basic idea about disease. It relied on the idea of disease as something specific, namely, as some *thing* apart from the person harboring it. In other words, disease was not so much a state of experience, say of feeling sick or in pain, but instead it denoted a specific causal entity, like a germ or foreign body that invaded the patient's own, thus existing outside of the patient's experience. This notion of disease specificity, it should be added with some

²³ Otto Albrecht, “Drei Fälle mit Anton's Symptom,” *Archiv für Psychiatrie und Nervenkrankheiten* 59, no. 2 (1918): 883–941.

²⁴ “Symptoms are the complaints of the patient that are spontaneously reported or elicited by the clinical history. Signs are observed by the physician, the patient, or the patient's friends or relatives and indicate the presence of abnormal functioning of one or more body systems...A syndrome is a constellation of signs and symptoms that seem to coalesce to provide a recognizable entity with its defining characteristics. Syndromes may be classified, and they are the clinical representatives of illness. The latter is what the patient presents to the physician with, which in part may represent the expression of disease. However, the presentation of an illness depends on many factors, including environmental and personality variables.” Jeffrey L. Cummings and Michael R. Trimble, *Concise Guide to Neuropsychiatry and Behavioral Neurology* (Washington, D.C.: American Psychiatric Pub, 2002), 1. Evans, Martyn, and Rolf Ahlzen. *Symptom*. Radcliffe Publishing, 2008.

irony, often diminished the patient's own specificity or individuality, with the effect that disease entities were often treated as more individuated than individual patients.²⁵

Anton drew on the intuitive force of disease specificity, which had gathered critical momentum in the last half of the nineteenth century,²⁶ to argue that what he had witnessed in his patients was not to be confused with some non-specific type of dementia, delirium, or delusion. Instead, it was a highly specific unconsciousness, confined only to the patient's lack of vision or hearing and nothing else. "Here," Anton emphatically declared, "I speak *not* of the illnesses, where, owing to the profound reduction or abolition of mental activity through severe insanity or severe disturbances of consciousness, perception and judgment have actually ceased."²⁷ No, perception did not "cease," only the "self-perception" [*Selbstwahrnehmung*] of their symptoms.

²⁵ Fleck, *Genesis and Development of a Scientific Fact*, chap. 1, passim. Foucault writes, "[P]lus on envisage comme un tout l'unité de l'être humain, plus se dissipe la réalité d'une maladie qui serait unité spécifique ; et plus aussi s'impose, pour remplacer l'analyse des formes naturelles de la maladie, la description de l'individu réagissant à sa situation sur le mode pathologique." Michel Foucault, *Maladie mentale et psychologie*, 2e ed, Quadrige 198 (1954; repr., Paris: Presses universitaires de France, 1997), 11; Jacyna, *Lost Words: Narratives of Language and the Brain, 1825-1926*, 49; Rosenberg, "The Tyranny of Diagnosis," 240–247ff.

²⁶ Even before the germ theory of disease emerged, Rosenberg observes, "Pathological anatomy with its emphasis on localized lesions, physical diagnosis, the beginnings of chemical pathology, and studies of normal and abnormal physiological function all pointed toward the articulation of stable disease entities that could be—and were—imagined outside their embodiment in particular individuals and explained in terms of specific causal mechanisms within the sufferer's body." Rosenberg, "The Tyranny of Diagnosis," 242. Stolberg, however, questions this historical narrative that makes disease specificity an idea specific to the rise of modern medicine: "The importance of an ontological conception of disease in pre-modern medicine has been denied time and again. Pre-modern medicine, it has been claimed, was based on an individualizing, basically physiological understanding of disease as a state of imbalance between the natural humors and their associated elementary qualities....The tendency to objectify disease, to focus the medical gaze on the disease rather than on the patient as a whole, which is often lamented today as one of modern medicine's central shortcomings, may thus well have much older roots." Stolberg, *Experiencing Illness and the Sick Body in Early Modern Europe*, 25–26.

²⁷ Anton, "Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit," 86–87.

Neither Merz's behavior nor Hochrieser's was to be confused with what was loosely called "general confusion" [*Verwirrtheit*].²⁸ That was because they both remained surprisingly lucid despite this conspicuous gap in their self-awareness. True, Anton admitted, relatives and friends of Merz found her to be "mentally changed" [*geistig verändert*].²⁹ Also, Hochrieser had arrived at his clinic in a state of "intense confusion and excitement" [*hochgrädig Verworrenheit und Erregung*].³⁰ But none of this apparent "madness" [*Blödsinn*], Anton judged, was of any "sufficient degree" [*ohne das genügender Grad von Blödsinn*].³¹ Sufficient for what? Presumably, to invalidate his diagnosis. Admitting to "confusion" was tantamount to contamination. It smudged the clean lines separating this new disease-picture from the nebula of mental disturbances lumped together under "madness." It forsook the gold standard of specificity and diagnostic purity that was thought to further medical knowledge. And so, Anton decided, the mental changes were marginal, insignificant, and insufficient to corrupt his conclusion that this was, in fact, a new and focused disease [*Herderkrankung*] of self-awareness.

That was a decision Anton made. Did he lower the bar of what constituted confusion? One may only speculate. We do know that a few years earlier he described

²⁸ Theodor Meynert, "Amentia, die Verwirrtheit," *Jahrbücher für Psychiatrie und Neurologie* 9 (1890): 1–112.

²⁹ Anton, "Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit," 92.

³⁰ *Ibid.*, 106.

³¹ *Ibid.*, 123.

similar behavior in two other patients in a publication from 1893.³² “Johann K.” was a sixty-five year-old accountant who for the past five years had suffered frequent bouts of anxiety and taken up drinking to ease his mind. A week before he came to the clinic, he had suddenly felt extremely dizzy and decided he needed to go home. On the way, he could not keep from staggering and drifting to the left. When he got home, he complained that the living room was “glowering” at him, and he saw “flies and flecks” in the air.³³ In the clinic, he complained that his daughter lay in the bed beside him, pestering him and making him engage in illicit activities, then sticking her finger into his eye. He also saw his wife sitting on the left side of his bed, making sexual advances toward the hospital warden. Later, he said that his left arm and leg did not belong to him, and he constantly tugged at the hand as if trying to pull off a glove. Anton concluded that he was “completely confused” [*sich völlig verworren*].³⁴

The second case was “Wilhelm H.,” a sixty year-old janitor from Vienna. He was admitted to the psychiatric clinic after being knocked unconscious by a moving trolley car. When he woke, he noticed a certain clumsiness with his left hand and numbness in his fingers as if they were made of leather. He also complained about the entire left side of his body feeling cold. At home, he said that a strange man had dumped water in his room and threatened him with a snake. When he arrived at the clinic, his left arm was

³² Anton, “Beiträge zu klinischen Beurtheilung und zur Localisation der Muskelsinnstörungen im Grosshirne.”

³³ *Ibid.*, 317.

³⁴ *Ibid.*, 317–320, 317ff.

completely paralyzed and his left leg was extremely weak. Anton made the following note, “The patient is slightly confused, [and] *is unaware of his paralysis.*”³⁵

One cannot say with certainty whether it was accidental or intentional that in these case histories, from his first observation in 1890 until his last publication in 1899, Anton gradually de-emphasized his patients' confusion, downgrading it successively from “completely confused” to “slightly confused” to “no sufficient degree of confusion.” But the question of his intention is not as important as the fact of his description. It shows that he had already made up his mind about what confusion looked like and what counted as its “sufficient degree.” Such decisions, however minute and difficult to detect, determined the diagnosis. They were the kind of decisions born of accumulated experience over years of training and apprenticing in psychiatric clinics across the Austro-Hungarian Empire. As Fleck would have probably said, Anton’s clinical perception was not so much direct as *directed*. What he saw, what he believed he was seeing, depended on how he was trained to look. It depended on a certain “readiness” to see, or what Fleck called “stylized visual perception.”³⁶ Anton's perception, undergirded by the belief that confusion could be focalized, and that disease affecting behavior could be localized, was a creation of his time, a product of its own history.

Fleck also wrote that to study knowledge, or epistemology, without studying history was like trying to study anatomy without embryology.³⁷ Ideas develop, he

³⁵ Ibid., 325.

³⁶ Fleck, *Genesis and Development of a Scientific Fact*, 84, 92–94.

³⁷ Ibid., 20–21.

seemed to be saying, not just in one's head but in one's time, and place. So too with Anton and the making of Anton's syndrome. He drew on a number of mentors in psychiatry and their recent work to arrive at his diagnosis of Merz and Hochrieser at the century's end. The first of these mentors was Arnold Pick (1851-1924), professor of psychiatry at the University of Prague.³⁸ When he was sixteen, Anton left his hometown of Saaz in the region of Bohemia (now the Czech Republic) for Prague to study medicine in 1874. There he took classes in anatomy from Hans Chiari (1851-1916), with a focus on physiology of the nervous system, and in the physical sciences from Ernst Mach (1838-1916), who had already begun to focus on the "psychophysics" of sensation and perception. It would seem that Anton's medical focus sprang from their combination, on one side neuroanatomy and on the other experimental psychology, and so it is a little less than surprising that after he earned his medical degree in 1882 he chose to train specifically in clinical psychiatry at an asylum on the outskirts of the city, in Dobrzan, which is where he met Arnold Pick.³⁹

Pick had worked, since 1877, at both the asylum in Prague, called the "Katerinskà," and the one in Dobrzan. In 1882, just as Anton arrived to assist him, Pick became the director of the "psychiatric institute" at Dobrzan, which gave him more liberty to pursue research as well as to stimulate Anton in his own.⁴⁰ And, apparently,

³⁸ Kertesz and Kalvach, "Arnold Pick and German Neuropsychiatry in Prague."

³⁹ Hartmann, "Gabriel Anton zum 70. Geburtstag"; B. Pfeifer, "Gabriel Anton," *Journal of Neurology* 130, no. 5 (1933): 185–96; Kumbier, Haack, and Herpertz, "Überlegungen zum Wirken des Neuropsychiaters Gabriel Anton (1858-1933)."

⁴⁰ Kertesz and Kalvach, "Arnold Pick and German Neuropsychiatry in Prague," 936.

stimulate he did. The same year, Pick published an extensive historical review of the clinical literature on what was called “illness-consciousness” [*Krankheitsbewusstsein*],⁴¹ or the specific kind of awareness of one's own illness, which was customarily referenced in cases of mental illness, that is, cases where insight was least expected. Pick was especially interested in the “phenomenology,” in this case meaning more simply, “symptomatology,” of the subjective varieties of insight. In a long paper published in the *Archiv für Psychiatrie und Nervenkrankheiten* under the title, “On Illness-Consciousness in Mental Illnesses,” Pick dissected the nuanced subjective experiences of feeling sick, which he arrayed on a spectrum from the vaguest “illness-feeling” [*Krankheitsgefühl*] to “illness-consciousness” and finally at the farthest extreme of clarity, “illness-insight” [*Krankheitseinsicht*]. He was very taken in by the qualified shadings of awareness described in, and sometimes by, the patients themselves, where occasionally the insight into their plight would immediately dawn upon them,⁴² but where usually their insight only faintly flickered like a “dim prehension” [*dunkler Ahnung*] that something was not right, something was changed, or something was missing.⁴³

⁴¹ Arnold Pick, “Über Krankheitsbewusstsein in psychischen Krankheiten,” *Archiv für Psychiatrie und Nervenkrankheiten* 13 (1882): 518–81.

⁴² “The awareness that one looks crazy and sounds crazy is present but still one can do nothing to change it.” *Ibid.*, 569.

⁴³ “...mit vorhandener dunkler Ahnung des ungehörigen Zustandes zusammenzuhängen scheint.” *Ibid.*, 564; A year after Pick's clinical-historical study, another Austrian psychiatrist Richard Krafft von Ebing (1840-1922) documented similar “twilight-states” of “illness-insight” in his own patients, also characterizing it as a kind of dawning or “partial dimming of awareness.” Richard Krafft-Ebing, *Lehrbuch der Psychiatrie auf klinischer Grundlage für praktische Ärzte und Studierende* (Stuttgart: F. Enke, 1883), 102–103.

Anton must have taken note. For, this was how he, too, would later describe the residual, fugitive awareness in Merz and Hochrieser. There seemed to persist in them some “dark knowledge” [*dunkle Kenntnis*] or “dim feeling” [*dunkles Gefühl*] that something was wrong, that they were changed somehow, even if they did not know, or could not say, exactly what it was.⁴⁴ There was a difference, though, between Pick's and Anton's interest in “illness-consciousness.” In fact, they were inversions of one another. While Pick was interested in the experience of feeling sick as a healthy sign in mentally ill patients, Anton was interested in the *lack* of feeling sick as a pathological sign in patients with brain disease but without any other indication of mental illness. So the difference hinged on not only the presence versus absence of awareness, or the experience of feeling sick, but also on the type of patient, that is, the presence or absence of mental illness.

Anton's focus was on absence—the absence of illness-awareness in the absence of mental illness. That was what made his diagnosis special, not to mention difficult to assess. How could he be sure, or at least what gave him enough confidence to believe, that awareness could be so circumscribed, so compartmentalized, as to form a clean break only at the edges of consciousness related to illness but nothing else? It was not enough that Pick may have imparted to him the idea of “illness-consciousness.” He needed more than this to make his case. He also relied on a specific understanding of

⁴⁴ Anton, “Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit,” 87, 122.

consciousness, which could admit, like the eye's retina, patches of "seeing-nothing" [*Nichtssehen*], as if there were "blind spots" [*blinde Flecke*] in the mind.⁴⁵

The idea that consciousness, and unconsciousness, might be comparable to sensation, and the lack thereof, was an old one.⁴⁶ But it was breathed new life in the latter half of the nineteenth century.⁴⁷ And one doctor in particular, a young psychiatrist named Carl Wernicke (1848-1905), whom Anton admired and cited often, used this model of mind to make a pivotal contribution to the study of mental illness and brain disease.⁴⁸ In 1874, Wernicke reported a new type of language disorder he called "sensory aphasia."⁴⁹ Unlike the usual accounts of aphasia in circulation at the time, Wernicke's patients had little trouble speaking or articulating words. Their main

⁴⁵ "Hier aber sah die Kranke *nichts*, hier waren gewissermaßen für die äußeren Wahrnehmungen die beiden Augen in zwei große blinde Flecke verwandelt. Analoge Zustände finden sich ja auch bei andern Gehirnläsionen: so gibt es hemiplegische Kranke mit Verlust der Bewegungsgefühle, wechle nicht wissen, dass sie gelähmt sind." It is worth noting that Anton includes, along with the cases of unawareness of blindness and deafness, also cases of the unawareness of hemiplegia, or partial paralysis. The term "*Nichtssehen*" was coined by the physiologist Johannes Müller to distinguish it from seeing "blackness" or "*Schwarzsehen*." Anton, "Blindheit nach beiderseitiger Gehirnkrankung mit Verlust der Orientierung im Raume," 45; M. Dufour, "Sur la vision nulle dans l'hémianopsie," *Revue médicale de la Suisse romande* 9 (August 20, 1889): 445–51; R.A. Sorensen, *Seeing Dark Things: The Philosophy of Shadows* (Oxford: Oxford University Press, 2008).

⁴⁶ Aristotle, *De Anima*, ed. David Ross (Oxford: Clarendon, 1961); For a lively reading of Aristotle's views on sensation, see, Daniel Heller-Roazen, *The Inner Touch: Archaeology of a Sensation* (New York: Zone Books, 2007), chap. 2–3, *passim*.

⁴⁷ Smith, *Inhibition*, chap. 4, *passim*.

⁴⁸ Otto M. Marx, "Nineteenth-Century Medical Psychology: Theoretical Problems in the Work of Griesinger, Meynert, and Wernicke," *Isis* 61, no. 3 (1970): 364–370; Harrington, *Medicine, Mind, and the Double Brain*, 71–73; Harrington, "Beyond Phrenology: Localization Theory in the Modern Era," 210–212; Guenther, "A Body Made of Nerves: Reflexes, Body Maps and the Limits of the Self in Modern German Medicine," chap. 2.

⁴⁹ Carl Wernicke, *Der aphasische Symptomencomplex: eine psychologische Studie auf anatomischer Basis* (Breslau: Cohn & Weigert, 1874); Carl Wernicke, "The Aphasical Symptom-Complex: A Psychological Study on an Anatomical Basis (translated)," ed. R.S. Cohen and Marx W. Wartofsky, *Proceedings of the Boston Colloquium for the Philosophy of Science*, Boston Studies in the Philosophy of Science, 4 (1969): 34–97.

problem, instead, was listening to and understanding what was spoken. It was not because they were deaf. They could hear sounds, but they could not recognize what sounds signaled words. In that sense, they heard the words, but they were “deaf” to the meaning.⁵⁰

That was the linchpin in Wernicke's report. His patients were uncomprehending, in effect, unconscious of speech, but speech alone. They could not understand what was spoken to them, and, moreover, they could not understand what was spoken *by them*. That is, they did not even realize the nonsense of their own jumbled words. It was as if a highly circumscribed part of consciousness were missing—the part for “speech perception,” so to speak, and along with it, “illness perceptions.”⁵¹ The patients with sensory aphasia failed to realize they had sensory aphasia because they could not understand their own speech. They did not realize that their words were no longer coherent words, just garbled sounds. In effect, such “loss of understanding” [*Mangel der Verständniss*] looked like the loss of hearing.⁵² The sound of words *as words* never made itself “heard” or “called out to consciousness” [*ins Bewusstsein gerufen werden könnten*], and so patients did not “hear” the errors in their own speech.⁵³ They never

⁵⁰ Anne Harrington, “The Brain and Behavioral Sciences,” in *The Cambridge History of Science*, ed. Peter Bowler and John Pickstone, vol. 6 (Cambridge ; New York: Cambridge University Press, 2009), 512.

⁵¹ The term “*Krankheitswahrnehmungen*” Anton would later employ for his interpretation of Wernicke’s aphasia. Anton, “Über Herderkrankungen des Gehirns die vom Patientin selbst nicht wahrgenommen werden,” 227.

⁵² He recounted how other staff members in the clinic assumed one patient with this type of aphasia was deaf because she understood nothing that was said to her. Wernicke, *Der aphasische Symptomencomplex*, 44.

⁵³ “Abgesehen von dem Mangel an Verständniss hat der Kranke also noch aphasische Erscheinungen beim Sprechen, bedingt durch das Fehlen dieser unbewussten von dem Lautbild geübten Correctur.” *Ibid.*, 23;

“sensed” the nature of their problem because the problem was in part their lack of sense—the sense of what words sounded like, or what Wernicke called their “sound-images” [*Klangbilder*].⁵⁴ This loss of sensory awareness, which entailed to some extent a loss of self-awareness, was intrinsic to sensory aphasia. It was based on “circumscribed” lesions in the brain, specifically, a “focal softening” [*Erweichungsheerd*]⁵⁵ of cerebral tissue in the first convolution, or outfolding, of the cortex in the left temporal lobe.⁵⁶ That was what finally “focused” the disease entity as well as the diagnosis, combining them as both “focal symptom of aphasia” [*Heersymptom der Aphasie*] and “focal disease of the brain” [*Heerderkrankung des Gehirns*], henceforth to be called “Wernicke’s aphasia.”⁵⁷

Yet again, as with Pick, Anton took note of Wernicke's work and incorporated it into his own. When in 1898 he first introduced the case of Juliane Hochrieser in the *Wiener klinische Wochenschrift* [*Vienna Clinical Weekly*], he drew direct parallels to Wernicke's cases. Both were, in a sense, “deaf” to the fact of their own disease.⁵⁸

Wernicke, “The Aphasic Symptom-Complex: A Psychological Study on an Anatomical Basis (translated),” 55.

⁵⁴ Wernicke, *Der aphasische Symptomencomplex*, 19–30, 21ff.

⁵⁵ *Ibid.*, 45.

⁵⁶ *Ibid.*, 46; Wernicke, “The Aphasic Symptom-Complex: A Psychological Study on an Anatomical Basis (translated),” 73; Norman Geschwind, “The Work and Influence of Wernicke,” *Boston Studies in the Philosophy of Science* 4 (1969): 1–33.

⁵⁷ Wernicke, *Der aphasische Symptomencomplex*, 46, 36.

⁵⁸ “Similar loss of illness-perceptions are proven in central disturbances of the acoustic system. In the often occurring disease of the first left temporal convolution, it becomes obvious that the individual does not understand words of language, and himself chooses false words in speech; this confused speech barely enters the sick man's consciousness, he lacks the self-correction and also the correct appraisal of how seriously he is affected. It happens that such sick people with sensory aphasia at examination, which

Wernicke's patients could not understand speech, including their own, which meant that they could not “hear” their aphasia. Anton's patient Hochrieser was in fact deaf to sounds, but she was also “mentally deaf to her deafness.”⁵⁹ Like Wernicke, Anton drew explanatory force from this comparison of consciousness to sensation. Not only did it help him illustrate the specific nature of his patient's loss, but it also helped him advance his claim that neither patient, Merz nor Hochrieser, was confused to any “sufficient degree.” Instead, their “confusion,” if one insisted, was strictly sensory, and yet mental. Merz was “mentally blind to her blindness.” Hochrieser was “mentally deaf to her deafness.” Each lacked the “sense” of her own lack of sensation. This was not a question of general confusion. It was a question of specific unawareness—like a blind spot, except in the proverbial “mind’s eye.”

According to Wernicke, the difference between such specific brain deficit and general mental illness was not hard to see, at least not for a doctor with “psychiatric training.”⁶⁰ That was in part because almost every German-speaking doctor who was “psychiatrically trained” [*psychiatrisch Gebildeten*], including Anton, ascribed to a model

from the serious attack have no idea and also no conclusions or thoughts at all of their verbal understanding, nor tie any affects to it. It appears that many sick people hold no answer in their questions and addresses, such acoustical additions and continuations of thought processes are neither anticipated nor missed. In other words, there are aphasias, which not sensed and not correctly judged by the sick man himself.” (my translation) Anton, “Über Herderkrankungen des Gehirns die vom Patientin selbst nicht wahrgenommen werden,” 227.

⁵⁹ Anton, “Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit,” 119.

⁶⁰ “Dass solche beweisende Fälle bisher noch nicht beobachtet, oder wenigstens nicht publicirt worden sind, beruht ausser auf der Seltenheit der Faelle an sich darauf, dass selbst durchweg erfahrene und intelligente Ärzte diesen Zustand fuer Verwirrtheit ansehen--wie ich selbst zu erfahren Gelegenheit hatte. Fuer den psychiatrisch Gebildeten, der die Formen der Verwirrtheit kennt, hat die Diagnose nicht die geringste Schwierigkeit.” Wernicke, *Der aphasische Symptomencomplex*, 24; Wernicke, “The Aphasic Symptom-Complex: A Psychological Study on an Anatomical Basis (translated),” 53.

of the mind that viewed consciousness as a fissile aggregate of discrete sensations.⁶¹ Perhaps it was not hard for a “psychiatrically trained” physician to spot the difference between general confusion and the discrete loss of sensory awareness, but that was mostly because his clinical vision was already primed and ready to perceive the difference.

All three psychiatrists, Wernicke, Pick, and Anton, would have shared this clinical vision, if for one important reason that they all shared the same mentor in Theodor Meynert (1833-1892), professor of psychiatry at the University of Vienna, who emphatically taught them how to look upon the mind in this manner.⁶² When Anton left Pick's service in Prague at the end of 1886, he moved next to Vienna to work for Meynert, where he remained until 1891.⁶³ By then, both Wernicke and Pick had already served their own separate stints as assistants to the Vienna professor, as had a fellow physician, Sigmund Freud (1856-1938). In fact, Anton narrowly missed working alongside Freud in Meynert's clinic by one year.⁶⁴ Nevertheless, he, like the others, was considerably influenced by Meynert's anatomical and clinical acumen.⁶⁵ It was Meynert,

⁶¹ Harrington, “Beyond Phrenology: Localization Theory in the Modern Era,” 259; Marx, “Nineteenth-Century Medical Psychology.”

⁶² Ackerknecht, *Kurze Geschichte der Psychiatrie*, 69.

⁶³ Pfeifer, “Gabriel Anton,” 186–187.

⁶⁴ Freud left Meynert's clinic in the fall of 1885, and Anton joined the clinic in the winter of 1887. Ellenberger, *The Discovery of the Unconscious*, 434–436.

⁶⁵ Anton looked back upon the years assisting in Meynert's clinic as his most formative period of medical training. The bond between the two was especially strengthened by the fact that Anton's daughter later married Meynert's grandson. Hartmann, “Gabriel Anton zum 70. Geburtstag,” 1506–1507; Anton believed that Meynert's influence on Freud was similarly extensive, in fact, more so than Freud himself

after all, who instilled in these younger psychiatrists the value of anatomical dissection to illuminate and, for that matter to validate, one's clinical judgment.⁶⁶ That was why Wernicke bothered to identify the specific area of damaged and “diseased” cortical tissue in the brains of his aphasic patients. It was also why Anton would later emphasize the similarly “focal” nature of his own patients' brain disease. They both reasoned that if such circumscribed brain damage, or focal lesions, could be isolated from the rest of relatively healthy cerebral tissue, then one could infer retrospectively that the patient's disorder had been likewise specific, discrete, and “pure.” That is, “pure” in the sense that the diagnosis ruled out other mental disturbances that would indicate a more general confusion or insanity. This style of reasoning in which the source of an illness could be isolated and pinpointed to a specific area of anomalous-looking tissue was called “organic localism” or “localization.”⁶⁷

The “doctrine of cerebral localization,” as it was more formally known, played an integral part in the making of Anton's syndrome as well as numerous other disease

acknowledged. Gabriel Anton, “Theodor Meynert. Seine Person, sein Wirken und sein Werk: Eine fachgeschichtliche Studie,” *Journal für Psychologie und Neurologie* 40 (1930): 274.

⁶⁶ Meynert emphasized that mental illness should be studied as if they were a “physiological experiment” to study the brain: “Man muss die Lehre von den Geisteskrankheiten zu einer vergleichenden Wissenschaft erheben, die Geisteskrankheiten als ein Experiment im Gehirne führen uns zu feineren und nicht minder sicheren Thatsachen, als unser physiologisches Experiment am Gehirne.” Theodor Meynert, *Sammlung von populär-wissenschaftlichen Vorträgen über den Bau und die Leistungen des Gehirns* (Wilhelm Braumüller, 1892), 220; Erwin H. Ackerknecht, *A Short History of Psychiatry* (Hafner Pub. Co., 1968); Ackerknecht, *Kurze Geschichte der Psychiatrie*; Erna Lesky, *Die wiener medizinische Schule im 19. Jahrhundert* (Graz: Böhlau, 1965); Erna Lesky, *The Vienna Medical School of the 19th Century* (Baltimore: Johns Hopkins University Press, 1976); Weisz, *Divide and Conquer*, 51; Eulner, *Die Entwicklung der medizinischen Spezialfächer an den Universitäten des deutschen Sprachgebietes*, 4:276.

⁶⁷ Robert M. Young, *Mind, Brain, and Adaptation in the Nineteenth Century: Cerebral Localization and Its Biological Context from Gall to Ferrier* (Oxford: Clarendon Press, 1970), 11; George Rosen, *The Specialization of Medicine with Particular Reference to Ophthalmology*, *Medicine & Society in America* (New York: Arno Press, 1972); Weisz, *Divide and Conquer*, 51.

entities in psychiatric medicine of the late nineteenth century.⁶⁸ Although it did not originate with Meynert, he was instrumental in refining it for clinical purposes and promoting it especially among psychiatrists in German-speaking regions of Europe. Because it was indispensable to Anton's formulation of this new "focal brain disease of self-perception," it will help to review some of its history.

The Doctrine of Cerebral Localization

In the early decades of the nineteenth century, disputes about the limits of science in matters of the soul, though long festering, erupted with special fervor when a new school of thought called "organology"—later "phrenology"—was developed by two Austrian physicians, Franz Josef Gall (1758-1828) and Johann Gaspar Spurzheim (1776-1832). The two traveled around lecturing on the so-called "faculties of the soul" and how they could be traced to specific compartments in the brain. Mental attributes like memory, the imagination, the passions, and even reason were subdivided, they argued, into discrete areas of tissue, or "organs," in the brain, which differed in size based on their relative strength, not unlike muscles of the body. Such an idea was not particularly savory to the more conservative Austrian authorities at the time, who forced Gall and Spurzheim out of Vienna until they ended up in Paris where, at least for a while, their ideas were more favorably received—until they came under new attack by a French physiologist named Pierre Flourens (1794-1867).⁶⁹

⁶⁸ Anne Harrington, "Psychiatrie und die Geschichte der Lokalisation geistiger Funktionen," *Nervenarzt* 60, no. 10 (1989): 603–11.

⁶⁹ Young, *Mind, Brain, and Adaptation in the Nineteenth Century*, chap. 1–2, passim; Roger Cooter, *The Cultural Meaning of Popular Science: Phrenology and the Organization of Consent in Nineteenth-Century*

Flourens did not have a problem with the idea of subdividing various functions in the brain. In fact, he had contributed to this theory of localization with his own research on pigeons and dogs, demonstrating that particular bodily functions like respiration were coordinated in parts of the brain stem. Where Flourens drew the line, however, was with *mental* functions and the traditional faculties of the soul. He reserved all of them, *tout court*, for the cerebrum, or the upper portion of the brain, and argued, contra Gall and Spurzheim, that it was impossible to circumscribe the functions any further. Despite the popularity of Gall's teaching, Flourens' argument was eventually favored among much of the academic establishment and helped slow most of the research on cerebral localization for the next several decades.⁷⁰

In 1860, however, the clinical interest in cerebral localization was revived when the French physician Paul Broca (1824-1880), who trained for a time in psychiatry, presented before the Society of Physical Anthropology new anatomical evidence. After consulting a series of reports from other French doctors, in particular Marc Dax (1771-1837) and Jean-Baptiste Bouillaud (1796-1881), Broca argued that there was an area of cerebral tissue in the left frontal lobe which was responsible for speech production. He discussed his recent examination of a patient, Monsieur Leborgne, who was nicknamed

Britain (Cambridge University Press, 1984), 1–15, passim; Harrington, *Medicine, Mind, and the Double Brain*, 7–9; Edwin Clarke and L. S. Jacyna, *Nineteenth-Century Origins of Neuroscientific Concepts* (Berkeley: University of California Press, 1987), 238–254; Hagner, *Homo cerebrialis*, 99–111, passim; Harrington, “The Brain and Behavioral Sciences,” 507–508.

⁷⁰ Young, *Mind, Brain, and Adaptation in the Nineteenth Century*, 67–71; Harrington, “Beyond Phrenology: Localization Theory in the Modern Era,” 207; Harrington, *Medicine, Mind, and the Double Brain*, 9–11; Hagner, *Homo cerebrialis*, 114–117; Pierre Flourens, *Examen de la phrénologie* (Paris: Paulin, 1842); Clarke and Jacyna, *Nineteenth-Century Origins of Neuroscientific Concepts*, 277–285.

“Tan” because he repeated the sound, “tan, tan, tan,” whenever he was asked a question.⁷¹ The critical point, according to Broca, was that Leborgne was not insane or delusional. In fact, he was capable of understanding most of what was said to him. It was just that he could not respond in any meaningful, coherent way. Broca took this to mean that Leborgne's language problem was a matter of *articulation*. He could still understand language, for his general intelligence was preserved. But he could no longer make the necessary movements with his lips, tongue, and jaw to say what he wanted to say. That was not because of some paralysis in the muscles of his face. Rather, it was due to Leborgne's loss of “verbal memory” of how to enunciate words.⁷² What he lacked was strictly the memory of how to *speak*, or actively to form audible words, and so Broca gave him the new diagnosis of “*aphémie*,” assuming it meant in ancient Greek “I speak.”⁷³

Broca was distinctly aware of the potential fallout from trying to localize in the brain something as significant as human language. After all, it was supposed to represent one of the primary virtues of human intelligence, one of the precious,

⁷¹ Hagner, *Homo cerebrialis*, 265–268; Harrington, *Medicine, Mind, and the Double Brain*, 35–48; Young, *Mind, Brain, and Adaptation in the Nineteenth Century*, 140–146; Paul Broca, “Remarques sur le siège de la faculté du langage articulé, suivies d’une observation d’aphémie (perte de la parole),” *Bulletins de la Société Anatomique* 36 (1861): 330–57; Paul Broca, “Perte de la parole. Ramollissement chronique et destruction partielle du lobe antérieur gauche du cerveau,” *Bulletins de la Société d’Anthropologie* 2 (1861): 235–38.

⁷² Harrington, *Medicine, Mind, and the Double Brain*, 44–45.

⁷³ Harrington points out that this was not what the term really meant, which is why it was soon changed to “*aphasie*.” So much for knowing the right words to name a language disorder. *Ibid.*, 43n4.

inviolable “faculties of the soul.”⁷⁴ Language was what distinguished humans from beasts, a distinction already under attack by Charles Darwin's theory of evolution, published just a year earlier.⁷⁵ And so, Broca tried to downplay the impact on general intelligence implied by Leborgne's disorder and to isolate the activity of articulation instead.⁷⁶ Conveniently for Broca, Leborgne died soon after he examined him, allowing for a fresh autopsy of the brain to confirm if indeed there were any signs of tissue damage in the area predicted. And sure enough, there were. Between the frontal and parietal lobes of the left cerebral hemisphere, the tissue was particularly soft and deformed. (There was also damage to other parts of his brain, but Broca discounted this.⁷⁷) In any case, the argument for cerebral localization was granted a new lease, cleared of its tarnished affiliation with Gall's phrenology, which Broca dismissed as the “system of bumps.”⁷⁸

Within just a few years of Broca's study, the young psychiatrist Theodor Meynert brought forward his own recent findings on aphasia. On February 9, 1866, Meynert gave a paper to the Imperial-Royal Society of Physicians in Vienna entitled, “A Case of Speech Disturbance, Anatomically Based.”⁷⁹ He described the case of a twenty-three year old

⁷⁴ Young, *Mind, Brain, and Adaptation in the Nineteenth Century*, 144; Harrington, *Medicine, Mind, and the Double Brain*, 50–51.

⁷⁵ Hagner, *Homo cerebrealis*, 235–236; Harrington, *Medicine, Mind, and the Double Brain*, 49.

⁷⁶ Harrington, *Medicine, Mind, and the Double Brain*, 45.

⁷⁷ *Ibid.*, 52–57.

⁷⁸ *Ibid.*, quoted on 44.

⁷⁹ Theodor Meynert, “Ein Fall von Sprachstörung anatomisch begründet,” *Medizinische Jahrbücher* 22 (1866): 152–87.

servant girl, who showed particular trouble with language. She was unable to repeat after him words like “head” [*Kopf*] and “hand” [*Hand*], substituting it with the word “yellow” [*gelb*]. She also mispronounced the word “coughing” [*Hutzen*], making a nonsensical sound, “*Husten*,” instead.⁸⁰ Initially, Meynert described her as having a “defective articulation” and “inhibition of verbal expression,” terms very similar to Broca's description of Leborgne.⁸¹ But there was something else he observed in the young woman's speech. Occasionally, she peppered it with “erroneous expressions which bore absolutely no relation to what she was trying to say.”⁸² That was because, Meynert believed, these mistakes had “escaped” her attention.⁸³ She made them unwittingly, as if she never heard them, as if she were unaware of this aspect of her condition.

When she died, Meynert dissected her brain⁸⁴ and found, in addition to lesions in Broca's area, damage to another, new structure Meynert named the “acoustic cord”

⁸⁰ *Ibid.*, 154–155.

⁸¹ *Ibid.*, 154; Theodor Meynert, “Anatomische Begründung gewisser Arten von Sprachstörungen,” *Österreichische Zeitschrift für praktische Heilkunde*, February 16, 1866, 199.

⁸² “[Sie]...einzelner zur Mittheilung nöthiger Wörter nicht habhaft werden konnte, z.B. *Kopf*, *Hand*, während ihr zum Ersatze der fehlenden Ausdrücke Wörter entschlüpften, die sich mit der beabsichtigten Mittheilung keineswegs deckten, so dass sie das Wort *gelb* für *Hand* produciren musste.” Meynert, “Ein Fall von Sprachstörung anatomisch begründet,” 1866, 154.

⁸³ One of the definitions for *entschlüpften* in the German dictionary *Duden Online* is “*unbedacht geäußert werden*,” literally, “to be expressed unthinkingly.” “Entschlöpffen,” <http://www.duden.de/suchen/dudenonline/entschl%C3%BCpfen>.

⁸⁴ The historian of science Michael Hagner interprets this as evidence that Meynert was “obviously less interested in the young woman's symptoms than he was in her brain.” Similarly, the historian of medicine Stephen Jacyna has broadly argued that those like Meynert typically treated the patients' lives as prologue and the brain autopsy as “climax” in their case histories. However, it is questionable how much one can infer Meynert's personal *interest* in his patient on the basis of how many pages he chose to write

[*Acusticusstrang*], connecting the “auditory labyrinth” [*Gehörslabyrinth*] of the inner ear to the “auditory cortex.”⁸⁵ Meynert reasoned from this anatomical evidence that the patient's aphasia involved “sensory images” of words which allowed for the auditory comprehension of language. According to Meynert, the sounds of words were first absorbed and registered as “sensory impressions” [*Sinneseindrücke*] in nerve endings that lined the auditory labyrinth of the inner ear. These sensations were converted into “sound-images” [*Klangbilder*] and transmitted to the cortex along the tract of nerve fibers in the “acoustic cord.” Once they reached the cortex, specifically the temporal cortex, they were stored along the inside wall of the Sylvian fissure in a region he called the “sound-field” [*Klangfeld*].⁸⁶ From there, they could cross the “threshold of consciousness” [*Schwelle des Bewusstseins*] to become auditory perceptions. However, the mere perception of speech was not the same as comprehension. To understand the meaning of spoken words required that their “sound-images” be combined, or “associated,” with their “memory-images” [*Erinnerungsbilder*] lodged in the temporal

about her symptoms. Neither Hagner nor Jacyna consider the possibility that Meynert's way of writing and his divided attention to brain anatomy reflected the attempt of a young doctor to conform to the norms of academic medicine at that time, particularly the norms of clinical examination and the mode of presenting research, rather than an unequivocal or “obvious” [*offensichtlich*] lack of *interest* in one's patients. Hagner, *Homo cereбрalis*, 269; Jacyna, *Lost Words: Narratives of Language and the Brain, 1825-1926*, 42, 52.

⁸⁵ Theodor Meynert, “Ein Fall von Sprachstörung anatomisch begründet,” *Allgemeine Wiener medizinische Zeitung* 11, no. 9 (February 28, 1866): 68; Alois Höfler, *Worte der Erinnerung an Theodor Meynert und an sein Verhältniss zur philosophischen Gesellschaft an der Universität zu Wien* (Wien: W. Braumüller, 1892).

⁸⁶ Meynert, “Ein Fall von Sprachstörung anatomisch begründet,” 1866, 152, 166–167, 182; Meynert, “Ein Fall von Sprachstörung anatomisch begründet,” February 28, 1866, 68; Hagner, *Homo cereбрalis*, 269–270; H.A. Whitaker and S.C. Etlinger, “Theodor Meynert's Contribution to Classical 19th Century Aphasia Studies,” *Brain and Language* 45, no. 4 (November 1993): 560–71; Paul Eling, “Meynert on Wernicke's Aphasia,” *Cortex* 42 (2006): 811–16.

cortex.⁸⁷ Should this part of the cortex be damaged or diseased, words may still be heard but they were no longer understood.⁸⁸

Such emphasis on perception in connection to understanding belonged to an older tradition in philosophy and psychology known as “sensationism” or “associationism.”⁸⁹ Initially outlined in the seventeenth century by English philosopher John Locke (1632-1704) in *An Essay Concerning Human Understanding*,⁹⁰ sensationism went through numerous modifications⁹¹ before Meynert adapted it to his purposes in the mid-nineteenth century.⁹² But basically, the emphasis remained throughout that everything about the human mind, including consciousness, the idea of the self, and

⁸⁷ Meynert used the terms “threshold of consciousness” and “memory-images” coined by the physicist-turned-philosopher Gustav Fechner (1801-1887). Gustav Theodor Fechner, *Elemente der Psychophysik* (Leipzig: Breitkopf und Härtel, 1860); Here is Fechner in a poignant passage describing what he meant by the threshold of consciousness: “Each of us himself notices that at any given moment an incomparably smaller amount of knowledge, thoughts, and desires actually occupies our mind, than could be available if prompted. This absent, albeit not distant knowledge, this persistent knowledge that we have—in what state is it within us?...Two thoughts are sufficient to entirely expel a third from consciousness, and to produce an entirely different state of mind....Just as we are accustomed to speaking of thoughts entering consciousness, I call the boundary that a thought seems to cross when it changes from being an entirely restrained state to the state of manifesting a degree of real thought: the threshold of consciousness.” Michael Heidelberger, *Nature from within: Gustav Theodor Fechner and His Psychophysical Worldview* (Pittsburgh: University of Pittsburgh Press, 2004), quoted on 33.

⁸⁸ Theodor Meynert, “Neue Untersuchungen über den Bau der Grosshirnrinde und seine örtlichen Verschiedenheiten. Vortrag, gehalten in der Sitzung der k.k. Gesellschaft der Ärzte am 20. November 1868,” *Medizinische Jahrbücher* 17 (1869): 23.

⁸⁹ Young, *Mind, Brain, and Adaptation in the Nineteenth Century*, 94–113; Harrington, “Beyond Phrenology: Localization Theory in the Modern Era,” 209–211.

⁹⁰ John Locke, *An Essay Concerning Human Understanding* (London: Printed by Eliz. Holt for Thomas Basset, 1690).

⁹¹ Goldstein, *The Post-Revolutionary Self*, chap. 1, 3; George S. Rousseau, *Nervous Acts: Essays on Literature and Sensibility* (Palgrave Macmillan, 2005), 83–111; Charles Taylor, *Sources of the Self: The Making of the Modern Identity* (Cambridge, Mass: Harvard University Press, 1989), chap. 9.

⁹² Meynert referred to the cortex and its “provinces” as a “physiological *tabula rasa*” to borrow from Locke. Meynert, “Ein Fall von Sprachstörung anatomisch begründet,” 1866, 179.

even the basis of personal identity, all originated in sensory experience, which could be traced from the sense organs like the eyes and ears, through the peripheral nerves of the body, and up into the brain. However, Meynert took experience even farther. He traced “sense-perception” [*Sinneswahrnehmung*] all the way to the outermost reaches of the brain, into the “hollow dome” [*Hohlkugel*] of the cortex.⁹³ There, he intimated, lay the answer to what made up the soul.⁹⁴

Driving this conviction was Meynert's particular method of studying brain anatomy, a technique of dissection known as “*Abfaserungsmethode*,” or the “method of splaying fibers,” which he passed on to Wernicke and Anton as well.⁹⁵ The technique, adapted from Karl Friedrich Burdach (1776-1847), involved pulling apart the “tracts” or “bundles” of fibrous tissue beneath the cortical surface of the brain.⁹⁶ Meynert used it

⁹³ Meynert, “Neue Untersuchungen über den Bau der Grosshirnrinde und seine örtlichen Verschiedenheiten. Vortrag, gehalten in der Sitzung der k.k. Gesellschaft der Ärzte am 20. November 1868,” 15.

⁹⁴ Although Anton agreed with Wernicke's tribute to Meynert that he “animated” or “en-souled” the brain [*Gehirn beseelt*], Kiefer's wry appraisal is probably more apt, namely, that Meynert “neurologized” or “em-brained” the soul [*Seele behirnt*]. Anton, “Theodor Meynert. Seine Person, sein Wirken und sein Werk: Eine fachgeschichtliche Studie,” 126; Mathias Kiefer, *Die Entwicklung des Seelenbegriffs in der deutschen Psychiatrie ab der zweiten Hälfte des 19. Jahrhunderts unter dem Einfluss zeitgenössischer Philosophie* (Essen: Verlag Die Blaue Eule, 1996), 36; T. Seli and C.M. Shapiro, “Neuropsychiatry--the Mind Embrained?,” *Journal of Psychosomatic Research* 43, no. 4 (October 1997): 329–33.

⁹⁵ Anton, “Theodor Meynert. Seine Person, sein Wirken und sein Werk: Eine fachgeschichtliche Studie,” 271; Wernicke, “The Aphasic Symptom-Complex: A Psychological Study on an Anatomical Basis (translated),” 38, 40.

⁹⁶ Guenther discusses in rich detail Meynert's method of dissection in her dissertation, *A Body Made of Nerves* (see especially Chapter 1). Incidentally, I have selected a different translation of *Abfaserung* from Guenther's chosen term, “cleavage,” in order to stress the string- or thread-like character of the fibers—the substance of “associationist” theory—being unraveled and unwoven, whereas cleavage more generally implies the separation of chunks of tissue. Guenther, “A Body Made of Nerves: Reflexes, Body Maps and the Limits of the Self in Modern German Medicine,” chap. 1; Cornelius Borck, “Fühlfäden und Fangarme. Metaphern des Organischen als Dispositiv der Hirnforschung,” *Ecce Cortex. Beiträge zur Geschichte des modernen Gehirns*, 1999, 144–76; Hagner, *Homo cerebrialis*; Meynert, *Sammlung von populär-wissenschaftlichen Vorträgen über den Bau und die Leistungen des Gehirns*, 361.

to delineate individual “pathways” [*Bahnen*] of fiber-tracts as they wound their way, or “projected,” from the spinal cord through the “pithy core” [*innere Gewebe*] of the midbrain and culminated in the outer layer, or “rind,” of the cortex [*Hirnrinde*].⁹⁷ This way of cutting into the brain's fibrous matter along what appeared to be natural “routes” helped him to visualize the function of the nervous system in a novel way. Now, not only was the cortex, or outer surface of the brain, mappable like a semi-spherical globe, but it was also *mineable* like a tunneled cave.⁹⁸ This was a new and more sophisticated theory of anatomical localization than either the phrenology of Gall or the surface localization of Broca. With Meynert's emphasis on pathways and fibers, he not only paved the way to a more penetrating localization of brain function but also a more daring anatomy of awareness.⁹⁹

Not long after, the physiologists began to catch up with the advances spearheaded by clinicians like Broca and Meynert.¹⁰⁰ Eduard Hitzig (1838-1907) and

⁹⁷ Many of the terms Meynert and others used for the cortex, subcortex, and spinal cord had distinct undertones of a pulpy, fibrous fruit. For instance, the cortex was the “*Rinde*” or “rind,” the subcortical structures were collectively the “*innere Gewebe*” or “inner pith/web/core,” and the spinal cord was called the “*Rückenmark*” or “posterior core/pith.”

⁹⁸ For an excellent historical and philosophical discussion of changing modes of visual representation of the brain, see Robert L. Martensen, *The Brain Takes Shape: An Early History* (Oxford: Oxford University Press, 2004).

⁹⁹ Meynert, “Ein Fall von Sprachstörung anatomisch begründet,” 1866, 153; Meynert, “Neue Untersuchungen über den Bau der Grosshirnrinde und seine örtlichen Verschiedenheiten. Vortrag, gehalten in der Sitzung der k.k. Gesellschaft der Ärzte am 20. November 1868”; Theodor Meynert, *Psychiatrie: klinik der Erkrankungen des Vorderhirns begründet auf dessen Bau, Leistungen und Ernährung* (W. Braumüller, 1884); Theodor Meynert, “Das Zusammenwirken der Gehirnteile,” *Verhandlungen des X. Internationalen medicinischen congresses, Berlin, 4.-9. august 1890.*, 1892 1890, 173–90.

¹⁰⁰ Michael Hagner, “Die elektrische Erregbarkeit des Gehirns,” in *Die Experimentalisierung des Lebens: Experimentalsysteme in den biologischen Wissenschaften 1850-1950* (Berlin: Akademie Verlag, 1993), 115n50; Ackerknecht, “Gudden, Huguenin, Hitzig. Hirnpsychiatrie im Burghölzli 1869-1879,” 66;

Gustav Fritsch (1838-1927), while stationed as military physicians in Nancy during the Franco-Prussian War, confirmed what Meynert and Broca had earlier suggested, namely, that the cerebrum played a role in more elementary sensory-motor functions. When they electrically stimulated the exposed cortex of research animals, they were able to elicit jerking and twitching movements, from which they concluded that even the highest anatomical structure of the brain was involved in the more basic functions of bodily sensation and movement. No longer could it be defended, as Flourens had wanted, that the cerebrum was indivisible or that its function, in mimicking the alleged unity of the soul, was uniform. Now, the once-vaunted cerebrum could be carved up and broken down into “circumscribed centers.”¹⁰¹ Thanks to Hitzig and Fritsch, there was a new zeal among physiologists to localize cerebral functions, so much so that many credited them for ushering in a “new era” of brain research.¹⁰²

Among the first physiologists who followed Fritsch and Hitzig's lead was Hermann Munk (1839-1912), who from 1877 to 1880 experimented on the brains of dogs and rats, selectively destroying portions of the cortex in search of the specific origins of sensory and motor functions.¹⁰³ From these painstaking, and potentially

Constantin von Monakow, *Fünfzig Jahre Neurologie: zwei Vorträge* (Zürich: Art. institut O. Füssli, 1924), 5n1.

¹⁰¹ Harrington, “Beyond Phrenology: Localization Theory in the Modern Era,” 211–212; Gustav T. Fritsch and Eduard Hitzig, “Über die elektrische Erregbarkeit des Grosshirns,” *Archiv für Anatomie, Physiologie und wissenschaftliche Medizin* 37 (1870): 300–332; Young, *Mind, Brain, and Adaptation in the Nineteenth Century*, chap. 7, passim.

¹⁰² Monakow, *Fünfzig Jahre Neurologie*, 11.

¹⁰³ Hermann Munk, *Über die Functionen der Grosshirnrinde: Gesammelte Mittheilungen aus den Jahren 1877-80. Mit Einleitung und Anmerkungen* (Berlin: Verlag von August Hirschwald, 1881); Stanley Finger,

painful, experiments, Munk claimed to have localized the area of the cortex responsible for vision. After surgically removing large sections of the posterior, occipital lobe from a dog's cortex, he produced a form of blindness he designated as “cortical blindness” [*Rindenblindheit*]. Similarly, after he damaged another portion of the occipital cortex in a second research animal, he produced a different kind of blindness he called “mind-blindness” [*Seelenblindheit*].¹⁰⁴ When the second dog recovered from the surgery, it could still see, that is, crudely register visual stimuli, but it no longer seemed to recognize what it saw.¹⁰⁵ Although Munk's findings were contested for years,¹⁰⁶ he still managed to spark the imagination of many physicians, including Anton.¹⁰⁷ He adopted Munk's “*Seelenblindheit*” and adapted it in an evocative turn of phrase to describe his

Origins of Neuroscience: A History of Explorations into Brain Function (Oxford: Oxford University Press, 2001), 87–88.

¹⁰⁴ Munk, *Über die Functionen der Grosshirnrinde*, 29–30.

¹⁰⁵ “Durch die Extirpation ist der Hund *seelenblind* geworden, d.h. er hat die Gesichtsvorstellungen, welche er besass, seine Erinnerungsbilder der früheren Gesichtswahrnehmungen, verloren, so dass er nichts kennt oder erkennt, das er sieht; aber der Hund sieht, die Gesichtsempfindungen kommen ihm zum Bewusstsein, kommen zur Wahrnehmung, und sie lassen Vorstellungen über die Existenz, die Form, die Lage der äusseren Objecte entstehen, so dass von neuem Gesichtsvorstellungen, von neuem Erinnerungsbilder der Gesichtswahrnehmungen gewonnen werden.” *Ibid.*, 29.

¹⁰⁶ William James, *The Principles of Psychology*, vol. 1 (1890; repr., New York: Dover Publications, 1918), 46–51, 46ff.

¹⁰⁷ Meynert, *Psychiatrie*, 135; Hermann Wilbrand, *Die Seelenblindheit als Herderscheinung und ihre Beziehungen zur homonymen Hemianopsie zur Alexie und Agraphie* (Wiesbaden: JF Bergmann, 1887); C.S. Freund, “Ueber optische Aphasie und Seelenblindheit,” *Archiv fuer Psychiatrie und Nervenkrankheiten* 20 (1889): 276–297; 375–416; Heinrich Lissauer, “Ein Fall von Seelenblindheit nebst einem Beitrag zur Theorie derselben,” *Archiv fuer Psychiatrie* 21 (1890): 222–70; Friedrich Müller, “Ein Beitrag zur Kenntniss der Seelenblindheit,” *Archiv fur Psychiatrie und Nervenkrankheiten* 24 (1892): 856–917; Mark Solms, Karen Kaplan-Solms, and Jason Brown, “Wilbrand’s Case of ‘Mind-Blindness,’” in *Classic Cases in Neuropsychology*, ed. Christopher Code, vol. 1, 2 vols. (Hove, East Sussex: Psychology Press, 1996), 89–110; Heinrich Lissauer, “A Case of Visual Agnosia with a Contribution to Theory,” trans. Marianne Jackson, *Cognitive Neuropsychology* 5, no. 2 (March 1988): 157 – 192.

patient, Ursula Mercz, and her singular loss of self-perception that made her effectively "mind-blind to her blindness."¹⁰⁸

And so, Anton built the idea of his disease entity on a number of others' ideas, not only about disease, but about perception, about consciousness, and about brain function. Localization became the central strategy which he, like Wernicke and Meynert and Broca before him, relied on to demonstrate and legitimate the hypothesis that what he had seen in his patients was real and worth writing about. It was not just another fleeting symptom amid the flora of confusion. No, Anton insisted, this was a new "focal disease of the brain" [*Herderkrankung des Gehirns*]¹⁰⁹—a lesion of awareness. It was specific, as a disease and as a diagnosis, susceptible to being pinpointed in tissue and recorded on photographic plates (see Figure 4). However, try as he might to draw the force of his argument from the precision of anatomy, this subjective disorder resisted any neat demarcation or clear definition.

¹⁰⁸ Anton, "Blindheit nach beiderseitiger Gehirnerkrankung mit Verlust der Orientierung im Raume," 45; Anton, "Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit," 104.

¹⁰⁹ Anton, "Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit."

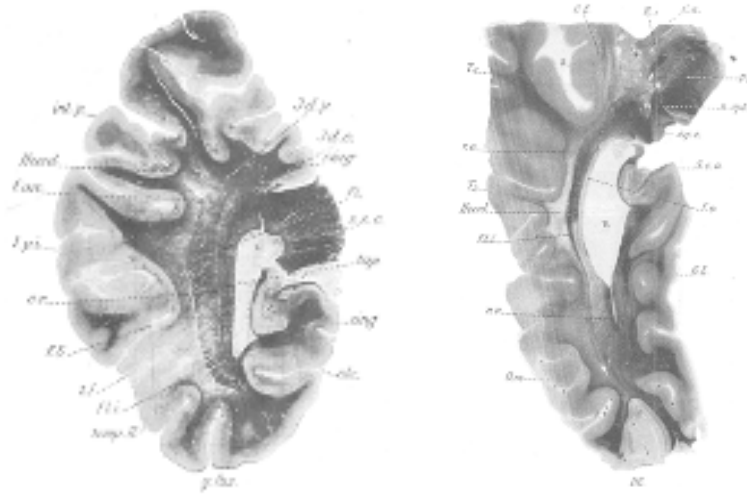


FIG. 2: "Figures 6.c and 7.e" from Tables 4 and 5, respectively, are photographic plates of slices taken from Ursula Mercz's brain.¹¹⁰

At the end of his paper, Anton reviewed his anatomical findings from Mercz and Hochrieser's dissected brains and acknowledged that the focal lesions were not as "focused" as he might have hoped. Where he expected, in the case of Mercz, for the tracts of nerve fibers running between the optical thalamus and occipital cortex to have been completely destroyed, he noticed instead that they were only partially severed. These residual fibrous connections suggested there may have been lingering "dark sensations" [*dunkle Empfindungen*], to borrow another phrase from Hermann Munk, which could have "masked" [*verdecken*] the loss of "conscious, cortical sense-perception" [*bewusste corticale Sinnewahrnehmung*], i.e. vision.¹¹¹ That possibility forced Anton to consider whether his patients might have retained, like Arnold Pick had so delicately described, a vestigial but "dim" insight into their illness after all.

¹¹⁰ *Ibid.*

¹¹¹ *Ibid.*, 122.

It cannot be ruled out with anatomical certainty that, in the three cases presented, a *dark feeling* might not be aroused along the central sensory pathways and *allow the individual to perceive the change in his condition*.¹¹²

Diagnostic certainty was elusive. Anatomical precision was no guarantee when it came to such a subjective disease entity as Anton's syndrome. Localization, even the more "reticular" localization promoted by Meynert and Wernicke, was not enough to weather the doubts over the value of dissection which were stirring among the younger ranks of German and Austrian psychiatrists.¹¹³ Such doubts were already public by the time Anton published his case histories in the late 1890s. To understand, however, where the criticism was coming from requires looking farther back to what gave the critics legitimacy in the first place. That means taking a closer look at what it meant to be in possession of the special knowledge and skills that Wernicke had so casually called "psychiatric training."¹¹⁴

The Rise of Academic Psychiatry

In 1865, psychiatry gained its first firm foothold in the academy. That year, Wilhelm Griesinger (1817-1868) was awarded the first chair in psychiatry at the University of Berlin.¹¹⁵ In addition, he was made director of the new "Section for

¹¹² Ibid., 121–122.

¹¹³ Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice*, 123; Harrington, *Reenchanted Science: Holism in German Culture from Wilhelm II to Hitler*, 78.

¹¹⁴ Wernicke, *Der aphasische Symptomencomplex*, 24.

¹¹⁵ Griesinger was not the obvious first choice, however. He was not especially versed in the study of mental disorders but rather focused initially on the study of infectious diseases. Like Rudolf Virchow and the Berlin "biophysicists," Griesinger embraced the "new physiology" in his clinical research. In 1842, he co-founded with Carl Wunderlich the *Archiv für physiologische Heilkunde*, which provided an important

Nervous Diseases and Mental Disorders” at the affiliate Charité Hospital. The combination was precisely as Griesinger had envisioned it since the early 1840s. His new role combined under the single rubric of psychiatry both the study of mental illnesses and brain diseases as well as absorbing into one role the responsibilities of both asylum director and clinical researcher. With this historic appointment, Griesinger set himself the task of putting psychiatry on track to becoming an academic discipline and clinical specialty as well as a legitimate part of “scientific medicine.”¹¹⁶

The notion of “scientific medicine” was not new. It had been in circulation since at least the early eighteenth century.¹¹⁷ But one of the differences in the 1860s was new funding.¹¹⁸ The same year that Griesinger was called to Berlin, the physiologist Carl

outlet for combining experimental science with clinical medicine. In 1845, he turned his attention to the study of mental illness in a textbook which he revised and expanded in 1861 to wider critical acclaim. And so, when the Ministry of Education decided to create a new chair in psychiatry, Griesinger's candidacy was particularly favored by Virchow and Du Bois-Reymond, who hoped he lead psychiatry into the "new era" of "scientific medicine." Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice*, 66–69; Eric J. Engstrom, “Neurowissenschaften und Hirnforschung,” in *Geschichte der Universität zu Berlin 1810-2010. Praxis ihrer Disziplinen, Transformation der Wissensordnung*, ed. Heinz-Elmar Tenorth, vol. 5 (Berlin: Akademie Verlag, 2010), 780; Eulner, *Die Entwicklung der medizinischen Spezialfächer an den Universitäten des deutschen Sprachgebietes*, 4:260; Ackerknecht, *Kurze Geschichte der Psychiatrie*.

¹¹⁶ Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice*, 58–61; Weisz, *Divide and Conquer*, 5; Timothy Lenoir, “Science for the Clinic: Science Policy and the Formation of Carl Ludwig’s Institute in Leipzig,” in *The Investigative Enterprise: Experimental Physiology in Nineteenth-Century Medicine*, ed. William Coleman and Frederic Lawrence Holmes (Berkeley: University of California Press, 1988), 160; Bettina Wahrig-Schmidt, *Der junge Wilhelm Griesinger im Spannungsfeld zwischen Philosophie und Physiologie: Anmerkungen zu den philosophischen Wurzeln seiner frühen Psychiatrie* (Tübingen: Narr, 1985).

¹¹⁷ John Harley Warner, *The Therapeutic Perspective: Medical Practice, Knowledge, and Identity in America, 1820-1885*, 2nd ed. (Cambridge, MA: Harvard University Press, 1986; Princeton, NJ: Princeton University Press, 1997), 247–248, 261ff.; John Harley Warner, “Science in Medicine,” *Osiris* 1 (1985): 37–58; Timothy Lenoir, *The Strategy of Life: Teleology and Mechanics in Nineteenth-Century German Biology* (Chicago: University of Chicago, 1989); William Coleman and Frederic Lawrence Holmes, *The Investigative Enterprise: Experimental Physiology in Nineteenth-Century Medicine* (University of California Press, 1988).

¹¹⁸ Cf. Coleman and Holmes, *The Investigative Enterprise*.

Ludwig (1816-1895) founded in Leipzig the first research institute dedicated to fostering connections between physiological science and clinical medicine.¹¹⁹ Ludwig's institute became what one historian called a “prototype for new physiological institutes.”¹²⁰ That was because he was able to align his institute's goals, and more broadly his disciplinary ambitions, with the changing needs of the modern industrializing state across a range of fields, including medicine, the military, and agriculture. And so, similarly Griesinger hoped to promote one such prototype for psychiatry.¹²¹

He began by seeking stronger institutional ties with universities, which would in time raise psychiatry to a full-fledged academic discipline with its own department, clinic, and laboratories. He lobbied for policy reform that relocated asylums from the countryside to urban universities.¹²² Once part of the university, the asylum was to become a research clinic, not unlike the model of Ludwig's institute in Leipzig. The psychiatric clinic, also modeled on other specialty clinics attached to the university, usually came with its own laboratory space, lecture halls, library, and assistants. The director of the clinic, often the former director of the asylum, would then be appointed

¹¹⁹ It was the same year that the French physiologist Claude Bernard argued that the medical ideas of health and disease were not absolutely different but rather connected along a relative spectrum of physiological indices, cumulatively called “*le milieu intérieure*.” Claude Bernard, *Introduction à l'étude de la médecine expérimentale* (Paris: J. B. Baillière et fils, 1865).

¹²⁰ Lenoir, “Science for the Clinic: Science Policy and the Formation of Carl Ludwig’s Institute in Leipzig,” 145.

¹²¹ Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice*, 54–58.

¹²² *Ibid.*, 54–57.

to chair his own academic department on the medical faculty.¹²³ This was Griesinger's ideal, and it became a reality with the creation of at least eight new psychiatry departments and clinics across Germany as well as the assimilation of nearly a hundred smaller city asylums into university hospitals.¹²⁴ Griesinger's reforms generated the institutional momentum to help psychiatry achieve parity with other specialties in internal medicine.¹²⁵ In effect, he paved the way for the rise of a new kind of psychiatrist with one foot in the autopsy room and the other in the clinical ward.¹²⁶

Although Griesinger helped clear the way for psychiatry on the university faculty, it was Theodor Meynert of Vienna who put into practice what Griesinger preached.¹²⁷ In

¹²³ Weisz, *Divide and Conquer*, 53–54.

¹²⁴ F. Nissl, "Über die Entwicklung der Psychiatrie in den letzten 50 Jahren," *Verhandlungen des Naturhistorisch-Medizinischen Vereins* 8 (1908): 511.

¹²⁵ Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice*, 122–123, 126.

¹²⁶ Griesinger's appointment may have inaugurated a new way of doing psychiatry but not yet a new name. The journal Griesinger founded just before he died in 1868 offers a clue to this "nominal" confusion. He called it the *Archiv für Psychiatrie und Nervenkrankheiten* [Archive for Psychiatry and Nervous Diseases]. In part thanks to the journal's success, as new academic chairs and clinics in "psychiatry and nervous diseases" were created, that simple but troublesome conjunction "and" spread along with them throughout the German-speaking medical world. Trivial as it may have sounded, that "and" was an important testimony to the fact that psychiatrists continued to struggle with their as-yet-unsettled professional identity. Did the "and" mean that psychiatry was separate from the study of brain diseases? Or did it mean that the two were synonymous? Did the order of putting psychiatry before nervous diseases signal that one was subordinate to the other? It was not clear.¹ In other words, the verbal glue did not hold. No matter how the study of mental illness and brain disease was supposed to be combined into a so-called "double discipline" [*Doppelfach*], every feat of rhetoric to rename or repackage it could not but accentuate the problem, namely, that no one knew quite how to overcome the gap between the mind and brain. Ackerknecht, "Gudden, Huguenin, Hitzig. Hirnpsychiatrie im Burghölzli 1869-1879," 68; Wilhelm Griesinger, *Die Pathologie und Therapie der psychischen Krankheiten: für Aerzte und Studierende*, 2. umgearb. und sehr verm. Aufl. (Stuttgart: Adolph Krabbe, 1861); Eberhard Gabriel, "Zur Beziehung zwischen Psychiatrie und Psychotherapie in Wien im 20. Jahrhundert - Eine psychiatriegeschichtliche Einführung zu ihrer Entwicklung um die Jahrhundertwende," in *Gründe der Seele: Die Wiener Psychiatrie im 20. Jahrhundert*, ed. Brigitta Keintzel and Eberhard Gabriel (Wien: Picus, 1999), 15.

¹²⁷ Gröger, "Zur Entwicklung der Psychiatrie in der Wiener Medizinischen Schule," 34–35.

1861, he earned his medical degree from the University of Vienna, then quickly became an assistant physician at the new Imperial Asylum [*“die k.k. Irren-Heil- und Pflege-Anstalt”* or the *“niederösterreichische Landesirrenanstalt”*] in Vienna and later its prosector, performing autopsies on the brains of the deceased. In 1865, when Griesinger took up his chair of psychiatry in Berlin, Meynert joined the medical faculty in Vienna as a young docent lecturing on the *“Structure and Function of the Brain and Spinal Cord,”* which he based on his autopsy research at the state asylum.¹²⁸ Meynert moved with such ease between the asylum, laboratory, and lecture hall that he practically embodied Griesinger's ideal type of psychiatrist.¹²⁹

Yet, like Griesinger, Meynert was not trained in psychiatry. His background was in pathological anatomy, which he learned while in medical school under Carl von Rokitansky (1804-1878) who had, along with the Prussian pathologist Rudolf Virchow (1821-1902),¹³⁰ practically introduced the discipline to the medical curriculum.

Rokitansky became the dean of Vienna's medical school in 1849 and then the university

¹²⁸ Eulner, *Die Entwicklung der medizinischen Spezialfächer an den Universitäten des deutschen Sprachgebietes*, 4:276; Lesky, *The Vienna Medical School of the 19th Century*, 159; T. Meißel, “Freud und die österreichische Psychiatrie seiner Zeit,” in *Psychoanalyse und Psychiatrie: Geschichte, Krankheitsmodelle und Therapiepraxis*, ed. Heinz Böker (Berlin: Springer, 2006), 54.

¹²⁹ Anton, “Theodor Meynert. Seine Person, sein Wirken und sein Werk: Eine fachgeschichtliche Studie,” 267; William M Johnston, *The Austrian Mind: An Intellectual and Social History, 1848-1938* (Berkeley: University of California Press, 1972), 231; Peter J. Whitehouse, Konrad Maurer, and Jesse F. Ballenger, *Concepts of Alzheimer Disease: Biological, Clinical, and Cultural Perspectives* (Baltimore: Johns Hopkins University Press, 2000), 296.

¹³⁰ Volker Becker, *Der Einbruch der Naturwissenschaft in die Medizin: Gedanken um, mit, über, zu Rudolf Virchow* (Berlin: Springer, 2008).

rector in 1853.¹³¹ Fortunately for Meynert, who openly shared his teacher's zeal for anatomy, Rokitansky promoted his former student, in 1870, to the first chair of psychiatry in the Austro-Hungarian Empire.¹³²

Due to the organizational structure of the Empire, specialty chairs in Vienna almost inevitably spread “as a block” to other large cities in Austro-Hungary.¹³³ Vienna represented the central hub whose spokes stretched to Prague, Budapest, Innsbruck, and Graz, among other cities. Indeed, Vienna was arguably the epicenter of specialized medicine for all of Europe, surpassing even Paris by the mid- to late-nineteenth century.¹³⁴ According to some, the two cities formed an “axis” of medical specialism,¹³⁵ with Vienna having brought to “full fruition” what the Paris school initiated earlier in the century.¹³⁶

If the so-called fruit was the proliferation of medical specialties, then the labor to produce it was anatomical research based on the methods termed “clinical-

¹³¹ Lesky, *The Vienna Medical School of the 19th Century*, 107–108; Sepp Leodolter, “Rokitansky und die k. und k. Gesellschaft der Ärzte,” *Wiener klinische Wochenschrift* 116, no. 23 (December 1, 2004): 771–771; Roland Sedivy, “Zum Gedenken an Carl Freiherr von Rokitansky,” *Wiener klinische Wochenschrift* 116, no. 23 (December 1, 2004): 769–70; Roland Sedivy, “200 Jahre Rokitansky – sein Vermächtnis für die heutige Pathologie,” *Wiener klinische Wochenschrift* 116, no. 23 (December 1, 2004): 779–87.

¹³² Sepp Leodolter, “Rokitansky und die k. und k. Gesellschaft der Ärzte,” *Wiener klinische Wochenschrift* 116, no. 23 (December 1, 2004): 771–771; E. Lesky, *Die Wiener Medizinische Schule Im 19. Jahrhundert* (Böhlau, 1965).

¹³³ Weisz, *Divide and Conquer*, 53.

¹³⁴ *Ibid.*, 49–50.

¹³⁵ Keintzel and Gabriel, *Gründe Der Seele*, 11.

¹³⁶ “The trend which had originated in the French school at the beginning of the century and focused on anatomic lesion [sic] of the organs--organicism--had come to full fruition.” Lesky, *The Vienna Medical School of the 19th Century*, 109.

anatomical”¹³⁷ in Paris and “pathological-anatomical”¹³⁸ in Vienna. In each case, the goal was to dissect select organs and to discover therein circumscribed areas of damaged tissue which were believed to have caused “focal symptoms” of a specific disease. The concept of disease specificity, according to these schools, was based primarily on the specific organs affected and the location of their lesions. In large part, it was this idea of organic specificity that facilitated organizational specificity. Or in other words, localization begat specialization.¹³⁹

In 1868, Meynert published an article in Vienna's medical weekly “On the Necessity and Scope of an Anatomical Direction in Psychiatry.”¹⁴⁰ He belittled those who busied themselves with asylum design while neglecting anatomical research, saying that they were more concerned with the insides of buildings than the “interior structure of the brain” [*das Innere des Hirnbaues*].¹⁴¹ For Meynert, the “experienced psychiatrist”

¹³⁷ Goetz, Bonduelle, and Gelfand, *Charcot*; John E. Lesch, *Science and Medicine in France: The Emergence of Experimental Physiology, 1790-1855* (Cambridge, Mass: Harvard University Press, 1984).

¹³⁸ Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice*, 99; Weisz, *Divide and Conquer*, 51.

¹³⁹ Although it was not universally indicative of the formation of every specialty, the situation in Vienna adhered closely to this pattern which historian George Rosen has called “organic localism,” and which the nineteenth-century physician Carl Wunderlich parodied as, “every organ has its priest.” Accordingly, the brain's new Brahmin-like priests were academic psychiatrists like Theodor Meynert. The trouble was, if Wunderlich's metaphor can be so stretched, there were other “priests” of psychiatry with different “theologies” and “sacraments”—like aetiologies and therapies. Rosen, *The Specialization of Medicine with Particular Reference to Ophthalmology*, 3–4; Jewson, “The Disappearance of the Sick-Man from Medical Cosmology, 1770-1870,” 236; Weisz, *Divide and Conquer*, 51. Rosenberg issues a useful caution against assuming that the term “specialization” somehow explains itself: “Insofar as we ask such ideal types [“specialization” or “professionalization”] to serve as both description and explanation of change, we have to some extent chosen to mislead ourselves.” Rosenberg, “Toward an Ecology of Knowledge,” 441.

¹⁴⁰ Theodor Meynert, “Über die Nothwendigkeit und Tragweite einer anatomischen Richtung in der Psychiatrie,” *Wiener medizinische Wochenschrift* 18 (May 3, 1868): 573–76.

¹⁴¹ *Ibid.*, 576.

was not necessarily one who spent years in the asylum but one with experience in the laboratory performing autopsies. He echoed Griesinger and Rokitansky when he proclaimed the “roots of an exact psychiatric science” were to be found in the “anatomical facts” of brain disease.¹⁴²

Not everyone shared Meynert's opinion. The director of the Imperial Asylum, Ludwig Schlager (1828-1885), bristled at Meynert's attack on psychiatrists who did not embrace anatomical research. On the contrary, Schlager believed that Meynert was too distracted by the inflated promises of anatomy, which left patients waiting for the inevitably “slow and painful” advance of brain research while they wasted away in a “sorrowful condition.”¹⁴³ Moreover, Schlager complained that Meynert had overstepped his jurisdiction by siphoning patients from the asylum to his clinic, where he simply waited for them to die so he could dissect their brains.¹⁴⁴

Shortly after Meynert's promotion to the chair of psychiatry, Schlager and others petitioned that he be dismissed. But thanks to Rokitansky's patronage, Meynert was spared. In fact, he was promoted again to take charge of a whole new psychiatric clinic

¹⁴² To underscore the primacy of anatomy in psychiatry, Meynert turned to the neo-Kantian philosopher Hermann Lotze who confirmed, in Meynert's view, the inadequacy of relying on the patient's testimony from personal experience and introspection. He quoted directly from Lotze at the opening of his article, “Our soul knows nothing...of our body...whose interior it neither sees nor understands without outside help.” Especially when we are sick, we realize how little we actually know about the inside of our own bodies. It was then, in that moment of realization, Meynert concluded, that one must depend on a physician because he possessed the requisite anatomical knowledge of the diseased body. In a similar way, he reasoned, psychiatrists should aspire to the kind of knowledge of mental illness that was “grounded in the physiology of the brain.” The patient, after all, could not be trusted. Real “insight” into illness had to come from the outside. *Ibid.*, 573.

¹⁴³ Lesky, *The Vienna Medical School of the 19th Century*, 339–341.

¹⁴⁴ *Ibid.*, 340; Magda Whitrow, “The Early History of the Vienna Psychiatric Clinic,” *History of Psychiatry* 1, no. 4 (December 1, 1990): 419–25.

just so that he could continue his research unimpeded. From 1875 on, there were two independent departments of psychiatry at the University of Vienna. Nowhere else in Europe was the discipline so well represented. As a result, Vienna became the mecca of psychiatric research.¹⁴⁵

It was precisely during this period Anton began training as a psychiatrist. He studied medicine at the University of Prague only a year after the creation of Meynert's second department of psychiatry. After assisting first for Pick in Dobruška and then for Meynert in Vienna, he was appointed “extra-ordinary professor of psychiatry and nervous diseases” at the University of Innsbruck, its first such position.¹⁴⁶ It was there he began writing about his clinical encounters back in Vienna with the patients, Johann K. and Wilhelm H. And then, three years later in 1894, just within a month of moving to the University of Graz, he encountered Ursula Merz.¹⁴⁷

Even while psychiatry continued to expand throughout the German *Sprachgebiet*, including the Austro-Hungarian Empire and Switzerland, there was growing concern, especially in southwestern Germany, about its emphasis on anatomical research. When Meynert died in 1892, what the historian Erwin Ackerknecht dubbed the “two pillars” of psychiatry—the asylum and laboratory—had already started

¹⁴⁵ L. Schönbauer, *Das medizinische Wien: Geschichte, Werden, Würdigung* (Urban & Schwarzenberg, 1944), 340–341; Eulner, *Die Entwicklung der medizinischen Spezialfächer an den Universitäten des deutschen Sprachgebietes*, 4:276–277; Lesky, *The Vienna Medical School of the 19th Century*, 159, 340–341; Gröger, “Zur Entwicklung der Psychiatrie in der Wiener Medizinischen Schule,” 35.

¹⁴⁶ Eulner, *Die Entwicklung der medizinischen Spezialfächer an den Universitäten des deutschen Sprachgebietes*, 4:278–281; Hartmann, “Gabriel Anton zum 70. Geburtstag.”

¹⁴⁷ Anton’s predecessors at Graz were Richard Krafft von Ebing and Julius Wagner von Jauregg (1857–1940), who later succeeded Meynert in Vienna. Eulner, *Die Entwicklung der medizinischen Spezialfächer an den Universitäten des deutschen Sprachgebietes*, 4:277; Pfeifer, “Gabriel Anton,” 186.

to show cracks.¹⁴⁸ Chipping at each of these pillars were critics like the psychiatrist Emil Kraepelin (1856-1926) and the neuropathologist Wilhelm Erb (1840-1921). Kraepelin, for his part, had lost patience with the inflated promise of microscopic neuroanatomy. Instead, he preferred to focus on detailed clinical observation in an effort to formulate better “natural histories” and prognoses of mental illnesses.¹⁴⁹ Meanwhile, Erb argued that brain research be turned over to pathologists like himself, leaving the business of looking after asylums to the psychiatrists.¹⁵⁰

Ironically, Kraepelin's and Erb's critique of the combination of anatomy and asylum work owed to psychiatry's success from this very combination.¹⁵¹ Kraepelin was in a position to criticize the dependence on neuropathology largely thanks to the neuropathological research that helped create such academic chairs. Similarly, Erb had the clout to demand institutional independence of neuropathological research precisely because of the initial gains clinical psychiatrists made in that area of research. Consequently, their criticism was, in the words of historian Eric Engstrom, “a shot across the bow.”¹⁵² The gunners took aim at their own crew.

¹⁴⁸ Ackerknecht, “Gudden, Huguenin, Hitzig. Hirnpsychiatrie im Burghölzli 1869-1879,” 68.

¹⁴⁹ Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice*, 125–132.

¹⁵⁰ *Ibid.*, 124.

¹⁵¹ Engstrom argues that the legitimacy won with the help of pathological anatomy was later leveraged to criticize the dependence on pathological anatomy, primarily at the alleged expense of clinical commitments: “By the 1890s, however, the very same professional status that pathology had helped to secure, now made it easier for psychiatrists to strategically distance themselves from pathological anatomy and to employ psychological models to lay claim to new professional tasks.” *Ibid.*, 126.

¹⁵² *Ibid.*, 124.

There are some problems, however, with Engstrom's conclusion that neuropathology was generally "in retreat."¹⁵³ It runs the risk of projecting consensus across the entire *Sprachgebiet* from a cohort of critics mostly concentrated in southwestern Germany. True, the physicians of Heidelberg and Munich, such as Kraepelin, Erb, Franz Nissl (1860-1919), and later Karl Jaspers (1883-1969), each regarded Meynert's enterprise with suspicion and derided his so-called "brain mythology" [*Hirnmythologie*].¹⁵⁴ Still, it can be misleading to insinuate on the basis of their regional critique that psychiatrists who practiced neuropathology were everywhere in retreat. For instance, new chairs and clinics dedicated to clinical psychiatry and equipped with laboratories for neuropathology were continually being added throughout the Austro-Hungarian Empire well into the 1920s.¹⁵⁵ In that part of Europe at least, neuropathology was in anything but retreat.¹⁵⁶

Even so, it must be said that the emphasis psychiatrists placed on clinical observation and description grew toward the end of the century, as did research

¹⁵³ *Ibid.*, 123.

¹⁵⁴ Ellenberger, *The Discovery of the Unconscious*, 284; Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice*, 123; Karl Jaspers, "Die phänomenologische Forschungsrichtung in der Psychopathologie," *Zeitschrift für die gesamte Neurologie und Psychiatrie* 9, no. 1 (1912): 406.

¹⁵⁵ Weisz, *Divide and Conquer*; Keintzel and Gabriel, *Gründe Der Seele*; Eulner, *Die Entwicklung der medizinischen Spezialfächer an den Universitäten des deutschen Sprachgebietes*; Karl Bonhoeffer, "Psychiatrie und Neurologie," *Monatsschrift für psychiatrie und neurologie* 37, no. 2 (1915): 94–104.

¹⁵⁶ Anton's own career testifies to this fact. He continued to combine clinical psychiatry and neuropathology, indeed along with neurosurgery, and he ensured his students—e.g. Fritz Hartmann, Hermann Zingerle, and Paul Schilder (see Chapter 3)—likewise trained in neuroanatomical research. Gabriel Anton and Fritz Gustav von Bramann, "Balkenstich bei Hydrozephalien, Tumoren und bei Epilepsie," *Münchener medizinische Wochenschrift* 55 (1908): 1673–77; Gabriel Anton and Fritz Gustav von Bramann, *Behandlung der angeborenen und erworbenen Gehirnkrankheiten mit Hilfe des Balkenstiches* (Berlin: S. Karger, 1913); Kumbier, Haack, and Herpertz, "Überlegungen zum Wirken des Neuropsychiaters Gabriel Anton (1858-1933)."

interests in the subjective, “inner” experience of patients. Both the emerging research techniques of experimental psychology and the application of what was widely called “psychological analysis,” not just Freud's variant, were increasingly adopted by academic psychiatrists.¹⁵⁷ Even Anton's paper attests to the changes. Published the same year as Freud finished writing *The Interpretation of Dreams*, Anton framed this new disease entity as a “new diagnostic aid”¹⁵⁸ and thus a valuable contribution to not only the “*psychology of focal symptoms*” and “*subjective symptomatology*” but also for the “practical understanding” of patients and their experiences.¹⁵⁹ Localization of the brain remained part of psychiatric research, but it was only one part. The other

¹⁵⁷ The first prominent critics of Anton's research were fellow Austrian psychiatrists, Redlich and Bonvincini who published voluminous detailed reports of patients who denied blindness but who also manifested many other complications, suggesting overall mental confusion, insanity, or a confabulatory behavior called “Korsakoff's syndrome.” Redlich especially explicitly urged for the use of “intelligence exams” [*Intelligenzprüfungen*] and “psychological analyses” to determine more accurately the extent of each patient's dementia or delirium. Redlich and Bonvincini, “Über mangelnde Wahrnehmung (Autoanästhesie) der Blindheit bei cerebralen Erkrankungen”; Emil Redlich and Giulio Bonvincini, “Ueber das Fehlen der Wahrnehmung der eigenen Blindheit bei Hirnkrankheiten,” *Jahrbuechern fuer Psychiatrie und Neurologie* 29 (1908): 1–134; Emil Redlich and Giulio Bonvincini, “Weitere klinische und anatomische Mitteilungen über das Fehlen der Wahrnehmungen der eigenen Blindheit bei Hirnkrankheiten,” *Neurologisches Centralblatt* 30 (1911): 227–35; Engstrom, “Neurowissenschaften und Hirnforschung,” 788.

¹⁵⁸ “Es scheint mir die Erwartung gerechtfertigt, dass in diesem Verhalten des Individuums sich ein neuer diagnostischer Behelf ergeben werde.” Anton, “Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit,” 119.

¹⁵⁹ “Die *Psychologie der Herdsymptome*—um das Frühere zusammenzufassen—besonders der Gehirnrindenverletzungen wurde bei dem allgemeinen Bestreben, *äusserlich* eruirbare Herdsymptome zu finden, vielleicht zu wenig berücksichtigt; und doch kommt dieser *subjectiven* Symptomatik gewiss ein hoher Werth zu, sowohl für die diagnostischen Aufgaben, als auch für das practische Verständniss des Kranken.” (italics original) *Ibid.*, 88–89.

remained, albeit in varying guises, what Anton called “the hunt for mental symptoms.”¹⁶⁰

The Question of the Soul and Scientific Materialism

Ideas about disease always bump into other ideas. And the disease-picture is quickly complicated when those other ideas are about the nature of experience, particularly the patient's experience of disease, or illness. In the history of psychiatry, this connection between the idea of disease and illness-experience has always been especially conspicuous and complex. In part it is because the patient's experience, at least statements about it, has often formed the basis for making a psychiatric diagnosis.¹⁶¹ Hallucinations, delusions, obsessions, phobias, and the like are among the historical hallmarks of such diagnoses.¹⁶² Not so much the body but the mind is somehow diseased. It is from this extremely crude and sketchy picture of the “sick mind” on which psychiatry has, by and large, relied for its most basic intelligibility.¹⁶³

¹⁶⁰ “Demgemäss hat die Fahndung nach klinischen Symptomen sich nicht auf die Gehörstörungen zu beschränken, sondern in besonderem Maasse die psychischen Mitsymptome festzustellen.” Ibid., 120.

¹⁶¹ Rosenberg, “Contested Boundaries.”

¹⁶² Berrios, *The History of Mental Symptoms*.

¹⁶³ Roy Porter, “The Body and the Mind, the Doctor and the Patient: Negotiating Hysteria,” in *Hysteria Beyond Freud*, ed. Sander L. Gilman (Berkeley: University of California Press, 1993), 225–85; Goldstein, *Console and Classify*; Charles E. Rosenberg, *The Trial of the Assassin Guiteau: Psychiatry and the Law in the Gilded Age* (Chicago: University of Chicago Press, 1968); Foucault, *Maladie mentale et psychologie*; Michel Foucault, *Madness and Civilization: A History of Insanity in the Age of Reason* (New York: Vintage Books, 1973); F.B. Alberti, “Bodies, Hearts, and Minds: Why Emotions Matter to Historians of Science and Medicine,” *Isis* 100, no. 4 (2009): 798–810; Anne Harrington, *The Cure Within: A History of Mind-Body Medicine* (New York: W. W. Norton & Company, 2008); George Rousseau, “‘Brainomania’: Brain, Mind and Soul in the Long Eighteenth Century,” *Journal for Eighteenth-Century Studies* 30, no. 2 (2007): 161–91; Mark S. Micale and Paul Lerner, eds., *Traumatic Pasts: History, Psychiatry, and Trauma in the Modern Age, 1870-1930*, 1st ed. (Cambridge University Press, 2010).

But it has not always been *just* the mind. In both German and French the word for “mind” can also denote “soul.” (That is why some English physicians translated Hermann Munk's neologism, *Seelenblindheit*, “soul-blindness.”¹⁶⁴) Indeed, this semantic ambiguity echoes the persistent ontological ambiguity that in many ways lies at very core of psychiatry. What is the difference between the mind and soul, or between the soul and body? And if there is a difference, then what impact does it have on the distinction between disease and illness, at least or especially when, as in Anton's syndrome, the disease entity is defined solely by the patient's lack of experience of illness? In other words, the identity of psychiatry has always been bound up with not only ideas of disease but also ideas of the soul.¹⁶⁵

In 1843, the Austrian Ernst Baron von Feuchtersleben (1806-1849) became the first doctor to hold an academic position in the area of medicine loosely referred to in

¹⁶⁴ According to Walther Riese, neurologist and historian, by the time Munk proposed the term “*Seelenblindheit*,” the “soul” vanished from the vocabulary of the philosopher and physician.” Riese adds that Munk furthered the “decomposition of the soul” by “sacrificing” its name in the description of a localized disease entity like “*Seelenblindheit*”: “By defining (in 1881) mind-blindness as lack of visual representations or of the memory-images ('Erinnerungsbilder') of visual perceptions, he could indeed satisfy his contemporaries, since the essential element of the soul, namely its unifying character was sacrificed in favor of the then traditional *decomposition of the soul into various psychic functions related to as many cerebral regions.*” (his italics) Walther Riese, *Principles of Neurology in the Light of History and Their Present Use*, Nervous and Mental Disease Monographs, no. 80 (New York, 1950), 85.

¹⁶⁵ Michael Hagner, “Hirnforschung und Materialismus,” in *Materialismus-Streit*, ed. Kurt Bayertz, Myriam Gerhard, and Walter Jaeschke, vol. 1, 3 vols., Weltanschauung, Philosophie und Naturwissenschaft im 19. Jahrhundert (Hamburg: F. Meiner Verlag, 2007), 204–22; Harrington, “Beyond Phrenology: Localization Theory in the Modern Era”; Harrington, “Psychiatrie und die Geschichte der Lokalisation geistiger Funktionen,” 609; John R. Searle, “The Self as a Problem in Philosophy and Neurobiology,” in *The Lost Self: Pathologies of the Brain and Identity*, ed. Todd E. Feinberg and Julian Paul Keenan (Oxford University Press US, 2005); Oliver Sacks, “Neurology and the Soul,” in *The Enchanted Loom: Chapters in the History of Neuroscience*, ed. Pietro Corsi (New York: Oxford University Press, 1991), 366–70; Kiefer, *Die Entwicklung des Seelenbegriffs in der deutschen Psychiatrie ab der zweiten Hälfte des 19. Jahrhunderts unter dem Einfluss zeitgenössischer Philosophie*; D. H. Brendel, “Philosophy of Mind in the Clinic: The Relation between Causal and Meaningful Explanation in Psychiatry,” *Harvard Review of Psychiatry* 8, no. 4 (2000): 184–91; Roderick M. Chisholm, “The Self in Austrian Philosophy,” in *On Metaphysics* (Minneapolis: University of Minnesota Press, 1989), 156–61.

those days as psychiatry. It was he who, five years before, wrote a short book *On the Dietetics of the Soul*.¹⁶⁶ In it, he tried to argue for the “power of the human spirit [*Geist*] over the body,” proposing that the dietetics of the soul was principally about “self-knowledge...[and] self-control.”¹⁶⁷ In 1845, he wrote one of the earliest medical textbooks on psychiatry, which he preferred to calling the “medical care of the soul” [*ärztlichen Seelenkunde*]. The same year, however, the young Griesinger would also release the first edition of his textbook on psychiatry, *The Pathology and Therapy of Mental Illnesses for Doctors and Students*, in which he briskly declared that mental illness was first and foremost to be studied as brain disease.¹⁶⁸ He made very few concessions to the relevance or reliability of “self-knowledge,” much less the “human spirit.” Griesinger’s book quickly eclipsed Feuchtersleben’s, not least because he preferred to distance psychiatry from philosophy and align it openly with “scientific materialism.”¹⁶⁹ Certainly, he was far from alone at the time.

¹⁶⁶ Ernst Feuchtersleben, *Zur Diätetik Der Seele*, 27. Aufl (1838. Repr., Wien: C. Gerold’s Sohn, 1865).

¹⁶⁷ Lesky, *Die wiener medizinische Schule im 19. Jahrhundert*, quoted on 153.

¹⁶⁸ Many assume that Griesinger argued that “all patients with so-called mental illness are really just individuals with brain disease.” This attribution appears, on closer reading, to be somewhat apocryphal, originating likely from Ackerknecht. Griesinger instead wrote that mental illness was best studied and “recognized” in terms of brain disease: “Zeigen uns physiologische und pathologische Thatsachen, dass dieses Organ [erkrankt bei Irresein] nur das Gehirn sein kann, so haben wir vor Allem in den psychischen Krankheiten jedesmal Erkrankungen des Gehirns zu erkennen.” The difference is that the latter is more modest and circumspect by avoiding equivalent identity between the two. Schott and Tölle, *Geschichte der Psychiatrie*; Ackerknecht, *Kurze Geschichte der Psychiatrie*; Shorter, *A History of Psychiatry*; Guenther, “A Body Made of Nerves: Reflexes, Body Maps and the Limits of the Self in Modern German Medicine.”

¹⁶⁹ Marx, “Nineteenth-Century Medical Psychology,” 369; Wahrig-Schmidt, *Der junge Wilhelm Griesinger im Spannungsfeld zwischen Philosophie und Physiologie: Anmerkungen zu den philosophischen Wurzeln seiner frühen Psychiatrie*, 137; Engstrom, *Clinical Psychiatry in Imperial Germany: A History of Psychiatric Practice*, 59.

In 1841, a young theology student named Ludwig Feuerbach (1804-1872) published a book called *The Essence of Christianity*.¹⁷⁰ Feuerbach was one of the “young Hegelians,” meaning that he was dissatisfied with the older generation of philosophers and theologians who emphasized the importance of G.W.F. Hegel's idea of the “Mind” or “Spirit” [*Geist*] to the detriment of individual sensory experience or “sensibility.”¹⁷¹ He criticized older scholars who viewed Hegel's idea of individual alienation from the “Spirit” as a separation from God. Instead, Feuerbach argued that the very idea of God was a mere projection of human needs. The alienation from God was really the alienation from oneself, one's own experiences, needs, and desires. The only way to overcome such self-alienation and self-deception was to recognize one's own projected experiences and, ultimately, to reclaim one's own corporeal, sensuous, and material existence.

This emphasis on the material basis of human existence, known as materialism, quickly became the watchword among many young Hegelians besides Feuerbach—among them the young Karl Marx (1818-1883), David Friedrich Strauss (1808-1874), and Bruno Bauer (1809-1882). However, their rally around materialism signaled more than an intellectual dispute. It articulated a more widespread sentiment of rebellion against the conservative political establishment across Europe. While Feuerbach disrobed the

¹⁷⁰ Ludwig Feuerbach, *The Essence of Christianity*, Dover ed, Dover Philosophical Classics (Mineola, N.Y: Dover Publications, 2008).

¹⁷¹ Frederick Gregory, *Scientific Materialism in 19th Century Germany* (Dordrecht: D. Reidel, 1977); John Edward Toews, *Hegelianism: The Path Toward Dialectical Humanism, 1805-1841* (Cambridge University Press, 1985), 219; Lesky, *The Vienna Medical School of the 19th Century*, 114.

spiritual trappings of religious belief to expose its underlying sensuous origins, others aimed more literally to dethrone the monarchical and ecclesiastical centers of power.¹⁷²

In addition, there arose new voices for change within the sciences as well. In 1847, a group of four young scientists in Berlin, Hermann von Helmholtz (1821-1894), Emil Du Bois-Reymond (1818-1896), Carl Ludwig, and Ernst Brücke (1819-1892), issued a manifesto proclaiming all natural phenomena were knowable only through physics and chemistry. The immediate source of their agitation was a trend in the sciences known as *Naturphilosophie* which often paired well with Hegelian idealism and the perceived excesses of metaphysical speculation in the sciences.¹⁷³ Instead, the “Berlin Four” demanded a more restrained and distinctly un-idealist, un-Romantic, and un-philosophical form of “scientific materialism.”¹⁷⁴

The most vociferous expression of this new form of materialism came from the zoologist Karl Vogt (1817-1895), who in 1846 wrote an incendiary letter in Frankfurt's newspaper, the *Allgemeine Zeitung*. In the letter, he declared that “all thoughts stand in the same relation to the brain as...urine to the kidneys.”¹⁷⁵ He ridiculed the notion that

¹⁷² Paul W. Schroeder, *The Transformation of European Politics 1763-1848*, Oxford History of Modern Europe (Oxford: Clarendon Press, 1994); Toews, *Hegelianism*, chap. 8–10, passim.

¹⁷³ Paul F. Cranefield, “The Organic Physics of 1847 and the Biophysics of Today,” *Journal of the History of Medicine and Allied Sciences* 12, no. 10 (1957): 407–23; Paul F. Cranefield, “The Philosophical and Cultural Interests of the Biophysics Movement of 1847,” *Journal of the History of Medicine and Allied Sciences* 21, no. 1 (1966): 1–7.

¹⁷⁴ Hans-Jörg Rheinberger, *Historische Epistemologie zur Einführung* (Hamburg: Junius, 2007), 16–21; Hagner, “Hirnforschung und Materialismus,” 207; Hagner, “Die elektrische Erregbarkeit des Gehirns,” 259–260.

¹⁷⁵ Hagner, “Hirnforschung und Materialismus,” 206; Hagner, *Homo cereбрalis*, 225; Lenoir, “Science for the Clinic: Science Policy and the Formation of Carl Ludwig's Institute in Leipzig,” 152.

the soul had any influence on the brain as “pure nonsense.” Not surprisingly, Vogt's inflammatory remarks caused an outcry. It was not just that he had offended religious sensibilities by his pillory of the soul. He, like Feuerbach and the Berlin Four, had wielded materialism to strike at a distinct political opposition, not only among the intelligentsia but more broadly the clerical and aristocratic ruling classes. Allusions to the brain and the soul, therefore, became proxies for their social and political grievances.¹⁷⁶

Already by late 1847, social unrest had begun to spread throughout Europe. The consecutive failed harvests of 1846 and 1847, compounded by a recent cholera outbreak and made even worse by deplorable living conditions in urban areas were enough to tip the scales and trigger widespread revolts. In a matter of weeks, a larger number of university leaders and students joined forces with the urban working classes to agitate for major social and political reforms. By February of 1848, the King of France Louis-Phillipe had abdicated to England, which sent out a ripple effect of revolutions across the Continent. In Austria, Prince Metternich was forced to resign, and by the end of March, rulers in Prussia allowed a provisional parliament to convene in Frankfurt's Paulskirche to draft a new constitution. But in the end the alliance between workers and

¹⁷⁶ Hagner writes, “[D]ie Verbindungen von Hirnforschung, Materialismus und Politik in der Zeit des Vormärz zwar geknüpft, aber doch nicht ganz so ernst genommen wurden bzw. so wirkungsvoll waren, wie es sich deren Protagonisten vielleicht gewünscht hätten.” Hagner, “Hirnforschung und Materialismus,” 207; Hagner, *Homo cerebrialis*, 226–228; Jacyna, *Lost Words: Narratives of Language and the Brain, 1825-1926*, 67; Cranefield, “The Organic Physics of 1847 and the Biophysics of Today”; Cranefield, “The Philosophical and Cultural Interests of the Biophysics Movement of 1847.”

intellectuals was strong enough to be sustained, and the former politically conservative powers prevailed.¹⁷⁷

Many like the Prussian Rudolf Virchow and Austrian Carl von Rokitansky, Meynert's future teacher, reacted to the defeat of liberalism by tamping down their own heady rhetoric in favor of a more subdued and circumspect “worldview” of “scientific realism.”¹⁷⁸ The shift was subtly epistemological. They upheld the “worldview” that physics, chemistry, and biology formed the backbone of all knowledge, but they limited the scope of what fell within legitimate purview of that knowledge. Namely, questions like the relationship between the soul and the brain were treated with extra caution, if not avoided altogether.¹⁷⁹

In 1866, the philosopher Friedrich Albert Lange (1828-1875) published the first edition of his two-volume *History of Materialism*, in which he lambasted the naïveté of the pre-1848 materialists and praised the new vanguard of physiologists who stayed above the metaphysical fray. Lange's trouble with materialism was epistemological.¹⁸⁰

¹⁷⁷ Hagner, “Hirnforschung und Materialismus,” 210, 220.

¹⁷⁸ Historian of science Michael Hagner describes this subtle but critical shift with special regards to Carl Ludwig and his deliberate omission of the term “materialism” and its replacement by “realistic worldview”: “Mit dieser Haltung repräsentierte er eine Position, die sich von der erwünschten oder perhorreszierten Naturalisierung des Menschen so gut es ging fernzuhalten versuchte. Zwischen diesen beiden Extremen zogen sich Ludwig und auch die anderen Protagonisten der experimentellen Physiologie auf ihre methodischen Standards zur Absicherung einer wissenschaftlichen Erkenntnis zurück. In einer zentralen Passage seines Lehrbuchs vermied Ludwig sogar den Begriff 'Materialismus' und redete stattdessen von der 'realistischen Weltanschauung,' die er de Idealismus entgegenstellte.” Ibid., 218.

¹⁷⁹ This was particularly evident during the so-called “materialism debate” [*Materialismusstreit*] at the University of Göttingen in 1854. Ibid., 218–219; Thomas E. Willey, *Back to Kant: The Revival of Kantianism in German Social and Historical Thought, 1860-1914* (Wayne State University Press, 1978), 45.

¹⁸⁰ Gregory, *Scientific Materialism in 19th Century Germany*, 149; Friedrich Albert Lange, *Geschichte des Materialismus und Kritik seiner Bedeutung in der Gegenwart*, 3. Aufl (1866. Iserlohn: J. Baedeker, 1876), 348.

He believed that scientists should refrain from any reference to the soul, declaring, “A psychology without the soul!”¹⁸¹ Soon it became a rallying cry for future generations of philosophers, physiologists, and psychologists.¹⁸²

One of the most conspicuous signs that physiologists had learned to appreciate avoiding the “question of the soul” [*Seelenfrage*] was a speech by the Berlin physiologist Emil Du Bois-Reymond (one of the Berlin Four) in August of 1872. At the forty-fifth annual Convention of German Natural Scientists and Doctors in Leipzig, Du Bois-Reymond delivered his address with the title, “On the Limits of Natural Knowledge.”¹⁸³ In lockstep with Lange, he declared that the “riddle” of consciousness lay irredeemably outside human ken.¹⁸⁴ He chastised those like Vogt for their “brazen” disregard of the legitimate epistemic boundaries of science. As for the true nature of the relationship between the brain and soul, for that matter, between energy and matter itself, Du Bois-Reymond coyly conceded, “We will never know,” concluding with the Latin recitation, “*Ignorabimus.*”¹⁸⁵

¹⁸¹ Lange, *Geschichte des Materialismus und Kritik seiner Bedeutung in der Gegenwart*, 381.

¹⁸² Danziger, *Constructing the Subject*, 206n16.

¹⁸³ Emil Heinrich Du Bois-Reymond, *Über die Grenzen des Naturerkennens*, 2. Aufl (Leipzig: Verlag Von Veit, 1872).

¹⁸⁴ “Weder wäre damit das Bewusstsein überhaupt erklärt, noch für die Erklärung des einheitlichen Bewusstseins des Individuums das Mindeste gewonnen...Dass es vollends unmöglich sei, und stets bleiben werde, höhere geistige Vorgänge aus der als bekannt vorausgesetzten Mechanik der Hirnatome zu verstehen, bedarf nicht der Ausführung.” *Ibid.*, 26–27.

¹⁸⁵ Rheinberger, *Historische Epistemologie zur Einführung*, 10–11, 15–18; Du Bois-Reymond, *Über die Grenzen des Naturerkennens*, 34; Kurt Bayertz, Myriam Gerhard, and Walter Jaeschke, *Der Ignorabimus-Streit: Texte von E. du Bois-Reymond, W. Dilthey, E. von Hartmann, F. A. Lange, C. von Nägeli, W. Ostwald, W. Rathenau und M. Verworn* (Meiner Verlag, 2012), vii–xv.

If this show of deference on the matter of the soul seemed like a gesture of modesty, there was probably more to the show.¹⁸⁶ Physiologists like Du Bois-Reymond may have managed to tiptoe discreetly around the question of the soul, but the situation was rather different and arguably more difficult for psychiatrists. For them, the study of the brain and behavior was not merely the opportunity to align themselves with physiologists and thereby to make their research appear more scientific. In psychiatry, the study of mental illness and the question of how to relate the mind and the brain offered a critical test of Du Bois-Reymond's epistemology.

If the neuropathology of mental illness would ultimately confirm that all perceptual experience and all subjective awareness were nothing but physical and material, then the inner life was nothing but another side of the "outer" world. There was, in effect, nothing distinctly "inner" about the life of the mind.¹⁸⁷ That seemed to be the fulcrum on which psychiatry's fate as a science rested. That was why psychiatrists

¹⁸⁶ The display of modesty leveraged credibility. By making a show of self-restraint, the *Ignorabimus* speech signaled a different but equally ambitious bid for scientific authority. Steven Shapin and Simon Schaffer, *Leviathan and the Air-Pump: Hobbes, Boyle and the Experimental Life* (Princeton: Princeton University Press, 1989), 65–69; Donna Jeanne Haraway, *Modest-Witness@Second-Millennium.FemaleMan-Meets-OncoMouse: Feminism and Technoscience* (New York: Routledge, 1997), 26.

¹⁸⁷ Meynert already made up his mind, announcing matter-of-factly that the "inner" is just what goes on inside the body, as if looking into the eyes of another person granted all the access needed to determine his level of "insight": "Die Pupille sei die Pforte, durch welche unser Blick in das Innere eines anderen Menschen dringt. Hier ist das Psychische schon eine Nebenvorstellung beim Worte 'Innere.' Sie erweckt die Empfindung in einen Brunnen machte, 'einer unergründlichen Tiefe.'" Meynert, *Psychiatrie*, 261. So too Wernicke, who was confident that the source of consciousness was unarguably the brain and brain alone: "Es ist heute eine allgemein geläufige und kaum mehr discutierte Ansicht, dass der Sitz des Bewusstseins in die Grosshirnrinde zu verlegen ist." Carl Wernicke, "Über das Bewusstsein," *Allgemeine Zeitschrift für Psychiatrie und psychisch-gerichtliche Medicin* 35 (1879): 421. Gregory describes the consequence of this sort of equation: "All was outer experience for him. Reflections and perception both could be explained by motions communicated to the brain from the outside world." Gregory, *Scientific Materialism in 19th Century Germany*, 12.

like Anton could not help but be drawn to the question of the soul. It was the ultimate crucible by which to prove their worth to the rest of science, but it was also the very reason they had such a difficult time forging their scientific credentials in the first place. The paradox of psychiatric identity, therefore, was that its value to science consisted of making pronouncements on human nature, but in so doing it exposed its essential vulnerability as being something other than just a science. Psychiatrists enjoyed a unique position in that they straddled the border between medicine and philosophy. However, that also meant they did not entirely belong to either one.

If the question of the soul could not be settled, then perhaps it could be re-fashioned. All researchers needed was to focus on *phenomena* of the mind, the appearances or “symptoms” [*Seelenerscheinungen*], rather than worrying about its soul-like essence.¹⁸⁸ However, there was one critical problem that remained. Even after confining study to mental phenomena, psychiatrists, psychologists, philosophers, and physiologists all still had to contend with the *unity* of the mind.¹⁸⁹ That is, the feeling that one's experiences cohered and belonged to one indivisible, unified self—one personal consciousness. Yet any attempt to decide on such unity, either to affirm or deny it, would offend the metaphysical modesty of science. True, it was not the same as arguing for the “materiality” of the soul, or its proposed equivalence to the brain. However, to inquire into the unity of the mind and consciousness encroached on this question of the soul. For, the soul was traditionally invoked to account for the felt unity

¹⁸⁸ Willey, *Back to Kant*, 39–46.

¹⁸⁹ Engstrom, “Neurowissenschaften und Hirnforschung,” 778; A. Goodman, “Organic Unity Theory: The Mind-Body Problem Revisited,” *The American Journal of Psychiatry* 148, no. 5 (May 1991): 553–63.

of experience, indeed, the subjective “*feltness* of feeling” at all.¹⁹⁰ It permitted the sense—as feeling and as meaning—that one's mind was one's own. What but the soul made for the inner life, the sheer possibility that anything should appear to consciousness at all?¹⁹¹

A Symptom of the Times?

“The *self* is unsaveable,” the physicist Ernst Mach grimly concluded toward the end of the nineteenth century.¹⁹² Consciousness consisted of only a conglomerate of sensory experiences and nothing more. The mind was an accidental assembly and tentative “bundle” of fleeting impressions. Having once been Anton’s professor in Prague, Mach moved to the University of Vienna in 1895, just as his former student Gabriel Anton sank his scalpel into the deceased, “soul-blind” brain of Ursula Mercz.

¹⁹⁰ James, *The Principles of Psychology*, 1:189.

¹⁹¹ Few appreciated this conundrum better than the Austrian philosopher Franz Brentano (1838-1917). In 1874, he published *Psychology from an Empirical Point of View*, in which he critiqued Du Bois-Reymond's "atomistic hypothesis" for reducing consciousness to “a multiplicity” of sensations. If consciousness were a mere collection of sensations, and finally atoms, then what allowed for the recognition of any sensation as one’s own? What was the proverbial “cord or wire, or the like, that ties things together”? Contra the atomistic thesis of consciousness, Brentano believed it better compared to a “flowing stream.” It could not be divided up. The American psychologist and philosopher William James later modified Brentano's analogy into the now-famous phrase, “stream of consciousness.” With this fluid, elusive unity of “inner perception” and emphasis on “intentional” consciousness, Brentano helped inspire the philosophy known as “phenomenology.” Franz Clemens Brentano, *Psychologie vom empirischen Standpunkt* (Leipzig: Duncker & Humblot, 1874); Liliana Albertazzi, *Immanent Realism: An Introduction to Brentano* (Dordrecht, Holland: Springer, 2006); W. Baumgartner, “Franz Brentano: ‘Grossvater der Phänomenologie,’” *Studia Phaenomenologica*, no. III (1–2) (2003): 15; R.D. Rollinger, *Austrian Phenomenology: Brentano, Husserl, Meinong, and Others on Mind and Object*, Phenomenology and Mind, Bd. 12 (Frankfurt: Ontos, 2008).

¹⁹² “Das *Ich* ist unrettbar.” Literally, “The *I* [ego] is unsalvageable.” Ernst Mach, *Beiträge zur Analyse der Empfindungen* (G. Fischer, 1886), 18n12; Ernst Mach, *The Analysis of Sensations, and the Relation of the Physical to the Psychological*, trans. C.M. Williams (Chicago: Open Court Publishing, 1897), 19–20; Hagner, *Homo cerebrialis*, 288.

Compared side by side, Anton and Mach may seem like an odd couple, but their research was rather complementary. Anton pursued in pathological terms the counterpart of Mach's philosophical dictum. He described a peculiar dissolution of the self as he delineated the breakdown of self-awareness in his patients. If sensory perception was all there was behind the illusion of the self, then so too with "self-perception." It could be disassembled and dissected. It, too, was an illusion of "cerebral atoms."¹⁹³

In this view, nothing appeared certain or stable. Everything, mourned the poet Hugo von Hofmannsthal, "slips and flows away."¹⁹⁴ Experiences no longer mattered, wrote Robert Musil, because "we can no longer relate them to ourselves." All of life "notoriously turned toward abstraction."¹⁹⁵ If at the turn of the twentieth century, Vienna, indeed the Austro-Hungarian Empire, existed in an "age of uncertainty,"¹⁹⁶ as some historians suggest, then Anton's new disease entity fit in rather well for its time. Here was an illness indeterminately defined by the illusion of health, exposing in its wake a fractured vestige of the coherent self and leaving nothing else to hold on to but a stranded bundle of brain fibers.

¹⁹³ Friedrich Albert Lange, *History of Materialism and Criticism of Its Present Importance: History of Materialism since Kant*, vol. 2 (London: K. Paul, Trench, Trübner, & Company, Limited, 1881), 325.

¹⁹⁴ Paul Schilder, "Über das Selbstbewusstsein und seine Störungen," *Zeitschrift für die gesamte Neurologie und Psychiatrie (Neurologisches Centralblatt)* 20 (1913): quoted on 511.

¹⁹⁵ Robert Musil, *The Man Without Qualities: A Sort of Introduction and Pseudoreality Prevails*, vol. 1 (New York: Vintage Books, 1996), 708.

¹⁹⁶ Deborah R. Coen, *Vienna in the Age of Uncertainty: Science, Liberalism, and Private Life* (Chicago: University of Chicago Press, 2007); Carl E. Schorske, *Fin-De-Siècle Vienna: Politics and Culture* (New York: Knopf, 1979); Allan Janik and Stephen Toulmin, *Wittgenstein's Vienna* (New York: Simon and Schuster, 1973).

On second thought, Anton's disease of self-perception implied a latent expectation that self-perception was otherwise normal and reliable. How else could it be "diseased," unless, when "healthy," it could be counted on for sound insight? This was the paradox of its own making. Anton's syndrome depended on an idea of decomposable consciousness yet exposed a deeper reliance on that same consciousness, prior to the disease, being inextricably, inexplicably *intact*.

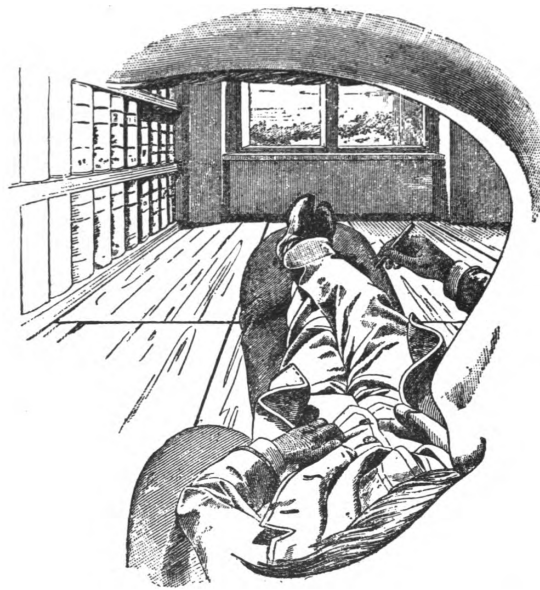


Fig. 1.

Figure 3. Ernst Mach's "phenomenalist" sketch from his own first-person point of view, despite his worldview that any lasting coherence of personal identity was ultimately indefensible.¹⁹⁷

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For Anton's 60th birthday celebration, a philosophy professor from Graz hailed his former colleague for having unmasked the myth of "inner perception." The "struggle between medicine and philosophy," the philosopher boasted, was simply because

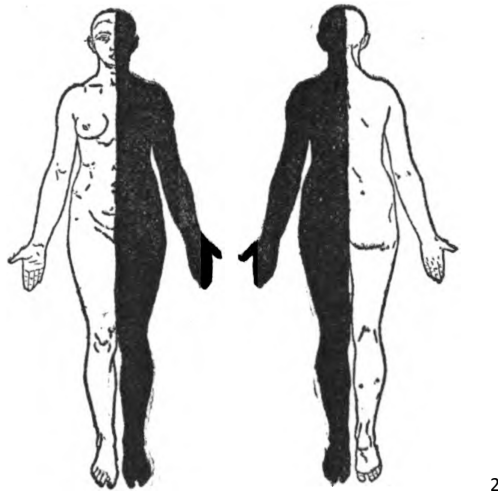
¹⁹⁷ Mach, *Beiträge zur Analyse der Empfindungen*, 14.

“[T]he healthy man forgets his body and can abstract from it, whereas the sick man is always reminded of the physical limitations of his existence.”¹⁹⁸ On this last point, however, the philosopher seems to have forgot something himself, namely, the central lesson of Anton’s syndrome. Illness did not necessarily remind one of the physical limits of bodily existence. It did not always lead to a greater self-awareness or the realization that one cannot always rely on one’s body or one’s senses. Sometimes, Anton pointed out, illness was just the opposite of awareness.

¹⁹⁸ Hugo Spitzer, “Psychologie und Gehirnforschung,” *European Archives of Psychiatry and Clinical Neuroscience* 59, no. 2 (1918): 422.

Chapter 2
**The Revolution of Indifference,
or How One Half of Hysteria Became Two New Brain Diseases**

*"Indifference is a sign that one has not understood, that one is in error."*¹



In 1914, at a meeting of the *Société de Neurologie* in Paris, the French-Polish physician Joseph Babinski (1857-1932) briefly reported on two patients with a very unusual type of "mental trouble."³ Each suffered from left hemiplegia, a form of extreme weakness bordering on paralysis that affected one half of the body. The first patient was not in the clearest state of mind, but Babinski assured his colleagues that

¹ Taylor, *Sources of the Self*, 161.

² Figure 1. Schematic of hysterical patient with left-sided hemianaesthesia. Pierre Janet, *The Major Symptoms of Hysteria: Fifteen Lectures Given in the Medical School of Harvard University* (New York: The MacMillan Company, 1907), 153.

³ Babinski began his report with the following announcement: "Je désire attirer l'attention sur un trouble mental que j'ai eu l'occasion d'observer dans l'hémiplégie cérébrale, et qui consiste dans ce fait que les malades ignorent ou paraissent ignorer l'existence de la paralysie don't ils sont atteints." Babinski, "Contribution à l'étude des troubles mentaux dans l'hémiplégie organique (anosognosie)," 845.

she appeared to be intelligent “for the most part.”⁴ She exhibited no signs of hallucinations, confabulations, or confusion. She still remembered past events, carried on normal conversations, and even asked about the latest news. Overall, she behaved relatively normal, except that, “She seems almost completely to ignore the existence of her hemiplegia.” She “never complains about it,” Babinski added, or “even alludes to it.” When he asked her to try to lift her left arm, her face went suddenly blank. She remained “motionless, in guarded silence, as if the question had been addressed to someone else.” He could not bring her attention back to the conversation until he dropped the topic of her hemiplegia. A while later, the patient, “having remained for some time in a demented [*démentiel*] state,” passed way.⁵

The second case Babinski reported was similar. She also suffered from a left-sided hemiplegia of which she appeared to be unaware. Even though her memory was “excellent” and her conversation remained “lively,” she was blithely ignorant of her lame left arm. When Babinski asked her to try to raise it, she fell silent like the first patient. Moments later, suddenly she blurted out, “There, it's done” [*Voilà ; c'est fait*]. All the while, her left arm lay still by her side. Did she really think she had moved it? Did she really not understand what was wrong? It certainly seemed that way, Babinski thought. “This ignorance [*ignorance*] of the patient” appeared quite sincere, quite “real.” And if so, he thought it needed a new name.

⁴ “[Elle] conservé en grande partie ses faculté intellectuelles et affectives.” Ibid.

⁵ Ibid.

It is, I believe, permissible to avail oneself of a neologism to designate this state and to call it *anosognosie*.⁶

The name, "*anosognosia*,"⁷ was an amalgamation of ancient Greek,⁸ which Babinski translated loosely as the "*lack of awareness of illness*."⁹ This was his chosen neologism, his diagnostic creation, a new name to christen a new disorder. That was the bold insinuation. But even more boldly one might wonder whether it was the other way around instead: a new disorder created by a new name. That is my claim in this chapter.

In the same paper, Babinski introduced yet another new name. This one he called, "*anosodiaphoria*," which meant (or he intended it to mean) "*indifference to illness*."¹⁰ Briefly, he explained,

I have also observed in some hemiplegics, who without ignoring the existence [*ignorer l'existence*] of their paralysis, seem to attach no importance to it, as if

⁶ "Il est, je crois, permis de se servir d'un néologisme pour désigner cet état et de l'appeler *anosognosie*." Ibid., 846.

⁷ Pronounced *ã-nõ'sog-nõ'së-ã*.

⁸ "a" = *lack*; "nosos" = *disease*; "gnosis" = *knowledge*.

⁹ And the name stuck. Babinski, "Anosognosie"; J.A. Barré, L. Morin, and J. Kaiser, "Etude clinique d'un nouveau cas d'anosognosie de Babinski," *Revue Neurologique* 39 (1923): 500–503; Åke Barkman, "De l'anosognosie dans l'hémiplégie cérébrale. Contribution clinique à l'étude de ce symptôme," *Acta Medica Scandinavica* 62, no. 1 (1925): 235–54; Bisiach, Luzzatti, and Perani, "Unilateral Neglect, Representational Schema and Consciousness."; E. Bisiach et al., "Unawareness of Disease Following Lesions of the Right Hemisphere: Anosognosia for Hemiplegia and Anosognosia for Hemianopia," *Neuropsychologia* 24, no. 4 (1986): 471–82; Heilman, Barrett, and Adair, "Possible Mechanisms of Anosognosia"; Papagno and Vallar, "Anosognosia for Left Hemiplegia: Babinski's (1914) Cases"; H. Branch Coslett, "Anosognosia and Body Representations Forty Years Later," *Cortex* 41, no. 2 (2005): 263–70; K.G. Langer, "Babinski's Anosognosia for Hemiplegia in Early Twentieth-Century French Neurology," *Journal of the History of the Neurosciences* 18, no. 4 (2009); Prigatano, "Historical Observations Relevant to the Study of Anosognosia."

¹⁰ "*Diaphoria*," which Babinski modeled on the ancient Greek word, *diaphoros* or διαφορία, means "difference," which means that "*anosodiaphoria*," literally translated, would come out something like the "lack of difference in illness." Babinski intended "*indifference*" or "lack of interest," however, not the "lack of difference."

it were a minor inconvenience [*malaise insignifiant*]. Such a state could be called *anosodiaphorie* (ἀδιαφορία, indifférence, insouciance).¹¹

Both anosodiaphoria and anosognosia, he continued, were likely “cerebral” [*cérébrale*] in origin, probably due to specific “lesions in the right hemisphere,”¹² since both patients’ types of hemiplegia affected the left side of their body.¹³ But beyond that, Babinski did not specify any further. Toward the end of his presentation, as he opened the floor to his colleagues, he closed with the tentative question: “Should we thus admit that anosognosia is real?” For the moment, he seemed to think that it was, but even he could not be sure.¹⁴ Even he had to wonder whether it existed only in name, as a mere diagnostic novelty of his own creation.

Clearly, Babinski was fond of inventing new words. It only seemed fitting when faced with new clinical phenomena. But it was never clear how truly *new* this phenomenon was. For one, Gabriel Anton had already described a similar state of unawareness more a decade and a half earlier.¹⁵ Babinski appears to have been

¹¹ Babinski, “Contribution à l’étude des troubles mentaux dans l’hémiplégie organique (anosognosie),” 846.

¹² *Ibid.*, 847.

¹³ Due to the cross-over of nerve fibers in the brain stem, known as the “pyramidal decussation,” sometimes nerve damage and brain disease, if confined to one side, will manifest on the opposite, or “contralateral,” side of the body. It depends, however, on whether the tissue damage occurs either above or below the cross-over point near second cervical vertebra of the spine. Robert J. Schwartzman, *Differential Diagnosis in Neurology* (Amsterdam: IOS Press, 2006).

¹⁴ Babinski, “Contribution à l’étude des troubles mentaux dans l’hémiplégie organique (anosognosie),” 846.

¹⁵ In his 1899 paper, Anton made multiple references to patients who “barely noticed” their “unilateral paralysis” [*einseitige Körperlähmung wird vom Kranken wenig bemerkt und beachtet*]. He also reported individual cases of such unawareness in 1893. Anton, “Beiträge zu klinischen Beurtheilung und zur Localisation der Muskelsinnstörungen im Grosshirne,” 317, 325; Anton, “Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit,” 86–87.

unaware of Anton's work, though. He never cited anything by him, and for that matter, Anton never mentioned Babinski in connection with “his” disorder. Furthermore, a number of other doctors, most of them Babinski's own colleagues in Paris, had already documented a similar kind of “indifference” among patients with a different type of disorder known as “hysteria.” Although he knew of their work, he avoided any direct comparisons between the “old” diagnosis of hysteria and the two “new” diagnoses of anosognosia and anosodiaphoria. But the question remains, why?

This chapter turns a critical eye to Babinski’s own study of hysteria. It focuses on a particular symptom known as “hysterical indifference” and examines what it meant to two very different French doctors at the turn of the century. It begins with the research of Pierre Janet (1859-1947), the philosopher-physician who featured the psychopathology of hysterical indifference prominently in his psychological studies of perception. Then, the discussion takes up Babinski's early interest in hysteria. It reviews his tumultuous relationship with the neurologist Jean-Martin Charcot (1825-1893) and considers the potential sources of his later transformation, both of the hysteria diagnosis and himself, following Charcot’s death. Finally, I return to Babinski's 1914 paper on anosognosia and anosodiaphoria for closer treatment. I argue that Babinski, like Anton, drew from Janet's understanding of hysterical indifference in order to reformulate his own “newer” version, effectively translating it from a dubious psychological disorder of the will into a genuine disease of the brain.

*“Une indifférence particulière”*¹⁶

When Anton published the cases of Ursula Mercz and Juliane Hochrieser in 1899, he underlined the fact that their form of unawareness was not the result of general confusion or diffuse brain disease. Some doctors may conflate the two, but Anton remained convinced that if one were “psychiatrically trained,” as Wernicke said, then it was always “obvious” [*auffällig*] how this kind of behavior was due to a “focal” cause from a particular region of the brain. Still, there was room for diagnostic confusion, Anton noted. Apart from the question of insanity or dementia, some doctors might mistake Mercz’s or Hochrieser’s unawareness as “psychogenic” [*psychogene*], that is, “hysterical.”¹⁷ Even he admitted there was “a noteworthy similarity.”¹⁸

Some patients with hysteria showed signs of hemiplegia [*halbseitige Lähmung*] and hemianaesthesia [*halbseitige Gefühlslähmung*], which looked rather like the patients Anton observed. The hysterics also barely noticed, if ever, that they could not move or feel one side of their body. Their “experience” was “psychologically understandable,” reasoned Anton, because any “signal” or “stimulus” coming from that side of the body no longer “aroused psychological combinations” in the mind.¹⁹ That

¹⁶ Pierre Janet, *État mental des hystériques. Les stigmates mentaux*, vol. 1, Bibliothèque médicale Charcot-Debove (Paris: Rueff et cie, 1892), 44.

¹⁷ Anton, “Über die Selbstwahrnehmung der Herderkrankungen des Gehirns durch den Kranken bei Rindenblindheit und Rindentaubheit,” 88.

¹⁸ *Ibid.*

¹⁹ “[E]s scheint eine psychologisch verständliche Erfahrung, dass die Körperseite, von der kein Signal und kein Anreiz kommt, in der Folge *indifferent* wird und minder im Bewusstsein vertreten ist; es werden eben von dieser Seite keine psychische Cominationen mehr angeregt.” *Ibid.*, 87.

meant their affected half of the body was only “weakly represented to consciousness” [*minder im Bewusstsein vertreten ist*], causing them to be “indifferent” toward it.²⁰ The only difference, then, between this hysterical version of indifference and Anton’s version was the role of the brain.

For Anton, and for most of his counterparts, this was no trivial distinction. He argued emphatically that the loss of “self-perception” in his own patients was something distinct and new because it was “organic” rather than “psychogenic.” That meant it was caused by organic disease in the brain and not some “psychic” perturbation of the mind. Even so, the “noteworthy similarity” he drew with hysteria was not trivial either. As we will see, Anton applied the very language the French doctors used for hysterical indifference in order to explain his patients’ behavior as well. Among the French physicians he cited was the psychologist and philosopher Pierre Janet. Beginning in the mid-1880s, Janet dedicated his studies to the psychopathology of hysteria and, perhaps more than any other physician of his time, he was particularly keen to explore the hysterical phenomenon of “indifference.”

Janet began his career in philosophy and psychology with a special interest in the nature of perception. In 1885, having learned about experiments with hypnosis by the Parisian neurologist Jean-Martin Charcot,²¹ the young philosopher, working from the

²⁰ “Ueber diesen Ausfall jedoch brachte die Kranke niemals Klagen vor, sie blieb gegen diesen Defect stets und vollkommen indifferent.” *Ibid.*, 87, 107, 121.

²¹ Since 1876, after serving on a commission to test the therapeutic effects of hypnosis, Charcot began experimenting with hypnotic techniques on his own patients at the Salpêtrière hospital. In 1882, almost immediately after taking up the new chair created in neurology, he gave a paper before the *Académie des sciences* in which he claimed to have induced hysterical symptoms, including forms of paralysis, just by

port city of Le Havre, set out to perform his own hypnotic experiments on a woman named “Léonie.”²² Almost as soon as he published his initial findings in 1886, Janet was praised not only by Charcot himself but also by the American philosopher and psychologist, William James, who declared his work to be “the most important step forward in psychology.”²³

What made Janet's research with Léonie so significant was that it provided the first experimental evidence of more than one consciousness at work, at the same time, in one and the same individual. He was able to hypnotize her and impart to her suggestions of tasks which she would then carry out to the letter, albeit unwittingly to her “primary consciousness.” According to Janet, her “secondary” consciousness was at play, performing the tasks he asked of her while hypnotized, yet utterly at a remove from her primary consciousness. Although researchers had already generated evidence of this multiplicity of awareness, no one had yet shown, until Janet, that such “consciousnesses” could operate in the same person *simultaneously*. This discovery for Janet would prove pivotal not only to his theory of consciousness but also his future study of hysteria.²⁴

using hypnosis. Jean-Martin Charcot, “Sur les divers états nerveux déterminés par l’hypnotisation chez les hystériques,” *Comptes-rendus hebdomadaires des séances de l’Académie des Sciences* 94 (1882): 403–5; Anne Harrington, “Metals and Magnets in Medicine: Hysteria, Hypnosis and Medical Culture in Fin-de-Siècle Paris,” *Psychological Medicine* 18, no. 1 (February 1988): 23–28.

²² Pierre Janet, “Note sur quelques phénomènes de somnambulisme,” *Bulletins de la Société de psychologie physiologique* 1 (1885): 24–32; Ellenberger, *The Discovery of the Unconscious*, 338.

²³ Ann Taves, “Religious Experience and the Divisible Self: William James (and Frederic Myers) as Theorist(s) of Religion,” *Journal of the American Academy of Religion* 71, no. 2 (2003): quoted on 304.

²⁴ *Ibid.*, 307; Alexandra Bacopoulos-Viau, “Automatism, Surrealism and the Making of French Psychopathology: The Case of Pierre Janet,” *History of Psychiatry* 23, no. 3 (2012): 259–76.

In 1889, Janet built upon his previous research with Léonie to publish his doctoral thesis in philosophy, *Psychological Automatism [L'Automatisme psychologique]*.²⁵ At the end of that year, he then began to pursue another degree, this time in medicine, recognizing that it would vastly improve his access to research subjects and, in particular, patients with hysteria. And so, having recently moved to Paris to take up a teaching position in philosophy, Janet, now also a medical student, began to experiment with patients at Charcot's clinic in the Salpêtrière Hospital.²⁶

In the first years of Janet's medical study, his mentor Charcot was embroiled in a public scandal over controversial research using hypnosis on hysterical patients. From 1887 until Charcot's death in 1893, many doctors grew suspicious of his claims. In particular, the doctor Hippolyte Bernheim (1840-1919) of Nancy accused Charcot and his students of having inadvertently contaminated every one of their experiments on patients at the Salpêtrière. He argued that despite all their sensational reports, there was one simple explanation, and that was "suggestion." What Bernheim meant was that in every experiment Charcot or one of his students had carelessly dropped hints to patients as to what sort of "symptoms" their doctors expected of them. Instead of evidence from his patients, Bernheim claimed, Charcot had merely elicited elaborate "well-trained" performances. His patients, intentionally or not, simply put on a show,

²⁵ Pierre Janet, *L'automatisme psychologique: essai de psychologie expérimentale sur les formes inférieures de l'activité humaine* (Paris: Félix Alcan, 1889).

²⁶ Ellenberger, *The Discovery of the Unconscious*, 340.

and eager doctors like Charcot had willingly let themselves be entertained and ultimately deceived.²⁷

In the wake of Bernheim's biting critique, many of Charcot's former students cautiously distanced themselves from him.²⁸ Others, however, like Pierre Janet, rallied to his defense. During Charcot's final years, while trying to recover his reputation, Janet began publishing his research from Charcot's clinic for his medical thesis, *The Mental State of Hystericals* [*État mental des hystériques*].²⁹ Based on this clinical research, he stressed repeatedly that the hysterical symptoms he observed in Charcot's patients at the Salpêtrière were authentic and real. Naturally, he was familiar with Bernheim's critique. He admitted that hysteria was, in fact, difficult to observe and verify. Symptoms were often "contradictory." They "disappeared momentarily or changed their location with the greatest ease."³⁰ But that did not necessarily invalidate or disconfirm the diagnosis. Nor did it imply that the symptoms were simulated or only fabricated by the doctor's "power of suggestion." Hysteria, Janet declared, did not

²⁷ Harrington, *The Cure Within*, 57–60; Micale, *Approaching Hysteria*, 89; Goetz, Bonduelle, and Gelfand, *Charcot*, 200; Ellenberger, *The Discovery of the Unconscious*, 101.

²⁸ "Insensitive to issues of suggestion, he faltered scientifically, and consequently much of Charcot's work in this domain was superseded by Janet and Freud or dismissed by Babinski." Goetz, Bonduelle, and Gelfand, *Charcot*, 329.

²⁹ Janet, *État mental des hystériques. Les stigmates mentaux*; Pierre Janet, *État mental des hystériques. Les accidents mentaux*, vol. 2, 2 vols., Bibliothèque médicale Charcot-Debove (Paris: Rueff, 1894).

³⁰ Pierre Janet, "Anesthésie," ed. Charles Robert Richet, Paul Langlois, and Louis Édouard Lapicque, *Dictionnaire de Physiologie* (Paris: F. Alcan, 1895), 511.

depend on the stability of bodily symptoms. Indeed, it did not depend on the body but on the mind, a “specific mental state.”³¹ That was the definitive feature of hysteria.³²

The specific mental state was what Janet called “dissociation” or “disaggregation.” Similar to his first research subject, Léonie, whom he had been able to hypnotize and experiment on her “secondary consciousness,” Janet likewise believed that all hysterical patients, independent of hypnosis, suffered from a lack of integrated personal awareness. Their mind was “dissociated,” which meant that part of consciousness was “distracted” [*distrain*]. Such “a remarkable distraction” could lead also to a profound “indifference.”³³ Above all, they became “indifferent to their illness.”³⁴ This aspect of hysteria, Janet pointed out, was “well-known,” but he was the first to make it central to his diagnosis and, in particular, to use it as evidence that, regardless of “suggestion,” hysteria was very real.³⁵

The first individual instance of hysterical indifference that Janet discussed came from his older brother, Jules, who was also a physician in Paris at the time.³⁶ While Pierre taught philosophy and studied medicine at the Salpêtrière, Jules was an intern at another hospital nearby, the Pitié. There he met a patient whose case would become

³¹ Philippon and Poirier, *Joseph Babinski*, 313.

³² “Le mot « état mental d'une malade » doit désigner aujourd'hui pour le savant toutes les modifications qui peuvent survenir dans tous les phénomènes psychologiques, dans les sensations, les souvenirs, les perceptions, les associations d'idées.” Janet, *État mental des hystériques. Les stigmates mentaux*, 1:3.

³³ *Ibid.*, 1:52.

³⁴ Pierre Janet, “Quelques définitions récentes de l’hystérie,” *Archives de Neurologie* 26 (1893): 2.

³⁵ Janet, *L'automatisme psychologique*, 97.

³⁶ Ellenberger, *The Discovery of the Unconscious*, 334.

one of the cornerstones of his younger brother's psychopathology of hysteria. But although Pierre Janet credited his older brother for bringing the case to his attention, there is scant evidence of Jules Janet's own account of his patient. What follows, instead, is taken from a monograph written by Pierre Janet, entitled, "Hysterical Anaesthesia" [*L'Anesthésie hystérique*], which he presented one spring afternoon in 1892 at a weekly meeting inside the Salpêtrière.³⁷

A twenty year-old woman had tripped and fallen through the glass of a door, shattering it and badly cutting the inside of her right hand. By the time she arrived at the hospital, her hand was considerably swollen, but she was still able to move it. However, she complained of having no more feeling or sensation in the injured hand. This was not surprising, given the depth of the wound. What was surprising was that the young woman was also numb, "from top to bottom," on the entire left side of her body. And yet, she never uttered a word of it. According to Janet (it is not clear exactly which), "she was an hysteric."³⁸ He asked her, or imagined asking her,

How is it, mademoiselle, you have just complained about a numbness that covers such a tiny area in the palm of your right hand, but you do not even notice that you have absolutely no sensation in the left side of your body?³⁹

To which the woman replied, or "would have been able to reply with the utmost assurance,"⁴⁰

³⁷ Pierre Janet, *L'Anesthésie hystérique* (Paris: Progrès Médical, 1892).

³⁸ Janet, *État mental des hystériques. Les stigmates mentaux*, 1:8.

³⁹ *Ibid.*, 1:8–9; Pierre Janet, *The Mental State of Hystericals: A Study of Mental Stigmata and Mental Accidents* (New York: G. P. Putnam's sons, 1901), 15; Janet, *The Major Symptoms of Hysteria: Fifteen Lectures Given in the Medical School of Harvard University*, 163.

What do you care? I notice what I feel, my insensitivity in the palm of my right hand bothers me and my insensitivity on the left side of my body never has. You're the doctor, explain it as you like.⁴¹

Apparently, the young woman never noticed the numbness on her left side until her doctor brought it to her attention, but even then she still did not care. Explain as you like, she said—or Janet said she “would have been able to say.” He seemed to relish the irony of the situation.⁴² Here was this doctor, his older brother, who was more bothered by his patient's condition than she was. In fact, that was why he was interested in her condition: *because* she was not.

Pierre Janet would return to this case on numerous occasions in various lectures and other publications over the next decade.⁴³ The reason was that it supported his principal claim. Hysteria could be defined and, most importantly, it was *real*. After the story of the young woman, he concluded:

Hysterical anaesthesia, then, has, in all its forms, characteristics of its own...It is absolutely a matter of indifference to the patient, who, before it was pointed out to her, was even ignorant of it.⁴⁴

⁴⁰ “...à mon avis, elle aurait pu répondre avec plus d'assurance.” Janet, *L'Anesthésie hystérique*, 9.

⁴¹ *Ibid.*; Janet, *The Mental State of Hystericals*, 15; Janet, *The Major Symptoms of Hysteria: Fifteen Lectures Given in the Medical School of Harvard University*, 163.

⁴² The irony went deeper than probably either Janet appreciated. By virtue of the patient's lack of interest, we may say that the doctor's own was all the more accentuated, as if against a “blank canvas” of another consciousness. But more so, against the patient's lack of her own voice, Janet could ventriloquize her with what he imagined or wanted her to say. Janet L. Beizer, *Ventriloquized Bodies: Narratives of Hysteria in Nineteenth-Century France* (Ithaca: Cornell University Press, 1994), 127.

⁴³ He retold the story of this young woman almost verbatim during his guest lectures at Harvard Medical School in 1907. Janet, *The Major Symptoms of Hysteria: Fifteen Lectures Given in the Medical School of Harvard University*, 162–163.

⁴⁴ Janet, *L'Anesthésie hystérique*, 20; Janet, *The Mental State of Hystericals*, 16; Janet, *The Major Symptoms of Hysteria: Fifteen Lectures Given in the Medical School of Harvard University*, 161–162.

One of the central defining features of hysteria was this ingenuous “ignorance,” this innocent indifference. For Janet, it proved the patient’s sincerity. It underlined the fact that her condition was not fake or contrived but authentic and real. After all, he asked, “Do hystericals take any particular interest or pleasure in having their arms pierced through with needles?” Why would they feign their indifference if it only invited more pain? True, it also brought them attention, but was that enough? He continued,

Is it that they come to boast of their numbness? But I have already indicated that they are unaware of it. It is we who reveal it to them, and they who say to us: “If you are worried about our insensitivity, do not worry us with it, [for] we did not bring it to your attention and we are not anxious to be rid of it.”

This rendering of a hypothetical patient's response was Janet's way of emphasizing the authenticity of indifference. If a patient did not protest, how could she *not* be trusted? Such patients clearly did not care about their doctor's attention. And so, Janet reasoned, they were not malingering or dissimulating their symptoms. In other words, he implied, they were not to be *blamed*. Something else was at play beyond their control, even if still within their mind.⁴⁵

One of Janet's key sources for this argument was the physician Charles Lasègue (1816-1883). Lasègue recognized early on the importance of indifference in the making of hysteria. In 1864, he described a young woman from the Salpêtrière who was completely numb on the left side of her body without ever realizing it.⁴⁶ No matter what

⁴⁵ Rosenberg reflects on such moral implications of assigning blame and displacing agency to disease entities rather than human individuals. Rosenberg, “Contested Boundaries,” 420–421; Rosenberg, *The Trial of the Assassin Guiteau*, 64–67; Goldstein, *Console and Classify*, 50–51.

⁴⁶ Lasègue, “De l’anesthésie et de l’ataxie hystériques.”

he tried, whether he pinched, tickled, or even stabbed her with needles, he could not make her feel anything on the left half of her body. At one point, he picked up her arm and slammed it down hard on a nearby table. But still, she felt no pain and did not even register a response except for the loud slap of her hand hitting the tabletop.⁴⁷ From Lasègue's perspective, she "did not perceive such sensations which, in another state of mind, she would have been scarcely able to tolerate."⁴⁸ The cause of this "cutaneous anaesthesia," he decided, was hysteria. It created in her a state of "indifference," which made her severely "distracted" and "absent-minded" [*distrain*]⁴⁹—or literally, "pulled away" her attention from that half of her body.⁴⁹ Such mental detachment meant that she could no longer notice or perceive what went on in part of her body because a part of her consciousness was stretched out, "dis-tracted."⁵⁰

Three decades later, Janet looked to Lasègue and took up his language of "indifference" and "distraction," making them central concepts in his own definition of hysteria.⁵¹ He re-structured the theory of hysteria around such "mental states" as

⁴⁷ *Ibid.*, 386–388.

⁴⁸ *Ibid.*, 391.

⁴⁹ *Ibid.*; Charles Lasègue, "Catalepsies partielles et passagères," *Archives générales de médecine* 2 (1865): 385–402; Charles Lasègue, *Etudes médicales* (Paris: Asselin, 1884), 901–902.

⁵⁰ Crary contemplates some of the cultural and aesthetic appeal in depicting the loss of attention as the loss of "tension." Jonathan Crary, *Suspensions of Perception: Attention, Spectacle, and Modern Culture* (Cambridge, Mass: MIT Press, 1999); Michael Hagner and Cornelius Borck, "Mindful Practices: On the Neurosciences in the Twentieth Century," *Science in Context* 14, no. 04 (2001): 507–10; Shigehisa Kuriyama, "The Historical Origins of Katakori," *Japan Review* 9 (1997): 127–49; Sabine M. Arnaud, "Narratives and Politics of a Diagnosis: The Construction and Circulation of Hysteria as a Medical Category, 1730–1820" (Doctoral Dissertation, The City University of New York, 2007), 405.

⁵¹ Later, Janet wrote he was "happy to have M. Lasègue confirm an opinion which we have maintained for several years: hysterical anaesthesia is a certain species of absent-mindedness [*distrain*]." Janet, *The*

indifference, inattention, and distraction, and with them he re-drew Charcot's distinction between hysterical symptoms which were called "stigmata" versus those which were caused "accidentally" [*cause occasionnelle*] or by "traumatic" events in a patient's life. Janet explained the distinction this way,

Patients are aware of the accidents caused by a fixed idea, [whereas] patients with stigmata are so indifferent that most of the time they ignore them...The thought of the accident determines the nature of the symptom, in other words, the patient actualizes [*réalise*] his symptom as he thinks about it. On the contrary, we find in the stigmatic patient complications [*caractères compliqués*] about which the subject has no idea.⁵²

Unlike the traumatic type of hysteria in which the patient was often preoccupied with her condition, the "stigmatic" variety occurred because of some internal physiological "weakness" that prevented the patient from even *noticing* her condition until someone else bothered to point it out.⁵³ The difference between the two forms of hysteria, therefore, was not only in terms of causation, but also in terms of the patient's own experience of her illness.

However, not everyone was as attentive as Janet to the importance of indifference. In 1893, in the very same issue of the *Archives de Neurologie* in which

Mental State of Hystericals, 32; Charles Lasègue, "De la toux hystérique," *Archives générales de médecine* 5, no. 3 (1854): 513–31; Charles Lasègue, "Les Hystériques, leur perversité, leurs mensonges," *Annales médico-psychologiques*, 4th ser 6 (1881): 111–18; Goldstein, *Console and Classify*, 328; Goetz, Bonduelle, and Gelfand, *Charcot*, 175.

⁵² Janet, "Quelques définitions récentes de l'hystérie," 1893, 2.

⁵³ Janet alternately characterized the weakness as "cerebral," "moral," and "mental." For "moral weakness" [*faiblesse morale*], see, Janet, *L'automatisme psychologique*, 478; Janet, "Quelques définitions récentes de l'hystérie," 1893, 7. For "laziness" [*paresse*] and "lack of will" [*aboulie*], see, Janet, *État mental des hystériques. Les stigmates mentaux*, 1:122. For "mental weakness" [*faiblesse de synthèse psychique*], Janet, *L'automatisme psychologique*, 308. For "cerebral weakness" [*faiblesse cérébrale*], Janet, "Quelques définitions récentes de l'hystérie," 1893, 5.

Janet stressed the difference between hysterical stigmata and hysterical accidents, Sigmund Freud argued the exact opposite. The stigmata of “so-called non-traumatic” hysteria, he declared, were caused by the “same mechanism” as traumatic hysteria. In effect, there was no difference between the hysterical accident and the hysterical stigma.⁵⁴

It was not that Freud was unaware of Janet’s distinction. He and co-author Josef Breuer had earlier cited Janet’s experimental research, for which Janet thanked them, although in the same breath, he pointed out that Freud and Breuer had misunderstood some of his ideas, namely, the diagnostic difference between hysterical stigmata and accidents.⁵⁵ Ironically, Janet’s article was immediately followed by Freud’s, in which he even more pointedly ignored Janet’s distinction. However, he did agree with Janet on something. “I say with M. Janet,” he wrote, “that a banal, popular idea of the organs and of the body is at play in hysterical paralyses as well as hysterical anaesthesias.”⁵⁶ This “popular” idea, he continued, implicitly still in agreement with Janet, was “not based on a deep knowledge of the anatomy of the nerves but on our perceptions, tactile

⁵⁴ “Nous démontrons dans ce mémoire que les symptômes permanents de l’hystérie dite non traumatique s’expliquent (à part les stigmates) par le même mécanisme que Charcot a reconnu dans les paralysies traumatiques.” Sigmund Freud, “Quelques considérations pour une étude comparative des paralysies motrices organiques et hystériques,” *Archives de Neurologie* 26 (1893): 42.

⁵⁵ “Mais le travail le plus important qui soit venu confirmer nos anciennes études est sans contredit l’article de MM. Brener [sic] et Frennd [sic] récemment paru dans le *Neurologisches centralblatt*. Nous sommes très heureux que ces auteurs dans leurs recherches indépendantes aient pu avec autant de précision vérifier les nôtres et nous les remercions de leur aimable citation.” Pierre Janet, “Quelques définitions récentes de l’hystérie,” *Archives de Neurologie* 25 (1893): 437.

⁵⁶ Freud, “Quelques considérations pour une étude comparative des paralysies motrices organiques et hystériques,” 40.

and especially visual.”⁵⁷ Freud attributed this observation to Janet, while Janet, in turn, attributed it to Charcot.⁵⁸ The plot thickens, however. Freud neglected to mention Janet’s conclusion, “The patient is absolutely indifferent [and] even ignores [hysterical anaesthesia] until it is pointed out to her.”⁵⁹ For Janet, the patient’s indifference to her symptoms was part of her “ignorance” of her anatomy, or “anatomical construction of the organs.”⁶⁰ Not so for Freud.

Instead, he claimed in a “now-famous”⁶¹ passage that “hysteria behaves [*se comporte*] in its paralyses and other manifestations as if anatomy did not exist or as if it [*elle*] had no awareness of it.”⁶² It is important to note that Freud chose the word “hysteria” [*l’hystérie*], not “the hysteric” [*l’hystérique*]. He was saying that the illness itself, not the patient, “behaved” as if the anatomy did not exist. The disorder “had no

⁵⁷ “Je dis avec M. Janet, que c’est la conception banale, populaire des organes et du corps en général, qui est en jeu dans les paralysies hystériques comme dans les anesthésies, etc. Cette conception n’est pas fondée sur une connaissance approfondie de l’anatomie nerveuse mais sur nos perceptions tactiles et surtout visuelles.” Ibid.

⁵⁸ Jean-Martin Charcot, *Leçons du mardi à la Salpêtrière*, vol. 1, 2nd ed. (Paris: A. Delahaye et Emile Lecrosnier, 1887; repr., Paris: Progrès Médical, 1892), 225, 227; Janet, *L’automatisme psychologique*, 292; Janet, *État mental des hystériques. Les stigmates mentaux*, 1:6, 20ff.; Janet, “Anesthésie,” 510; Janet, *The Mental State of Hystericals*, 10; Janet, *The Major Symptoms of Hysteria: Fifteen Lectures Given in the Medical School of Harvard University*, 163.

⁵⁹ “Elle [*l’anesthésie hystérique*] est absolument indifférente au malade qu’il ignore même, avant qu’on ne la lui ait signalée.” Janet, *État mental des hystériques. Les stigmates mentaux*, 1:20.

⁶⁰ Ibid.

⁶¹ Harrington, *Medicine, Mind, and the Double Brain*, 251.

⁶² “J’affirme par contre que la lésion des paralysies hystériques doit être tout à fait indépendante de l’anatomie du système nerveux, puisque *l’hystérie se comporte dans ses paralysies et autres manifestations comme si l’anatomie n’existait pas, ou comme si elle n’en avait nulle connaissance.*” (italics original) Freud, “Quelques considérations pour une étude comparative des paralysies motrices organiques et hystériques,” 39.

awareness." It [*elle*] was the one that did not understand the rules of anatomy.⁶³

Assuming Freud understood the rules of French grammar, he appears to have intentionally personified hysteria. The effect was that he seemed less interested in whether the patient herself, "the hysteric," was aware of her symptoms. That species of awareness mattered little to the diagnosis since Freud did not share Janet's distinction between the two types of hysteria, stigma and trauma, which depended on the *patient's* awareness. For Freud, the diagnosis was the same, regardless of the patient's awareness, indifference or no.

It may come as something of a surprise that Freud, not Janet, is the one most cited for the phrase, "*belle indifférence*."⁶⁴ However, not even Freud himself, proprietary though he was, claimed authorship. Nor did he credit Janet and Lasègue.⁶⁵ Instead, he claimed Charcot had once used the phrase in one of his lectures, although there is no written record of it.⁶⁶

⁶³ Usually authors choose to translate Freud's "*hystérie*" as "the hysteric." Philippon and Poirier, *Joseph Babinski*, 316; Harrington, *Medicine, Mind, and the Double Brain*, 251; Goetz, Bonduelle, and Gelfand, *Charcot*, 207.

⁶⁴ Josef Breuer and Sigmund Freud, "Fräulein Elisabeth von. R... (Freud)," in *Studien über Hysterie* (Leipzig: Franz Deuticke, 1895), 116; Joseph Breuer and Sigmund Freud, *Studies on Hysteria* (New York: Basic Books, 2009), 135; Sigmund Freud, "Die Verdrängung," *Internationale Zeitschrift Für Psychoanalyse* 3, no. 3 (1915): 136; Sigmund Freud, "Repression," in *The Standard Edition of the Complete Psychological Works of Sigmund Freud*, ed. James Strachey and Anna Freud, vol. 14 (London: Hogarth Press, 1953), 156.

⁶⁵ Another physician who discussed the phenomenon of indifference, but whom neither Freud nor Janet cited, was another former student of Charcot, Albert Pitres (1848-1928). Albert Pitres, *Des anesthésies hystériques*, Clinique médicale de l'hôpital Saint-André (Bordeaux: G. Gounouilhou, 1887), 26–28.

⁶⁶ In fact, the only allusion Charcot made to it was in deference to Lasègue, whom he acknowledged had "very judiciously" urged that doctors actively look for signs of hysterical anaesthesia among patients who, "once its existence was revealed to them, showed their total surprise." There is no explicit mention of *indifférence*, however. Jean-Martin Charcot, *Leçons sur les maladies du système nerveux* (Paris: Progrès Médical, 1886); Cf. Jon Stone et al., "La Belle Indifférence in Conversion Symptoms and Hysteria," *The British Journal of Psychiatry* 188, no. 3 (March 1, 2006): 204–209.

Pierre Janet's contribution to the study of hysterical indifference is often overshadowed by Freud's. However, Janet was the one who analyzed it most and made the most use of it.⁶⁷ Indeed, he used it not only for clinical purposes, that is, to argue that hysteria was real, but also for philosophical ends, to argue for the "unity of the self" [*l'unité du moi*].⁶⁸ Because Janet's initial decision to study medicine stemmed from his early philosophical interests in perception and psychological experiments on consciousness, it will help to consider more closely some of his philosophical ideas about attention and indifference, consciousness and the "subconscious," and finally his concept of self-perception.

The Unity of the Self

Above all, Pierre Janet's uncle, Paul Janet (1823-1899), helped shape his nephew's early ideas about consciousness, perception, and the self.⁶⁹ Paul Janet was a professor of philosophy at the Sorbonne and a member of the *Institut de France*. It was he, above all, who helped launch Pierre Janet's career by presenting his nephew's first paper on the hypnosis of Léonie before an audience in Paris, including Charcot. Paul Janet also wrote a number of books on philosophy and the history of philosophy,

⁶⁷ One historian of psychiatry, Mark Micale, briefly mentions Janet's, not just Charcot's, role in the study of indifference. Micale, *Approaching Hysteria*, 110.

⁶⁸ Janet, *L'automatisme psychologique*, 435.

⁶⁹ Ellenberger, *The Discovery of the Unconscious*, 334, 356–357; Carroy and Plas, "How Pierre Janet Used Pathological Psychology to Save the Philosophical Self"; Jacqueline Carroy and Régine Plas, "La genèse de la notion de dissociation chez Pierre Janet et ses enjeux," *Évolution psychiatrique* 65, no. 1 (2000): 9–18.

including a critique of scientific materialism.⁷⁰ He ascribed to the school of “spiritualist” psychology, also known as “eclectic” philosophy, which was founded earlier that century by his predecessor at the Sorbonne, the philosopher Victor Cousin.⁷¹ According to Cousin, and Janet after him, it was possible to gain insight into one’s mind and thereby to examine the contents of consciousness based on careful self-observation, or direct introspection. This confidence in the mind's transparency to itself translated, for Janet, into the mind's *unity* of the self. The mind, ultimately the self, was *indivisible*. Thus, it could be examined by oneself. The “inner life” [*la vie intérieure*] was open to anyone trained in careful self-reflection and introspection.⁷²

However, French spiritualist psychology came under repeated attacks. Foremost among the critics was Auguste Comte (1798-1857) who ridiculed the claim of introspection as a “manifest impossibility.”⁷³ No one could observe himself and examine his mental state without interrupting the state of mind he presumed to observe. “This

⁷⁰ Paul Janet, *Le matérialisme contemporain en Allemagne; examen du système du docteur Büchner*, Bibliothèque de philosophie contemporaine (Paris: G. Baillière, 1864).

⁷¹ Goldstein, *The Post-Revolutionary Self*, passim, 165–171ff.

⁷² The historian Jan Goldstein has challenged this way of reasoning by arguing that a “systematic awareness of mental interiority” did not presuppose or require the belief in “a single tight-knit unit” or self. She writes, “But consciousness alone does not necessarily safeguard the integrity of the self.” Part of the reason Goldstein argues in favor of this disconnect may reflect a modern wariness of yoking the difficult idea of an “extended self,” that is, a sort of soul-in-the-world, to the need to believe our feelings are still someone “ours,” whoever “we” may be from one moment to the next. *Ibid.*, 6–7; Franz Clemens Brentano, *Psychology from an Empirical Standpoint*, ed. Oskar Kraus and Linda L. McAlister, trans. Antos C. Rancurello, D.B. Terrell, and Linda L. McAlister, Paperback ed, International Library of Philosophy (London: Routledge, 1995), 130, 134–135, 168–169ff.; James, *The Principles of Psychology*, 1:189; Chisholm, “The Self in Austrian Philosophy,” 158.

⁷³ James, *The Principles of Psychology*, 1:quoted on 188.

pretended psychological method,” Comte ruled, “is then radically null and void.”⁷⁴ Such withering conclusion, therefore, made it difficult to defend psychology as a “science.” Indeed, Comte wanted to eliminate the very word “psychology.”⁷⁵ According to his version of the sciences, all knowledge must be based on empirical observation, and since he believed that no psychologist could ever directly peer inside the mind and observe it at work, he concluded there was no place for psychology in this epistemological schema he called “positivism.”⁷⁶

Psychological philosophers like Paul Janet, however, appealed Comte's harsh verdict. Janet insisted on psychology's legitimacy, and he argued that even introspective psychology was compatible with the “new” experimental psychology developed by Hippolyte Taine (1828-1893) and Théodule Ribot (1839-1916).⁷⁷ This catholic approach of embracing both philosophy and psychology, insisting on both the unity of the mind and the positivist-like⁷⁸ penchant for empirical experimentation, Paul Janet passed on to his nephew.⁷⁹ Judging from the clever reading proposed by historians Jacqueline Carroy

⁷⁴ Ibid.

⁷⁵ Carroy and Plas, “How Pierre Janet Used Pathological Psychology to Save the Philosophical Self,” 234; Vincent Guillin, “La fille de la servante. Science, psychologie et philosophie chez Théodule Ribot et William James” (Doctoral Dissertation, Université de Paris 1 Panthéon-Sorbonne, 1998), 91.

⁷⁶ Mary Pickering, *Auguste Comte: Volume 1: An Intellectual Biography* (Cambridge: Cambridge University Press, 1993); Thomas Meaney, “The Religion of Science and Its High Priest,” *The New York Review of Books*, October 25, 2012.

⁷⁷ Carroy and Plas, “How Pierre Janet Used Pathological Psychology to Save the Philosophical Self,” 234.

⁷⁸ Carroy and Plas contest the description of Taine and Ribot as “positivists.” Instead, they suggest the term “positive psychologists.” I prefer the terms quasi-positivist or positivist-like, in order to preserve the partial resemblance to Comte's positivism, not only in Taine and Ribot but also in Paul Janet's own openness to experimental research. Ibid.

and Régine Plas, Pierre Janet similarly balanced his work between semi-spiritualist, philosophical convictions—namely, in the unity of the self—and his scientific experimentalist convictions, which made him suspicious of introspection.⁸⁰

Janet held together these two somewhat disparate belief systems, indeed, divergent ways of making knowledge, despite others who lobbied ultimately for the separation of experimental psychology from philosophy, particularly in the universities.⁸¹ Janet's attempted rapprochement between psychological philosophy, experimental psychology, and clinical psychopathology was, therefore, increasingly rare. Nevertheless, he managed to defend a spiritualist-like belief in the unity of the self alongside a positivist-like disavowal of introspection with the help of an idea he called “personal perception” [*perception personnelle*].⁸²

This kind of perception served to “synthesize” [*synthétiser*] and “connect” [*rattacher*] every new sensation of the body to the “self or personal consciousness” [*le moi ou la personnalité*].⁸³ One could not have any sense of the self, he went on to say, were it not for the activity of personal perception that collected and united all

⁷⁹ Carroy and Plas goes so far as to suggest that it might even look as if Pierre Janet “plagiarized” his uncle. *Ibid.*, 236.

⁸⁰ *Ibid.*, 237.

⁸¹ *Ibid.*

⁸² Janet, *L'automatisme psychologique*, 435; Janet, *État mental des hystériques. Les stigmates mentaux*, 1:41; *ibid.*, 1:30; Janet, *État mental des hystériques. Les accidents mentaux*, 2:38, 40.

⁸³ Janet, *L'automatisme psychologique*, 306; Janet, *État mental des hystériques. Les accidents mentaux*, 2:38.

sensations into this “communion.”⁸⁴ And yet, perception was not possible without the self through which it cohered. It was impossible to perceive anything without implying oneself in the act of perceiving. That was the essence of personal perception for Janet. Always implicit in every act of perception was the ability to say, “*I feel, I see.*”⁸⁵ That was what made perception “personal.” It was not so much about the perception *of* the self as it was perception-*with*-the-self, or by virtue of having a self. That was the subtle difference which allowed Janet to distance himself from the spiritualist belief in introspection, at the same time it let him preserve his belief in an indivisible self.

Janet developed this idea of perception that derived from the unity of self based on the works of the seventeenth-century philosopher-priest Nicolas Malebranche (1638-1715). In 1886, he edited a volume by Malebranche to use for teaching his students of philosophy.⁸⁶ Malebranche tried to integrate the teachings of St. Augustine and Descartes, arguing that perception proved the existence of the soul. What but the soul, endowed by God, could account for the experience of consciousness? he reasoned. For Malebranche, perception ultimately came from, or was “powered” by, God.⁸⁷ This style of reasoning appears to have appealed to Janet, although he made little recourse to either the terms “God” or “soul” in his own writing. Still, he followed Malebranche in

⁸⁴ Janet, *L'automatisme psychologique*, 38.

⁸⁵ *Ibid.*, 306; Janet, *État mental des hystériques. Les stigmates mentaux*, 1:18, 39, 41.

⁸⁶ Nicolas Malebranche, *De la recherche de la vérité: Livre II, première partie (chap. I et V), deuxième et troisième parties*, ed. Pierre Janet (Paris: F. Alcan, 1886); Ellenberger, *The Discovery of the Unconscious*, 337.

⁸⁷ Taylor, *Sources of the Self*, 119, 129ff.

his argument that perception was not enough by itself. Something else had to enable it, to direct it, and allow it to cohere. For Janet, that something else was “the self or the personal consciousness.”⁸⁸ This was an abrupt about-face from the stance of Ernst Mach who, in the same year of 1886, triumphantly declared the self “unsaveable.”⁸⁹ Even so, Janet was poised to “save” it.

Janet, like many psychologists at the time, wanted to examine cases of individuals who seemed to have completely lost their sense of self.⁹⁰ That was why he later pursued the psychopathology of hysteria. It was also why he was especially interested in the symptoms of hysterical anaesthesia. In such cases, patients appeared to be numb without any clear explanation. Their peripheral nerves were not damaged and their body responded in the usual ways to artificial electrical stimulation, and yet the patients themselves never seemed to feel anything. The indifferent hysteric no longer cared what happened to certain areas of her body, as if she lacked the sense of it belonging to her and her self, as if she no longer possessed a coherent self.

For some psychologists, like Taine and Ribot, this was precisely their interpretation. Patients did not just *seem* to lose a part of themselves, they *did* lose a part of themselves. And that was because, in Taine and Ribot’s view, the self consisted of nothing more than *parts*. It was not indivisible, as the spiritualists believed. Cases of hysterical anaesthesia helped to reinforce the disintegration of the idea of the self.

⁸⁸ Janet, *L’automatisme psychologique*, 306.

⁸⁹ Mach, *Beiträge zur Analyse der Empfindungen*, 18; Ernst Mach, *L’Analyse des sensations: le rapport du physique au psychique* (Nîmes, France: Editions Jacqueline Chambon, 1996).

⁹⁰ Carroy and Plas, “How Pierre Janet Used Pathological Psychology to Save the Philosophical Self,” 234.

Consciousness was divisible all the way down. However, Janet looked for a different explanation. In keeping with his belief in the unity of the self, he claimed that consciousness was divisible only by *degrees*, not absolutely.⁹¹ No patient with hysteria was irrevocably unconscious.⁹² Rather, it was more accurate to say that they were *distracted* [*distract*]. That meant their overall “field of awareness” was “narrowed,” or “contracted,” but it did not mean it was permanently lost.⁹³

This language was significant. It helped Janet argue that the sense of self, even if never directly attainable through introspection, always remained *intact*. It remained indivisibly unified. That was the crucial implication in his portrayal of “*indifférence*.” Hysterics might seem as if they had lost awareness of one half of their body, but in fact, that awareness was merely distracted or “pulled away” in another direction from the sensations still coming from that region of the body.⁹⁴ Ultimately, they remained aware *on some level* and conscious *to some degree*, although no longer enough to be able to say, “It is *I* who feel, it is *I* see.”⁹⁵

⁹¹ Ibid.

⁹² “Chez ces personnes, en effet, aucune sensation n'est perpétuellement inconsciente, elle ne l'est que momentanément.” Janet, *L'automatisme psychologique*, 310.

⁹³ “*rétrécissement du champ de la conscience*.” Ibid., 308; Janet, “Quelques définitions récentes de l'hystérie,” 1893, 7.

⁹⁴ “Cet individu qui a déjà le champ de conscience très rétréci n'est pas un anesthésique, c'est simplement un *distract*.....On lui pince le bras gauche, on lui demande s'il sent le pincement, et à sa grande surprise, le patient constate qu'il ne sait plus sentir consciemment.” Janet, *État mental des hystériques. Les stigmates mentaux*, 1:22.

⁹⁵ Janet, *The Major Symptoms of Hysteria: Fifteen Lectures Given in the Medical School of Harvard University*, 171–172.

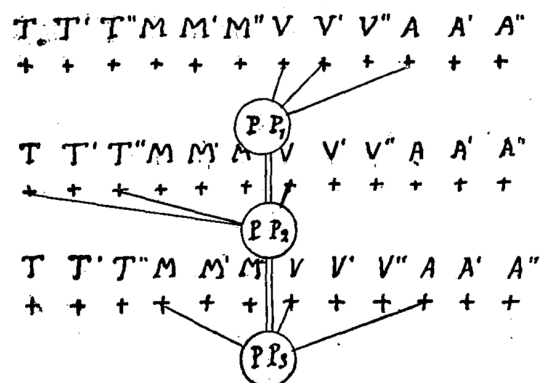


FIG. 3.

Janet illustrated this distinction in a schematic drawing (above). The lines indicated which sensations were still connected by way of the “personal perception” (“PP”) to the self or personality, and thus remained possible for the patient to recognize as hers. (The T's represented different tactile sensations, the V's visual, the A's auditory, and the M's sensations in the muscles.) Janet predicted the patient would feel able to say of the personally connected sensations: “I felt those, I was aware of them.”⁹⁷ For the sensations which were not connected by way of personal perception, however, the patient would claim that “he does not know what we are talking about and he has no awareness of them at all.”⁹⁸ Still, Janet maintained, they were conscious to a lesser degree (“in our hypothesis, they are also conscious sensations.”).⁹⁹ Even though they lay “outside the personal consciousness” [*en dehors de la personnalité*], they lingered in

⁹⁶ Fig. 2: Schematic of “personal perception” in Janet, *État mental des hystériques. Les stigmates mentaux*, 1:41.

⁹⁷ Janet, *L'automatisme psychologique*, 308; Janet, *État mental des hystériques. Les stigmates mentaux*, 1:41.

⁹⁸ Janet, *L'automatisme psychologique*, 308.

⁹⁹ *Ibid.*

consciousness, albeit in a weakened and distracted form. They were what Janet described as “*subconscious*.”¹⁰⁰

In this “particular state among hysterics,” Janet concluded, consciousness remained intact despite its diminished “field” [*champ*]. Patients only appeared to be unaware, he insisted, because some sensations no longer were “connected” [*rattaché*] to the personal part of their perceptual consciousness. Momentarily, they could no longer say of these sensations, “I perceive.” Their attention was weak, but they were not completely distracted, not permanently unaware.

Such was Janet’s account of hysterical indifference. Ultimately, it was a way to reconcile his philosophical conviction in the unity of the mind with his psychopathology of the hysterical, distracted mind. His explanation of “*indifférence*,” therefore, served as an important pivot-point which helped hold together Janet's theoretical commitments as well as his research activities, both as a philosopher and physician.

The Dismemberment of Hysteria

Janet was far from the only doctor who tried to use hysteria as a window to peer into the mind. Of course, there was also Freud, though he was arguably more interested in the effect of memory in hysterical traumas than the effect on perception in hysterical stigmata. (But then again, he equated them all to trauma.) Besides Janet and Freud, however, there was still another who was also a former disciple of Charcot and intent

¹⁰⁰ Ibid., 337; Janet, *État mental des hystériques. Les accidents mentaux*, 2:30ff., 124.

on exploiting hysteria for its secrets. He was the French-Polish physician, Joseph Jules François Félix Babinski.

From 1885 to 1887, at the height of Charcot's career, Babinski worked for him as the operating head of the clinic [*chef de clinique*]. This period covered the short stretch of time during which Freud, on leaving Meynert's clinic in Vienna, visited Charcot's clinic from October 1885 until February 1886, although there is little record of Freud's interaction with either Babinski or Charcot. As historian Henri Ellenberger notes, the relationship between Freud and Charcot, if it can be called a "relationship," was more of an "existential encounter" than one of "master and disciple."¹⁰¹ The latter characterization was more apt for Charcot and Babinski. But unlike Freud or Janet, each of whom remained loyal "disciples" of Charcot long past his death, Babinski "the favored" would ultimately turn his back on "his old Master."¹⁰²

Some historians suggest Babinski felt "foolish and betrayed" in the wake of Bernheim's "devastating" critique.¹⁰³ Others have even speculated that he later developed a sort of "Oedipal rage" toward Charcot.¹⁰⁴ Upon further scrutiny, however, the situation appears to have been more complicated. To be sure, Babinski worked under Charcot in the best of times, and later perhaps also in the worst of times. Despite

¹⁰¹ Ellenberger, *The Discovery of the Unconscious*, 436.

¹⁰² Roudinesco, *La bataille de cent ans*, 68; Harrington, *Medicine, Mind, and the Double Brain*, 250.

¹⁰³ Harrington, *Medicine, Mind, and the Double Brain*, 249.

¹⁰⁴ Hacking, *Rewriting the Soul: Multiple Personality and the Sciences of Memory*, 135; Ian Hacking, *Mad Travelers: Reflections on the Reality of Transient Mental Illnesses* (Charlottesville, VA: University of Virginia Press, 1998), 71–72.

the criticism fulminating from Nancy where Bernheim held court, the young Babinski initially rallied to the defense of Charcot.

In 1886, he reported one of the boldest experiments on hysteria in hopes of refuting Bernheim's accusations and shoring up Charcot's reputation.¹⁰⁵ He started by having two young hysterical women from the Salpêtrière sit in chairs with their backs to one another. Both women suffered from a form of hemianaesthesia, but the first patient was affected on the right side of her body, whereas it had struck the second patient only on her left side. With them seated but not touching, Babinski then took out a magnet and placed it on the affected side of the first patient. After leaving it on her for a few seconds, he then removed it and walked over to the second patient to place the magnet on her unaffected right side. Quite rapidly, Babinski claimed, the second patient contracted the right-sided hemianaesthesia of the first patient via the exposure to the magnet. Meanwhile, the first patient regained sensation of her right side, while the second woman lost sensation on *both* sides of her body. In effect, he had "cured" the first patient, while leaving the second in a state of "total, complete" anaesthesia. Presumably with the use of a mere magnet, Babinski declared to have "transferred" hysterical symptoms between two people.¹⁰⁶

Charcot conducted a similar experiment ten years earlier, which was precisely the experiment that initially drew Bernheim's attention and later his attack. However,

¹⁰⁵ Joseph Babinski, "Recherches servants a etablir que certaines manifestations hysteriques peuvent etre transferees d'un sujet a un autre sujet sous l'influence de l'aimant," *Le Progrès Médical*, 2, 4, no. 47 (November 30, 1886): 1010–11.

¹⁰⁶ *Ibid.*, 1011; Harrington, "Metals and Magnets in Medicine," 32.

Charcot had only claimed to have transferred symptoms from one side to the other of the *same* patient's body, not two different patients as Babinski had done. When Bernheim tried to replicate Charcot's experiment, he failed and eventually redirected his energies to dispute Charcot's methods. Apparently, this criticism of Charcot did not faze the young Babinski—at least not at first. Despite Bernheim's belief that all hysterical symptoms, transferred or not, were due to "suggestion," Babinski insisted that his patients' hysteria was "founded on a somatic character" and "independent of all suggestion."¹⁰⁷ Neither suggestion nor "artificial" simulation could account for it. That was 1886. Six years later, Babinski appeared to change his mind.

By 1892, Charcot wielded considerably less influence after weathering years of Bernheim's and others' critical attention. Even so, Babinski did not abandon his mentor in the midst of this public *dénouement*, not yet. The young doctor's career still depended on the support of France's first academic neurologist. In March that year, Babinski took the examination to qualify as a candidate to join the medical faculty and teach in Paris. However, another one of Charcot's former assistants, Charles Bouchard, presided over the board of examiners and held a deep grudge against Charcot out of rivalry or jealousy or disputed priority.¹⁰⁸ According to multiple sources consulted at the time, Bouchard used his authority as president of the board to fail Babinski on the

¹⁰⁷ "Nous avons pris des malades hystériques, hommes ou femmes, présentant des manifestations hystériques...non plus artificielles, mais naturelles, c'est-à-dire survenues indépendamment de toute suggestion de notre part." Babinski, "Recherches servant à établir que certaines manifestations hystériques peuvent être transférées d'un sujet à un autre sujet sous l'influence de l'aimant," 1011.

¹⁰⁸ It is unclear which, though probably jealousy above all. The dispute over priority involved their former collaboration in discovering the causal connection between military aneurysms and cerebral hemorrhage. Vicente J. Iragui, "The Charcot-Bouchard Controversy," *Archives of Neurology* 43, no. 3 (1986): 292.

exam.¹⁰⁹ And although Charcot appealed repeatedly to other members of the board on Babinski's behalf, it came to no avail. Charcot, "the master of hysteria,"¹¹⁰ no longer commanded the same respect as a few years earlier. Babinski remained disqualified from joining the medical faculty and never took the exam again. A year later, Charcot unexpectedly passed away. Thereafter, Babinski would have to fend for himself outside of the university system with a conspicuous "lack of academic credentials" and little prospect of attracting many students.¹¹¹

Even before Charcot's sudden death, Babinski could already read the writing on the wall. After his rejection by the board of examiners in the spring of 1892, he knew he could no longer count on Charcot. In the fall of that year, he published an article that suggested he was beginning to lean in favor of Bernheim's theory of suggestion. His paper was on the co-existence, or "association," of hysterical and "organic" symptoms of the nervous system.¹¹² In it, he made the surprising concession that perhaps Bernheim was "right."¹¹³ Quoting him, Babinski wrote that hysterical symptoms were

¹⁰⁹ One of the board members was unable to attend due to sudden sickness, and instead of Bouchard finding a substitute, he ruled to continue with the examination, which culminated in a tied vote that gave him the final say. *Ibid.*

¹¹⁰ Goetz, Bonduelle, and Gelfand, *Charcot*, ix.

¹¹¹ According to Iragai, Charcot's death was a major factor in Babinski's decision not to retake the examination, "since he did not have the backing necessary to succeed." The effect on his career was a "lack of academic credentials" which "deprived him of pupils." Iragai, "The Charcot-Bouchard Controversy," 294.

¹¹² Joseph Babinski, "Association de l'hystérie avec les maladies organiques du système nerveux, les névroses et diverses autres affections," *Société médicale des hôpitaux*, November 11, 1892, 775–97.

¹¹³ *Ibid.*, 779n2.

treatable “independent of any direct material intervention” (such as magnets?).¹¹⁴ Treatment could determine and “disentangle” [*démêler*] hysterical and organic symptoms.¹¹⁵ While the former responded to psychotherapy [*psychothérapie*], the latter never would. Then he added another concession that revealed his change of opinion. “It seems to me,” he wrote, “suggestion has had the effect of clearing the way [*déblayer le terrain*] by eliminating only those problems which are related to hysteria.”¹¹⁶ Finally, Babinski had acknowledged the role of suggestion in hysteria. And this was only the beginning.



Fig. 17. — *Phénomène des orteils* (Babinski) [165] [167].

Fig. 3: Illustration of the Babinski reflex test and “positive” response.¹¹⁷

¹¹⁴ Ibid., quoted on 779n2.

¹¹⁵ Ibid., 777.

¹¹⁶ Ibid., 789; Philippon and Poirier, *Joseph Babinski*, 44–45; Joost Haan, Peter J. Koehler, and Julien Bogousslavsky, “Neurology and Surrealism: André Breton and Joseph Babinski,” *Brain* 135, no. 12 (2012): 3830–38.

¹¹⁷ J. Babinski, *Hystérie-pithiatisme et troubles nerveux d’ordre réflexe en neurologie de guerre*, Collection horizon; précis de médecine et de chirurgie de guerre (Paris: Masson et cie, 1917), 178.

In 1896, Babinski made a discovery that changed the way he approached hysteria for the rest of his career. He found that by stroking the bottom of a patient's foot, he could induce a dramatic response. Either the patient's foot flexed downward, curving around the arch, or the big toe flexed upward, away from the bottom of the foot. The latter reflex, he noticed, only occurred in patients who suffered nerve damage in the pyramidal motor neurons of the corticospinal tract. The former reflex indicated healthy nerves, at least in the pyramidal tract.¹¹⁸ Many physicians quickly recognized the diagnostic utility of such a test. Already by 1900, it became one of the most important and reliable techniques to help doctors determine the cause of a variety of contractures, palsies, and plegias by systematically ruling out hysteria if there was a “positive” or pathological sign of the big toe-reflex—thus, earning hysteria the “negative” distinction.¹¹⁹ Ultimately, the “Babinski response,” as it was later called, paved the way for his re-definition and re-translation of hysteria as antithetical to all organic disease and, moreover, entirely a product of the mind.¹²⁰

¹¹⁸ Joseph Babinski, “Sur le réflexe cutané plantaire dans certains affections organiques du système nerveux central,” *Comptes-rendus hebdomadaires des séances et mémoires de la Société de Biologie* 3, no. 48 (1896): 207–8.

¹¹⁹ Roudinesco, *La bataille de cent ans*, 68; Philippon and Poirier, *Joseph Babinski*, 318.

¹²⁰ Joseph Babinski, “Diagnostic différentiel de l’hémiplégie organique et de l’hémiplégie hystérique,” *La Lancette française. Gazette des hôpitaux civils et militaires* 73 (1900): 521–27, 533–37; Joseph Babinski, “Définition de l’hystérie,” *Revue neurologique* 9 (1901): 1074–80; Joseph Babinski, “Introduction à la semiologie des maladies du système nerveux. Des symptômes objectifs que la volonté est en capable de reproduire de leur importance en médecine légale,” *Gazette des Hôpitaux*, October 11, 1904; P.W. Nathan and M.C. Smith, “The Babinski Response: A Review and New Observations,” *Journal of Neurology, Neurosurgery & Psychiatry* 18, no. 4 (1955): 250; M.S. Okun and P.J. Koehler, “Babinski’s Clinical Differentiation of Organic Paralysis from Hysterical Paralysis: Effect on US Neurology,” *Archives of Neurology* 61, no. 5 (2004): 778.

In 1901, Babinski proposed to replace the term “hysterical” with a new word of his own making, “*pithiatique*,” which he translated as “able to be persuaded.”¹²¹ He argued that the only reliable definition of hysteria, or what he preferred to call “pithiatism,” was that its symptoms were “all susceptible of disappearing under the *exclusive* influence of persuasion.”¹²² There was nothing “organic” or “material” about it, neither in cause nor in cure. It originated completely within the patient's mind as a “mental affection,” created by an act of what he called, in keeping with Bernheim, “autosuggestion” [*s'autosuggestionner*].¹²³ The symptoms of hysteria, therefore, were not only suggested to the patient but also *by* the patient. It was *she* who allowed herself to be persuaded—by herself. Every symptom was, in a sense, voluntary, even simulated. That is, hysterical, or “pithiatic,” symptoms were mere imitations of organic symptoms due to nerve damage, and with the aid of his reflex test, Babinski now could expose their true “psychic” origins. Even if the patient was not expressly aware of simulating, he was still partially responsible and blameworthy on some level. He was what Babinski called a “semi-simulator” [*demi-simulateur*].¹²⁴

This rather stark formulation of hysteria helped “clear the way” indeed. It simplified the complicated schematics that others like Pierre Janet and Sigmund Freud retained from Charcot's work. Hereafter, Babinski proclaimed, the only important

¹²¹ Babinski, “Définition de l'hystérie,” 1079.

¹²² *Ibid.*, 1076.

¹²³ *Ibid.*, 1079, 1077.

¹²⁴ Joseph Babinski, “Démembrement de l'hystérie traditionnelle: pithiatisme,” *Semaine médicale* 29 (1909): 4; Philippon and Poirier, *Joseph Babinski*, 319.

difference between hysterical and organic symptoms was the absence or presence of the will [*la volonté*].¹²⁵ If the reflex responses were disturbed, then it could never be a question of hysteria. Only hysteria, or pithiatism, lay within the patient's control, a product of the mind, and subject of the will.¹²⁶

After 1901, Babinski stepped up his critique of the hysteria diagnosis and continued to promote its replacement with his new term, pithiatism. In 1908, he made his break from Charcot official, or at least most explicit. He called for not only the elimination of the word “hysteria” but moreover its “dismemberment.” He wanted to tear apart, limb from limb, symptom and sign, the entire diagnostic construct.¹²⁷ He confessed that while he had once been “impregnated” [*impregné*] by Charcot's ideas at the start of his career, recent research compelled him to “abandon the doctrine of my

¹²⁵ Joseph Babinski, “De la migraine ophthalmique hystérique,” *Archives de Neurologie* 20, no. 60 (November 1890): 312; Joseph Babinski, “Contractures organique et hystérique,” *Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris Séance* 10 (1893): 341; Babinski, “Introduction à la semiologie des maladies du système nerveux. Des symptômes objectifs que la volonté est en capable de reproduire de leur importance en médecine légale,” 1126.

¹²⁶ Some commentators seem eager to discern in Babinski's stark separation of hysteria and organic disorders a prelude or parallel to the presumably stark separation between French neurology and psychiatry. However, history is messier and does not admit of such differential diagnoses. For one, Babinski could never become a neurologist in the academic capacity, and he continued to receive patients even with non-neurological disorders. Moreover, he did not wish to let go of hysteria as a neurological diagnosis, or at least not to psychiatry as such. But these terms “neurology” and “psychiatry” were much, much more porous than they sound to us today. That is not to say that there were not differences in terms of doctors' training and preferred forms of treatment, their alliances and their theories of aetiology and nosology. But it is to temper the temptation of reading into one diagnosis a prophetic parallel in the entire medical profession. For such premonitory portrayals, see, Henri Baruk, “Neurologie et psychiatrie. Données historiques et actuelles,” *Annales Médico-Psychologiques* 2, no. 4 (1968): 533–534; Roudinesco, *La bataille de cent ans*, 72–73.

¹²⁷ “[C]'est surtout pare qu'il me fournit l'occasion, à la veille du jour où va s'engager une discussion générale sur le démembrement et la délimitation de l'hystérie, de préciser ma pensée sur un point qui pourrait prêter à confusion.” Joseph Babinski, “Instabilité hystérique (pithiatique) des membres et du tronc,” *Revue neurologique* 16, no. 6 (March 30, 1908): 260–261.

illustrious master.”¹²⁸ Despite Charcot's mistakes, which were typically “glossed over,” Babinski still maintained a “deep admiration for the great neurologist.”¹²⁹ But the “inevitable” time had finally come to “renounce” hysteria. Too many hysterical symptoms were mistaken for organic disorders, and a number of organic symptoms continued to be confused with hysteria. Now it was time to dismantle the diagnosis altogether and to reclaim those symptoms which were in reality organic, despite having been “grafted” into hysteria.¹³⁰ And so, it was not only a matter of dismembering, but also “re-membering,” or re-building, new disorders out of the old members of hysteria, especially those which were believed to be organic and therefore belonged to diseases of the brain and nerves.

Whether or not Babinski harbored “Oedipal rage” toward Charcot is somewhat beside the point. He was certainly ambitious for wanting to overhaul the “ungainly” and “excessive” diagnosis of hysteria.¹³¹ And he was increasingly cautious toward patients who may have been simulating. Likewise, he grew more attentive and vigilant toward

¹²⁸ Babinski, “Démembrement de l’hystérie traditionnelle,” 3.

¹²⁹ “[J]e n'en conserve pas moins—je tiens à le dire—une admiration profonde pour le grand neurologue dont les travaux sur l'hystérie, fort importants, d'ailleurs, malgré les erreurs qui s'y sont glissées, ne constituent qu'une faible partie d'une oeuvre imposante.” Ibid.

¹³⁰ Babinski, “Association de l’hystérie avec les maladies organiques du système nerveux, les névroses et diverses autres affections,” 781. It should be added that Janet himself anticipated a distant day in the future when the hysteria diagnosis would be dismantled: “Sans doute, il arrivera un moment où l'hystérie sera démembrée, et il n'est pas impossible de prévoir dès maintenant certaines subdivisions qui s'établiront plus tard.” Janet, “Quelques définitions récentes de l’hystérie,” 1893, 18. Charcot, however, adamantly proscribed such. Hysteria was “indivisible,” he said, in one of the lessons when Babinski was his chief assistant. “Si je n'admets pas que l'hystérie puisse être démembrée...j'admets cependant naturellement dans l'hystérie, espèce une et indivisible, des variétés, des formes; cela est élémentaire.” Charcot, *Leçons du mardi à la Salpêtrière*, 1:50.

¹³¹ Babinski, “Démembrement de l’hystérie traditionnelle,” 3.

his own susceptibility to self-deception and was known in later years for being extremely, some said pathologically, riddled with self-doubt.¹³² Perhaps that attitude developed in response to his experience at Charcot's clinic and his sensational experiments with magnets.¹³³ Regardless, his reflex tests represented for him one of the rare reliable opportunities to root out “subjective” and suggestive, pithiatistic symptoms from the “objective,” organic, and involuntary kinds.¹³⁴ It gave him something to hold onto, a compass to compensate for his own eroding trust in his patients and himself. A different problem arose, however, when Babinski could not rely on any reflex to evaluate a patient’s state of mind, particularly, a “mental trouble” that did not arise purely from within the mind but, he was convinced, had to do with a disease of the

¹³² The “diagnosis” that one of his former assistants suggested was “*la maladie du doute*,” which literally translated to the “illness of doubt,” although it would probably more accurately be compared to a form of compulsive behavior often directed to excessive self-examination and sometimes debilitating self-criticism. Philippon and Poirier, *Joseph Babinski*, 25–26; Albert Charpentier, *Un grand médecin, Joseph Babinski (1857-1932)* (Paris: Typographie François Bernouard, 1934).

¹³³ In 1912, Babinski visited the famous clairvoyant Eusapia Palladino and when he “discovered” her art of deception, he “flew into a towering rage,” according to his assistant Albert Charpentier. This is surprising given that others such as the physiologist and “psychical researcher” Charles Richet (1850-1935) had already exposed some of Palladino’s tricks in 1907. Philippon and Poirier, *Joseph Babinski*, 36; M. Brady Brower, *Unruly Spirits: The Science of Psychic Phenomena in Modern France* (Urbana: University of Illinois Press, 2010), 62–73; Sofie Lachapelle, *Investigating the Supernatural: From Spiritism and Occultism to Psychical Research and Metapsychics in France, 1853-1931* (Baltimore: Johns Hopkins University Press, 2011), 75–82; Bacopoulos-Viau, “Automatism, Surrealism and the Making of French Psychopathology,” 266.

¹³⁴ “Les symptômes peuvent être divisés en deux catégories, suivant qu'ils sont subjectifs ou objectifs.” Babinski, “Introduction à la semiologie des maladies du système nerveux. Des symptômes objectifs que la volonté est en capable de reproduire de leur importance en médecine légale,” 1125; Jean-Francois Allilaire et al., “Babinski et l’hystérie. Discussion,” *Bulletin de l'Académie nationale de médecine* 191, no. 7 (2007): 1137.

brain.¹³⁵ The question was, how to prove such a subjective symptom of the mind was, in fact, a rather objective disease of the brain.

Was It Real?

The act of naming a new disease, a new symptom, or a new syndrome was especially integral to the practice and identity of neurology. In many respects it remains so.¹³⁶ Although Babinski never occupied an official position in France as a “neurologist,” his reputation was long cemented in Paris and beyond as a doctor of internal medicine who preferred treating and studying patients with disorders of the brain and nerves. After all, what really made a neurologist anyway? Babinski belonged to the *Société de Neurologie*. In fact, he helped found it in 1899.¹³⁷ Still, he was never certified formally as a neurologist. There was, as of yet, no certification of the sort in France, not until the 1920s.¹³⁸ Upon closer inspection, then, it is not exactly clear what “made” Babinski, or any French physician for that matter, a *neurologist*.¹³⁹ Not unlike some of the disorders he diagnosed, the borders were so far unresolved and open-ended.

Still, names mattered. The *act* of naming was in itself an exercise in self-fashioning. French doctors, like Babinski, who aspired to be neurologists at the turn of

¹³⁵ Babinski, “Introduction à la semiologie des maladies du système nerveux. Des symptômes objectifs que la volonté est en capable de reproduire de leur importance en médecine légale,” 1125.

¹³⁶ See Chapter 5 of this dissertation. Lipowski and Kiriakos, “Borderlands between Neurology and Psychiatry,” 141.

¹³⁷ Philippon and Poirier, *Joseph Babinski*, 10.

¹³⁸ Weisz, “Regulating Specialties in France during the First Half of the Twentieth Century,” 458–459.

¹³⁹ Weisz points out that French doctors, until the 1920s, could define themselves practically as they pleased, so long as their peers informally affirmed them. *Ibid.*, 458.

the century, had no formal recourse to declare and defend their identity. The act of diagnosis was, therefore, an act of marking their territory.¹⁴⁰ That was why Babinski's dismemberment of hysteria was especially radical. He proposed, in effect, a new "map" to re-chart the old tracts of hysteria, not just to "clear the terrain" but to re-draw the lines of demarcation separating the "subjective" from the "objective," and thereby reclaiming newly-exposed patches at the border.¹⁴¹ One of those patches was the indifference to illness.

On June 11, 1914, Babinski presented to the *Société de Neurologie* two case reports with which he would herald the creation of two new kinds of brain disease, one called "anosognosia" and the other "anosodiaphoria." He gave a very brief account of each of the two patients, neither of whom he confessed to having examined as thoroughly as he would have liked, though apparently enough for him to declare that neither were of "perfectly sound" mind.¹⁴² In any case, without elaborating on his methods of mental evaluation, he announced the most "remarkable" feature of their condition: Despite being partially paralyzed, neither was willing or able to admit it.

The first patient avoided any question in connection with her hemiplegia and never spoke of it or complained about it. The second patient, when asked to try to lift her left arm, simply replied, "There, it's done," even though the arm never budged.

¹⁴⁰ Porter, "The Body and the Mind, the Doctor and the Patient."

¹⁴¹ Allilaire et al. use a provocative image of Babinski's "re-districting" [*cantanant*] hysteria. Allilaire et al., "Babinski et l'hystérie," 1336.

¹⁴² "Les fonctions psychiques, que je n'ai pas eu d'ailleurs le loisir d'étudier avec détails comme je l'aurais voulu, n'étaient peut-être pas dans un état d'intégrité parfaite." Babinski, "Contribution à l'étude des troubles mentaux dans l'hémiplégie organique (anosognosie)," 845.

Afterwards, she even teased the doctor. In the past, she said, he had always managed to heal her, but now, she claimed, “his science was powerless.”¹⁴³ Powerless to do what? If she did not realize she was paralyzed, then which “trouble” [*malaise*] did she mock her doctor for being powerless to heal? Babinski did not know how to explain it, except that perhaps each of the patients had some “vague notion” about their illness after all.¹⁴⁴

The first patient, he noted obscurely, sometimes complained of pain in her left shoulder, that is, on the hemiplegic side.¹⁴⁵ And the second patient, when she overheard some doctors discussing the use of “electrotherapy” on her hemiplegic limbs, responded, “Now why do you want to electrify [*électriser*] me? I am not, in any case, paralyzed.”¹⁴⁶ Perhaps, after all, each patient knew about her paralysis, faintly, but enough to know what to ignore and deny.

Was it real? The question constantly gnawed at Babinski throughout his career. He knew, in large part, he could never know for sure. He cautiously entertained the possibility that the patients merely “played” him, feigning their ignorance and indifference to elicit his curiosity and attention—“by coquetry, vanity, or dissimulation”¹⁴⁷—not unlike the hysterics he observed.¹⁴⁸ What remains puzzling,

¹⁴³ Ibid., 846.

¹⁴⁴ Ibid., 845.

¹⁴⁵ “[L]a malade...se plaignait parfois de douleurs à l’épaule gauche.” Ibid.

¹⁴⁶ Ibid., 846.

¹⁴⁷ Ibid.

¹⁴⁸ Babinski, “Introduction à la semiologie des maladies du système nerveux. Des symptômes objectifs que la volonté est en capable de reproduire de leur importance en médecine légale,” 1126; Babinski, “Démembrement de l’hystérie traditionnelle,” 4.

however, is that Babinski never mentioned the diagnosis of hysteria, or pithiatism, in relation to anosognosia and anosodiaphoria. Was that because he did not notice the similarity? Was it because he did not *want* to see it? After all, to admit any comparison, he would have also likely felt the need to defend their difference, that is, to prove that anosognosia was *not* simulated, and this was something he could not do, not with any certainty. True, he might have been able to verify the cerebral cause of the hemiplegia by way of reflex testing, and with it, to confirm that the paralysis was involuntary—thus, according to his criteria, *real*. But what about the *unawareness* of the hemiplegia? Could he ever confirm that it, too, was real? Did he have any way to rule out the possibility of simulation and demonstrate that the indifference was involuntary and “cerebral”? If not, did that mean anosognosia and anosodiaphoria might be related to hysteria after all?

∞

Many historians of medicine maintain that thanks to Babinski’s efforts the hysteria diagnosis “dissolved into nothing.”¹⁴⁹ Its dismembered limbs or former symptoms, they argue, “vanished into a hundred places,” becoming new disorders on their own and leaving no trace of their past.¹⁵⁰ Such a narrative, however, runs the risk of blankly accepting Babinski’s own account as historical truth. He was the one, in the first place, who tried to convince posterity that the old diagnosis had indeed dissolved

¹⁴⁹ Ackerknecht, *Kurze Geschichte der Psychiatrie*, 78.

¹⁵⁰ Mark S. Micale, “On the ‘Disappearance’ of Hysteria: A Study in the Clinical Deconstruction of a Diagnosis,” *Isis* 84, no. 3 (1993): 526.

into nothing. The question is, should we be convinced of his account? I do not think so.

Hysteria did not disappear, dissolve, or vanish into an unnameable nothing. Its traces were left behind in numerous places, not only among the so-called psychological or psychiatric diagnoses¹⁵¹ but also among nerve- and brain-related diseases, including anosognosia and anosodiaphoria. Viewed this way, such diseases do not appear as new as Babinski intended. Instead, they resembled the trace-symptom of hysteria which Janet called “*indifférence*.” But were Babinski to have compared either anosognosia or anosodiaphoria directly to hysteria, he would have implied that he denied their reality as genuine brain diseases. According to his definition, either they were real and unfeigned, that is, involuntary, or they belonged to the mind and contaminated by subjectivity. There was nothing in between. Such opposition and exclusion of any middle ground, however, was precisely what a number of physicians after Babinski rejected. It is to their ideas that the next chapter turns.

¹⁵¹ The philosopher Ian Hacking traces some features of this transformation in two of his books, *Rewriting the Soul* and *Mad Travelers*. He focuses his research on the historical trajectory of two hysterical symptoms, “multiple personality” and “fugue state,” both of which, in the aftermath of the putative “disappearance” of hysteria, had to stand alone as psychiatric diagnoses on their own. Hacking, *Rewriting the Soul: Multiple Personality and the Sciences of Memory*; Hacking, *Mad Travelers*, 1998.

Chapter 3

The Paradox of Health: Wartime Lessons from the Brain-Injured

“Perception hides itself from itself.”¹

At first, all was dark. Then, out of nowhere, heads slowly formed, floating mid-air, just two feet away. They were small and strange, plastic-looking and colored red, black, and white. In their faces, two dark holes filled the empty sockets where their eyes would have been. Then, as suddenly as they appeared, all faded back into the darkness.

In 1916, a thirty-eight year old soldier “David D.” was fighting for the Austrian-Hungarian army during World War I when a gas-bomb exploded near him, blinding him in both eyes. He was later taken to a clinic in Vienna, where he described to his doctor the vision above of tiny floating plastic heads with “empty, dark eye-sockets.”² According to the doctor, the psychiatrist Paul Schilder (1886-1940), “the patient perceived his blindness in the hallucinated heads.”³ Otherwise, though, he did not realize he could no longer see. The only time David D. “did not forget his blindness,” Schilder wrote, was when he “saw” the eye-less heads.⁴

In the same paper, Schilder described a similar case of another soldier-turned-patient from the war. He was a young officer who had been severely wounded in his

¹ Maurice Merleau-Ponty, *Phénoménologie de la perception*, Bibliothèque des idées (Paris: Gallimard, 1945), 85; Merleau-Ponty, *Phenomenology of Perception*, 2002, 67.

² “leere dunkle Augenhöhlen.” Paul Schilder, “Projektion eigener Körperdefekte in Trugwahrnehmungen,” *Neurologisches Centralblatt* 38 (1919): 300.

³ “Hier nimmt also der Kranke seine Blindheit an halluzinierten Köpfen wahr.” Ibid.

⁴ “Pat. vergißt seine Blindheit nicht, wenn er die augenlosen Köpfe sieht.” Ibid.

right arm, rendering it “completely useless.”⁵ During the first months following his injury, his wound became infected, causing a dangerously high fever of 41.8°C (107.2°F). For several nights, he lay awake delirious and screaming. Everything seemed to portend some terrible meaning, putting him in constant fear. As the feverish delirium subsided, Schilder noted the “strangest” part of all: Any time someone entered the patient’s room, suddenly that visitor’s left arm appeared to him as crippled and misshapen, or completely missing. And if the person happened to turn around with his back to the patient, then the illusion of the crippled arm switched to the opposite side. According to Schilder, the patient lacked “complete illness-insight” [*keine volle Krankheitseinsicht*]. The “delusions” [*Trugwahrnehmungen*] of seeing his own “body defect” [*Körperdefekt*] in other people was “nothing other than the expression of a wish to be freed of his crippling condition.”⁶ Neither the young officer nor “David D.” could identify with their injury enough to recognize it as their own. It was not that they completely lacked insight into their illness, but with the residual awareness they experienced, they “projected” it onto others, real and imagined.

A year earlier, in the summer of 1915, Johann Schneider, a soldier in the Imperial German Army, was marching through the deep mud when a mine exploded nearby. Bits of metal shrapnel flew in every direction, some of which pierced his skull and cut deep into his brain. Four days later, he woke up in a hospital with two open wounds in the back of his head. After several weeks in bed, he underwent an operation to remove the

⁵ “völlig gebrauchsunfähig.” Ibid.

⁶ “Hierin sehe ich einen Hinweis darauf, daß es sich um nichts anderes handelt, als um den Ausdruck des Wunsches unseres Kranken, von dem verstümmelnden Leiden befreit zu sein.” Ibid., 301.

metal shards still lodged in his brain tissue. The surgery was deemed a success, and on February 16, 1916, he was transferred to a clinic in Frankfurt for rehabilitation.⁷

At the clinic, Schneider seemed to recover quickly. But then one day, his doctor, the neurologist Kurt Goldstein (1878-1965), discovered something unusual. Apparently, Schneider could no longer read, not by the usual method at least. Instead of reading words by recognizing their overall visual shape, or “Gestalt,” he had to trace with his head ever so slightly every line and curve of each letter in a single word until he “kinaesthetically” recognized them—not so much with his eyes as with the movement of his body. In effect, Schneider was “word-blind” [*wortblind*] due to his brain injury, but he had learned, unwittingly, how to “read” in a novel way.⁸ According to Goldstein, the patient had no awareness of his disability and remained in disbelief when it was explained to him. Apparently, he had managed to “hide” it even from himself.⁹

In the first decades of the twentieth century, the “Anton-Babinski syndrome” emerged as a new disease entity, distinct from confusion and hysteria. Initially, most doctors believed that it involved a specific loss of awareness, confined to a particular aspect of one’s condition and caused by local tissue damage in the brain. In both

⁷ Kurt Goldstein and Adhémar Gelb, “Psychologische Analysen hirnpathologischer Fälle auf Grund von Untersuchungen Hirnverletzter,” *Zeitschrift für die gesamte Neurologie und Psychiatrie* 41 (1918): 9–11.

⁸ *Ibid.*, 27.

⁹ Kurt Goldstein and Adhémar Gelb, “Analysis of a Case of Figural Blindness,” in *A Source Book of Gestalt Psychology*, ed. Willis Davis Ellis (1918; London: Paul, Trench, Trubner, 1938), 317–319; Harrington, *Reenchanting Science: Holism in German Culture from Wilhelm II to Hitler*, chap. 5, 147–150ff.; Georg Goldenberg, “Goldstein and Gelb’s Case Schn.: A Classic Case in Neuropsychology?,” in *Classic Cases in Neuropsychology*, ed. Christopher Code, vol. 2, 2 vols. (Hove, East Sussex: Psychology Press, 2001), 281–99; J. J. Marotta and M. Behrmann, “Patient Schn: Has Goldstein and Gelb’s Case Withstood the Test of Time?,” *Neuropsychologia* 42, no. 5 (2004): 633–38.

respects, in its behavioral manifestation and anatomical origin, the disorder emerged because of its specificity. However, some doctors began to wonder whether perhaps it was *too* specific.

Many patients seemed as if they did not *want* to acknowledge their illness. They managed to avoid it only too well. It was as if they already “knew” what to ignore. If so, if they were aware of their illness on some level after all, then something more was at stake than “blind” lesions alone. Something more personal and motivated lurked behind the *denial*. Somehow on some level, patients knew what was wrong, but they allowed themselves to ignore it, effectively to “hide” it, even from themselves.

This chapter highlights a significant departure in the history of the unawareness of illness. It focuses on the early careers of two physicians, Paul Schilder and Kurt Goldstein, who formed part of a new generation of neurologists and psychiatrists known for their commitment to “holistic medicine.”¹⁰ Although a notoriously nebulous term that suggested only a loose affiliation of ideas, some of its self-professed adherents’ aims were to overcome a variety of oppositions and divisions between, for example, organic and psychogenic categories of disease, the study of individual behavior and their environments, and medical specialties in charge of mental illness and brain disease.¹¹ Schilder and Goldstein each devoted their professional and intellectual energies to

¹⁰ Harrington, “A Feeling for the Whole”; Ash, *Gestalt Psychology in German Culture*; Harrington, *Reenchanting Science: Holism in German Culture from Wilhelm II to Hitler*; Lawrence and Weisz, *Greater than the Parts*; Charles E. Rosenberg, “Holism in Twentieth-Century Medicine: Always in Opposition,” in *Our Present Complaint: American Medicine, Then and Now* (Baltimore: Johns Hopkins University Press, 2007), 139–65.

¹¹ Harrington, *Reenchanting Science: Holism in German Culture from Wilhelm II to Hitler*, xvii.

surmount such divisions and in doing so to redefine the basic understanding of illness and health. Part of the argument of this chapter, then, is to show how their unique interpretations of the unawareness of illness amplified and helped consolidate their “holistic” reforms.

The discussion opens with Schilder, who trained under Gabriel Anton before the war. Though loyal to his former mentor and his study of neuropathology, Schilder incorporated other ideas from psychoanalysis and phenomenology, which he used to explain the unawareness of illness in a unique turn-of-phrase, “organic repression.”¹² The chapter then shifts focus to Kurt Goldstein, a former student of Carl Wernicke, who set up a rehabilitation clinic in Frankfurt for soldiers with head injuries from the war. Goldstein also retained his training in neuroanatomy on the principles of cerebral localization, but he integrated it with insights from Gestalt psychology and existential philosophy. Above all, he emphasized the importance of every individual patient’s interactions with his environment as a way of coping. In that light, he proposed the most radical redefinition of anosognosia. It was not, he argued, a symptom of disease but the expression of a new form of health.

Finally, the chapter concludes with a look at the contributions of Schilder and Goldstein to the philosopher Maurice Merleau-Ponty (1908-1961). During the Second World War, Merleau-Ponty drew from case studies by the two doctors for his book,

¹² “Verdrängungsmechanismus mit organischen Unterlagen.” Paul Schilder, *Das Körperschema: Ein Beitrag zur Lehre vom Bewusstsein des eigenen Körpers* (Berlin: Springer, 1923), 21, 79. “organisch fundierten Verdrängungsfaktor.” Heinz Hartmann and Paul Schilder, “Zur Psychologie Schädelverletzter,” *Archiv für Psychiatrie und Nervenkrankheiten* 75 (December 1, 1925): 296. “Verdrängung im Bereiche des Organischen.” Paul Schilder and Erwin Stengel, “Schmerzasymbolie,” *Zeitschrift für die gesamte Neurologie und Psychiatrie* 113 (1928): 154.

Phenomenology of Perception. In particular, he consulted their views on anosognosia as a template for describing the nature of all perceptual experience. I argue that this creative point of insertion of medical thought into phenomenological philosophy testified to the increasingly eclectic, one might say “holistic,” domains of the sciences and medicine of the mind by the middle of the twentieth century.

“The Double Way”

Schilder’s body of work resists any easy definition. He studied neuroanatomy with Paul Flechsig and learned psychoanalysis from Sigmund Freud.¹³ He wrote on the phenomenology of Edmund Husserl and described a new brain disease, *Encephalitis periaxialis diffusa*, while working in the clinic of Gabriel Anton.¹⁴ He was both a physician and metaphysician, both anatomist and an analyst. Perhaps that was part of the reason he was unusual, since he drew from such a wide array of methods and theories for his research on the disorders of consciousness. But that was what ultimately bound together Schilder’s ranging interests and restless energy: the problem of consciousness. Essentially, he wanted to understand the nature of the self through

¹³ Paul Schilder, “Vita and Bibliography of Paul Schilder,” *Journal of Criminal Psychopathology* 2 (1940): 221–34; Dieter Langer, “Paul Ferdinand Schilder: Leben und Werk” (Johannes-Gutenberg-Universität, 1979); Donald A Shaskan and William L Roller, eds., *Paul Schilder, Mind Explorer* (New York, N.Y: Human Sciences Press, 1985); Hannah Decker, “Psychoanalysis in Central Europe,” in *History of Psychiatry and Medical Psychology*, ed. Edwin R. Wallace and John Gach, 2008, 605.

¹⁴ It was later re-named “Schilder’s disease.” Paul Schilder, “Zur Kenntnis der sogenannten diffusen Sklerose (über Encephalitis periaxialis diffusa),” *Zeitschrift für die gesamte Neurologie und Psychiatrie* 10 (1912): 1–60.

the coordinated roles of the body and the brain as they helped shape the awareness of one's personal identity, or "personality-consciousness" [*Persönlichkeitsbewusstsein*].¹⁵

Schilder earned his degree in medicine from the University of Vienna in 1909. As a student, he attended the lectures of Sigmund Freud and worked in the laboratory of the physiologist Sigmund Exner.¹⁶ Together, they seemed to have made a sufficient impression on him that he chose to concentrate his clinical research on disorders of the mind, which in the German-speaking world still fell under the "double discipline" [*Doppelfach*] of psychiatry and neurology.¹⁷ After medical school, he moved to Halle an der Salle, Germany, to assist Anton, who succeeded Wernicke in the wake of his recent death.¹⁸ There, Schilder worked on his medical thesis, which he enthusiastically dedicated to Anton and published in 1914 under the title, *Self-Consciousness and Personality-Consciousness: A Psychopathological Study*.¹⁹ With this first book, Schilder showed the earliest glimpse of his attempt to build on the psychopathology of "body-

¹⁵ Paul Schilder, *Selbstbewusstsein und Persönlichkeitsbewusstsein: Eine psychopathologische Studie*, Monographien aus dem Gesamtgebiete der Neurologie und Psychiatrie, Heft 9 (Berlin: J. Springer, 1914).

¹⁶ Gröger, "Zur Entwicklung der Psychiatrie in der Wiener Medizinischen Schule," 39; Schilder, "Vita and Bibliography of Paul Schilder"; Heinz Hartmann, "The Psychiatric Work of Paul Schilder," *Psychoanalytic Review* 31 (1944): 287–98.

¹⁷ Gabriel, "Zur Beziehung zwischen Psychiatrie und Psychotherapie in Wien im 20. Jahrhundert - Eine psychiatriegeschichtliche Einführung zu ihrer Entwicklung um die Jahrhundertwende," 15.

¹⁸ Pfeifer, "Gabriel Anton," 187; Hartmann, "The Psychiatric Work of Paul Schilder," 294.

¹⁹ Schilder, *Selbstbewusstsein und Persönlichkeitsbewusstsein: Eine psychopathologische Studie*.

awareness” [*Körperbewusstsein*] and to formulate out of it a new theory of the embodied self.²⁰

When the war began, he enlisted in the Austro-Hungarian army as a medic. Incredibly, while confined to the trenches, he studied for his doctorate in philosophy *in absentia*, as mortar shells exploded continuously overhead.²¹ He was fortunate enough to survive the harrowing ordeal and returned to Vienna in 1918, whereon he immediately assumed the position of assistant to the psychiatrist Julius Wagner von Jauregg (1857-1940), Theodor Meynert’s successor at the university clinic.²² That year, Schilder also published his dissertation in philosophy under the title, *Delusion and Knowledge: A Psychopathological Study*.²³ He quickly earned the approval of Wagner-Jauregg and was promoted to lecturer in 1921, allowing him to offer a range of courses, such as, “Pathophysiology and the Ground-Problems of Philosophy and Psychology,” “Neurological Research Methods,” and “Psychoanalytic Demonstrations.”²⁴

²⁰ Schilder, *Das Körperschema: Ein Beitrag zur Lehre vom Bewusstsein des eigenen Körpers*; Heinz Hartmann and Paul Schilder, “Körperinneres und Körperschema,” *Zeitschrift für die gesamte Neurologie und Psychiatrie* 109 (1927): 666–75; Schilder, *The Image and Appearance of the Human Body*.

²¹ Schilder, “Vita and Bibliography of Paul Schilder”; Langer, “Paul Ferdinand Schilder: Leben und Werk,” 54.

²² See Chapter 1.

²³ Paul Schilder, *Wahn und Erkenntnis: Eine psychopathologische Studie* (Berlin: J. Springer, 1918).

²⁴ Langer, “Paul Ferdinand Schilder: Leben und Werk,” 67n1; Ellenberger, *The Discovery of the Unconscious*, 846.

Schilder quickly joined the Vienna Psychoanalytic Society within a year of returning to Vienna, upon a personal invitation from Freud.²⁵ When in 1925 he was promoted to professor of psychiatry at the university, he received a letter of congratulations from Freud for becoming the first ever psychoanalyst to hold a full academic chair.²⁶ By virtue of this “dual affiliation,” therefore, Schilder represented a rare and important bridge-figure in postwar Austrian medicine, navigating between the two worlds of clinical psychiatry and psychoanalysis, despite what many perceived to be their increasing “polarization.”²⁷

Schilder believed he did not have to choose between one or the other. Although skilled in microscopic preparations of morbid brain tissue, he was also conversant in the nuanced “drive-psychology” of the psychoanalysts. He may have assisted Wagner-Jauregg at the university clinic administering malarial injections in patients with syphilis,²⁸ but he also devoted a number of hours every week at the newly built

²⁵ Paul Schilder and Herman Weidner, “Zur Kenntnis symbolähnlicher Bildungen im Rahmen der Schizophrenie,” *Zeitschrift für die gesamte Neurologie und Psychiatrie* 26 (1914): 201–44; Decker, “Psychoanalysis in Central Europe,” 605.

²⁶ Decker, “Psychoanalysis in Central Europe,” 605.

²⁷ “Vor allem in den Jahrzehnt nach dem Ersten Weltkrieg gab es einerseits Polarisierung zwischen Vertreten der klinischen Psychiatrie und der Psychoanalyse...zu anderen aber auch zumindest Doppelzugehörigkeiten von einzelnen Persönlichkeiten sowohl zum Stab der Psychiatrisch-Neurologischen Universitätsklinik unter Wagner-Jauregg als auch zu psychoanalytischen Institutionen. Die bedeutendste Persönlichkeit darunter ist Paul Schilder.” Gabriel, “Zur Beziehung zwischen Psychiatrie und Psychotherapie in Wien im 20. Jahrhundert - Eine psychiatriegeschichtliche Einführung zu ihrer Entwicklung um die Jahrhundertwende,” 24; Decker, “Psychoanalysis in Central Europe,” 593.

²⁸ Jean-Noël Missa, *Naissance de la psychiatrie biologique : Histoire des traitements des maladies mentales au XXe siècle* (Presses Universitaires de France, 2006), 38–57; Braslow, *Mental Ills and Bodily Cures: Psychiatric Treatment in the First Half of the Twentieth Century*, chap. 4; E.M. Brown, “Why Wagner-Jauregg Won the Nobel Prize for Discovering Malaria Therapy for General Paresis of the Insane,” *History of Psychiatry* 11, no. 44 (January 2000): 371–82; Julius Wagner von Jauregg, “Über die Einwirkung

psychoanalytic outpatient clinic.²⁹ For Schilder, it was a matter of following what he called “the principle of the double way.”³⁰ He did not want to have to pick sides, so he tried to straddle both.³¹ On an institutional and professional level, this was noteworthy enough, although his career would later suffer severe consequences for it. (In 1927, Wagner-Jauregg, newly a Nobel laureate, passed Schilder over for a promotion.³² And in 1935, Freud cut him out from his inner circle.³³) Instead of treading a “double way,” Schilder’s path resembled more of a “middle way.”³⁴ Indeed, this probably better

der Malaria auf die progressive Paralyse,” *Psychiatrischneurologische Wochenschrift* 20, no. 132 (1919 1918): 251; Shaskan and Roller, *Paul Schilder, Mind Explorer*, 26.

²⁹ Gröger, “Zur Entwicklung der Psychiatrie in der Wiener Medizinischen Schule,” 37–39; Decker, “Psychoanalysis in Central Europe,” 593; Helmut Gröger, “Zur Gründungsgeschichte des Psychoanalytischen Ambulatoriums,” *Bulletin - Sigmund Freud House* 18/1B (1993): 3–23; Eduard Hitschmann, “Zehn Jahre Wiener Psychoanalytisches Ambulatorium (1922-1932): Zur Geschichte des Ambulatoriums,” *Internationale Zeitschrift für Psychoanalyse* 18 (1932): 265–71.

³⁰ Paul Schilder, *Entwurf einer Psychiatrie auf psychoanalytischer Grundlage* (Leipzig: Internationaler Psychoanalytischer Verlag, 1925), 73; Isidore Ziferstein, “Paul Ferdinand Schilder: Psychoanalysis and Psychiatry,” in *Psychoanalytic Pioneers*, ed. Franz Alexander (New York: Basic Books, 1966), 459; Langer, “Paul Ferdinand Schilder: Leben und Werk,” 55.

³¹ Decker, “Psychoanalysis in Central Europe,” 593; Gröger, “Zur Entwicklung der Psychiatrie in der Wiener Medizinischen Schule,” 39; Erwin Stengel, “Paul Schilder,” *Internationale Zeitschrift für Psychoanalyse* 26 (1941): 377.

³² Their strained relationship may have been in some part due to the souring between Wagner-Jauregg and Freud after Freud publicly testified against Wagner-Jauregg’s clinical judgment in avoiding the use of psychotherapy during the war. Langer, “Paul Ferdinand Schilder: Leben und Werk,” 85; K.R. Eissler, *Freud as an Expert Witness: The Discussion of War Neuroses Between Freud and Wagner-Jauregg* (New York: International Universities Press, 1986); K.R. Eissler, “Julius Wagner-Jaureggs Gutachten über Sigmund Freud und seine Studien zur Psychoanalyse,” *Wiener klinische Wochenschrift* 70 (1958): 401–7; Ellenberger, *The Discovery of the Unconscious*, 470.

³³ Hale, *The Rise and Crisis of Psychoanalysis in the United States*, 121–123.

³⁴ This characterization fits with the theory of historian Paul Lerner, who argues that many of the German physicians during and after the war did not subscribe completely to Freud’s ideas but “experimented” with them from a “middle ground” perspective, thereby making some of the “greatest strides in adapting psychoanalysis.” Paul Frederick Lerner, *Hysterical Men: War, Psychiatry, and the Politics of Trauma in Germany, 1890-1930*, Cornell Studies in the History of Psychiatry (Ithaca: Cornell University Press, 2003), 165.

explains why his thought remains difficult to characterize, although it continues to be productive to analyze. For, it was on the intellectual and theoretical level that he made his most creative contributions.³⁵

“Organic Repression”

In 1923, Schilder published a small book called, “The Body-Schema: A Contribution to the Doctrine of Consciousness of One’s Own Body,” in which he sketched a new approach to the disorders of consciousness as all disorders of “body consciousness” [*Körperbewusstsein*]. He directly targeted Babinski, whose stark opposition of “organic” and “hysterical” symptoms he found no longer adequate or justified to describe either neuropathology or psychopathology.³⁶ Instead, Schilder stressed, even hysterical symptoms, such as the lack of feeling on one side of the body (including even the lack of feeling of one side of the body), could be understood from the study of organic brain diseases.³⁷ Reciprocally, numerous forms of brain injuries—many more than most doctors cared to admit—could benefit from the application of

³⁵ Paul Schilder, *Brain and Personality: Studies in the Psychological Aspects of Cerebral Neuropathology and the Neuropsychiatric Aspects of the Motility of Schizophrenics* (New York: Nervous and Mental Disease Pub. Co, 1931); Schilder, *The Image and Appearance of the Human Body*; Paul Schilder, *Mind: Perception and Thought in Their Constructive Aspects* (New York: Columbia University Press, 1942); Hartmann, “The Psychiatric Work of Paul Schilder,” 295.

³⁶ In fact, Schilder earlier critiqued Babinski’s division of “organic” versus “psychic” symptoms, arguing instead that each represented only different points of “view” and ultimately “complemented” the other. Schilder, *Selbstbewusstsein und Persönlichkeitsbewusstsein: Eine psychopathologische Studie*, 148; Schilder, *Das Körperschema: Ein Beitrag zur Lehre vom Bewusstsein des eigenen Körpers*, 23.

³⁷ “Hier liegt ein neuerlicher Beweis dafür vor, daß die Hysterie und die hysterischen Erscheinungen an Gebilden ansetzen, deren Struktur nur durch das Studium der organischen Hirnerkrankung verstanden werden kann.” Schilder, *Das Körperschema: Ein Beitrag zur Lehre vom Bewusstsein des eigenen Körpers*, 23.

“phenomenological psychology”³⁸ and the “laws of emotions” [*affektiven Gesetzmäßigkeiten*] according to Freud.³⁹ Ultimately, for Schilder, both “organically-based” and “psychically-conditioned” disorders shared a “deep common ground.”⁴⁰

To illustrate, Schilder analyzed Anton’s diagnosis, or the “problem of imperception” [*Problem der Nichtwahrnehmung*] of illness.⁴¹ He maintained, contra Anton, that such a phenomenon could never be understood only in terms of organic lesions of the brain. Crucially, one also had to take into account the patient’s “mental attitude” [*Seelenverfassung*]. Specifically, Schilder declared, the patient’s apparent unawareness always concealed a latent “wish not to know” [*Nichtwissenwollen*], which manifested in the her consistent “turning-away” [*Hinwegsehen*], not only by physically averting her eyes and head from the affected side of her body but also by “turning” her thoughts and attention away from the reality of her predicament.

In addition to a “mechanism in the brain” [*Hirnmechanismus*], Schilder asserted, there must also be “a mechanism of repression” [*einen Verdrängungsmechanismus*]

³⁸ Schilder repeatedly tried to distinguish himself from Karl Jaspers, another trained psychiatrist-turned-philosopher who assimilated many of Husserl’s ideas for a “phenomenological psychology.” According to Schilder, however, Jaspers’ version was a “misinterpretation” because he tried to use phenomenology in a “purely descriptive” manner, neglecting to draw any “direct relation” to Husserl’s later works. Schilder, *Selbstbewusstsein und Persönlichkeitsbewusstsein: Eine psychopathologische Studie*, 3; Hartmann, “The Psychiatric Work of Paul Schilder,” 294; Spiegelberg, *Phenomenology in Psychology and Psychiatry*, 322; Berrios, “Phenomenology, Psychopathology and Jaspers”; German E. Berrios, “Phenomenology and Psychopathology: Was There Ever a Relationship?,” *Comprehensive Psychiatry* 34, no. 4 (August 1993): 213–20; Susan Lanzoni, “Bridging Phenomenology and the Clinic: Ludwig Binswanger’s ‘Science of Subjectivity’” (Doctoral Dissertation, Harvard University, 2001).

³⁹ Schilder, *Das Körperschema: Ein Beitrag zur Lehre vom Bewusstsein des eigenen Körpers*, 1, 23.

⁴⁰ “Zwischen den organisch fundierten und den psychische bedingten Störungen besteht eine tiefe Gemeinsamkeit.” *Ibid.*, 23.

⁴¹ *Ibid.*, 21.

albeit one based on organic foundations [*mit organischen Unterlagen*].⁴² Therefore, without taking into account both the focal brain lesions and the “psychic factor of repression,” the unawareness of illness remained incomprehensible. “In every case,” Schilder concluded, “one had to be clear about the principled uniformity between the focal organic [*herdbedingten*] and general psychic [*allgemein psychischen*] mechanism of repression.” Only then could anyone hope to “come closer to [understanding] such things” as the denial of illness.⁴³

A year later, Schilder wrote his first patient history of such a hybrid phenomenon. Working alongside the psychologist and philosopher Heinz Hartmann, they examined the case of a young woman, “Hermine H.,” who was taken to the Franz-Josef Hospital on July 20, 1924, after suffering a bad bicycle accident, in which she hit her head hard on the pavement. According to her husband, she momentarily lost consciousness, and by the time they reached the hospital, she was bleeding from her ears and nose.⁴⁴

As she slowly regained consciousness in the hospital, she “expressly denied” [*verneint ausdrücklich*] ever having fallen off her bicycle. When Schilder asked her why then she lay in a hospital bed with a bandage wrapped around her head, she protested that she was not in a hospital but “someone’s camp bed,” and the white wrap on her head was “because I washed my hair.”⁴⁵ Next, he reached for a mirror and held it up to

⁴² Ibid.

⁴³ Ibid., 22.

⁴⁴ Hartmann and Schilder, “Zur Psychologie Schädelverletzer,” 287–288.

her face, asking her what she saw and whether it was, in fact, a bandage on head, to which she replied matter-of-factly, "There, that is a towel." They asked, what is a towel that covers a wound called? "It is a only a white towel, a clean hand towel," adding, "under the bandage is a small hand towel." For Schilder and Hartmann, the word "bandage" signaled a confession, a minor one perhaps, but a confession all the same.

They believed that Hermine H. was semi-aware of her head injury after all.⁴⁶

Because she used the "expression 'bandage'...the patient has some form of awareness of her illness, which she is determined to protect from herself."⁴⁷ The *patient*, therefore, not just the location or severity of her head injury, was responsible for not recognizing, for refusing to recognize, her condition. She knew just enough to know that she did not want to know any more. Despite the "non-perceiving" [*Nichtwahrnehmen*] of her head wound, she retained the mental capacity to perceive it.⁴⁸

The problem was not exactly that she had lost the awareness or experience of her condition. It never entirely "disappeared from the mental realm."⁴⁹ Rather, her "tendency to deny the head wound" was the result of her "un-insightfully pushing

⁴⁵ Ibid., 287.

⁴⁶ They referred to it as a "twilight" [*leicht Dämmeriges*] form of awareness, alluding to the "twilight-state" [*psychische Dämmerstand*] described by Richard Krafft von Ebing. Ibid.; Krafft-Ebing, *Lehrbuch der Psychiatrie auf klinischer Grundlage für praktische Ärzte und Studierende*, 102.

⁴⁷ "Der Ausdruck Verband zeigt bereits an, daß die Patientin irgendein Bewußtsein der Krankheit hat, welches sie aber mit Entschiedenheit von sich abzuwehren trachtet." Hartmann and Schilder, "Zur Psychologie Schädelverletzer," 292.

⁴⁸ "ein Nichtwahrnehmen eines Defektes, welchen die Patienten ihrem sonstigen Auffassungsvermögen nach wahrnehmen müßten." Ibid., 293.

⁴⁹ "das Erlebnis nicht dem seelischen Bereiche entschwindet." Ibid., 296.

away" [*uneinsichtig hinausdrängt*] certain experiences from their true "relations-to-the-self" [*Ichbeziehungen*]. In other words, she "displaces experiences by way of the tendencies to repress" [*Verdrängungstendenzen*].⁵⁰ The key for Schilder, and for Hartmann, was to argue for the preserved unity and integrity of individual experience, of the conscious mind, and ultimately of the whole self. From Schilder's earliest writing, he attacked the "bundle theory" [*Bündelstheorie*] of Ernst Mach and admirers like Hugo von Hofmannsthal who gave up the idea of a unitary self greater than the sum of its sensory elements.⁵¹ For Schilder, therefore, his idea of an "organically-founded repression factor" [*organisch fundierten Verdrängungsfaktor*] was a way to ensure that the experience of the self, the *Icherlebnis*,⁵² was never permanently lost or lesioned, only "pushed back."⁵³

Part of the difficulty in understanding Schilder's concept of "focal organic repression" lies in the interpretation of "repression" itself. Although he clearly relied on Freud's teachings, he preferred to use the term repression in a broader sense than the strictly psychoanalytic.⁵⁴ Indeed, that was one of the main concepts he borrowed from

⁵⁰ Ibid., 292.

⁵¹ Schilder, "Über das Selbstbewusstsein und seine Störungen," 511, 514; Schilder, *Selbstbewusstsein und Persönlichkeitsbewusstsein: Eine psychopathologische Studie*, 5–6; Paul Schilder, *Medical Psychology*, trans. David Rapaport (Berlin: Julius Springer, 1924; New York: International Universities Press, 1953), 298.

⁵² This experience of the I or "ego" [*Icherlebnis*] was crucially not the same as the consciousness of the *self* [*Selbstbewusstsein*]. Whereas the latter might be interrupted, as the more explicit form of reflective awareness, the former was implicitly always in the background of every experience. Schilder, *Selbstbewusstsein und Persönlichkeitsbewusstsein: Eine psychopathologische Studie*, 6.

⁵³ Hartmann and Schilder, "Zur Psychologie Schädelverletzer," 296.

⁵⁴ Some historians and former colleagues speculate whether Schilder's self-described "unorthodox" interpretations of Freud's ideas may have jeopardized his standing with the more orthodox analysts in

the phenomenological philosophers like Edmund Husserl and Max Scheler, who both used the term “repression” with deliberately less-than-psychoanalytic meaning.⁵⁵

For Scheler and Husserl, writing after 1910, the experience of the “ego” or the self [*Icherlebnis*]⁵⁶ was ultimately “indivisible and whole” [*ganzen ungeteilten*].⁵⁷

According to their versions of “ego-phenomenology,” it was possible for experiences to recede to the fringe or background of one’s awareness, but not to vanish completely, or irretrievably, from the mind.⁵⁸ Scheler elaborated on what this kind of repression meant,

[T]he experiences are indeed there but are not seen, and...are simply placed out of sight by an instinctive drive. Is it not necessary that the experience already be inwardly perceived if it is to be “repressed”?⁵⁹

The problem with Freud, he continued, was his conflation of the difference between an experience and the *perception* of an experience.⁶⁰ That difference was critical because,

New York after he moved to the United States. Hale, *The Rise and Crisis of Psychoanalysis in the United States*, 121–123; Langer, “Paul Ferdinand Schilder: Leben und Werk,” 53; Hartmann, “The Psychiatric Work of Paul Schilder,” 296.

⁵⁵ Nicholas Smith, *Towards a Phenomenology of Repression: Husserlian Reply to the Freudian Challenge* (*Stockholm Studies in Philosophy*), Stockholm Studies in Philosophy (Stockholm: Stockholm Universitet, 2010); Victor Biceaga, *The Concept of Passivity in Husserl’s Phenomenology* (Dordrecht: Springer, 2010), 24.

⁵⁶ Shigeru Taguchi, *Das Problem des “Ur-Ich” bei Edmund Husserl: Die Frage nach der selbstverständlichen “Nähe” des Selbst* (Dordrecht: Springer, 2006), 55.

⁵⁷ Max Scheler, “Die Idole der Selbsterkenntnis,” in *Vom Umsturz der Werte*, vol. 2 (Der Neue Geist, 1915), 100.

⁵⁸ Dermot Moran, *Edmund Husserl: Founder of Phenomenology* (Cambridge: Polity, 2005), 164; Edmund Husserl, *Ideas Pertaining to a Pure Phenomenology and to a Phenomenological Philosophy* (1913; The Hague: M. Nijhoff, 1980), 234; Max Scheler, “The Idols of Self-Knowledge,” in *Selected Philosophical Essays*, Northwestern University Studies in Phenomenology & Existential Philosophy (1915; Evanston: Northwestern University Press, 1973), 69.

⁵⁹ Scheler, “The Idols of Self-Knowledge,” 84.

although an experience might be repressed, or “pushed back,” it could never be altogether pushed *out*. It always lingered in the recesses of consciousness, in the background of experience; even if not perceived in its direct “relation-to-the-self” [*Ichbeziehung*], it remained connected to the “totality” of one’s being, one’s “body-self” [*Leiblich*], argued Scheler.⁶¹ That was the ultimate, “vaguely articulated whole” and the background against which all experiences either rose or receded but always remained in one unified self.⁶²

The appeal to Schilder of this later version of phenomenology was unmistakable from his earliest writing, but it took on a novel form in his later book about the “body schema.”⁶³ The term “body schema” was coined by British neurologists Henry Head (1861-1940) and Gordon Holmes (1876-1965) in 1911.⁶⁴ They described it as a form of awareness “charged with relation,” that is, not made up of discrete chunks, say, the

⁶⁰ *Ibid.*, 84n.

⁶¹ *Ibid.*, 88–89, 37. So too with the “feeling of sickness” [*Krankheitsgefühl*], Scheler wrote: “What a distance separates what anyone experiences from what he experiences with such knowledge that he can say what it is he is experiencing!” *Ibid.*, 45.

⁶² Scheler, “The Idols of Self-Knowledge,” 68n; Husserl, *Ideas Pertaining to a Pure Phenomenology and to a Phenomenological Philosophy*, 61; Taylor Carman, “The Body in Husserl and Merleau-Ponty,” *Philosophical Topics* 27, no. 2 (1999): 213; Moran, *Edmund Husserl*, 212.

⁶³ Schilder also drew on Carl Wernicke’s and Arnold Pick’s concepts of the “somatopsyché” and “autotopagnosia,” respectively. Carl Wernicke, *Grundriss der Psychiatrie in klinischen Vorlesungen* (Leipzig: Thieme, 1894); Arnold Pick, “Zur Pathologie des Ich-Bewusstseins: Studien aus den allgemeinen Psychopathologie,” *Archiv für Psychiatrie und Nervenkrankheiten* 38 (1904): 22–33; Arnold Pick, *Studien Zur Hirnpathologie Und Psychologie* (S. Karger, 1908); Arnold Pick, “Zur Pathologie des Bewusstseins vom eigenen Körper: Ein Beitrag aus der Kriegsmedizin,” *Neurologisches Centralblatt* 34 (1915): 257–65; Arnold Pick, “Störung der Orientierung am eigenen Körper. Beitrag zur Lehre vom Bewusstsein des eigenen Körpers,” *Psychologische Forschung* 1, no. 1 (1922): 303–18.

⁶⁴ Henry Head and Gordon Holmes, “Sensory Disturbances from Cerebral Lesions,” *Brain: A Journal of Neurology* 34 (1911): 181–189; Klaus Poeck and Bernt Orgass, “The Concept of the Body Schema: A Critical Review and Some Experimental Results,” *Cortex; a Journal Devoted to the Study of the Nervous System and Behavior* 7, no. 3 (September 1971): 254–77.

awareness of one's arm or ankle, but of a unified, fluid, and relational awareness of one's body in space and in *motion* from one position to the next.⁶⁵ What attracted Schilder to the concept of the body schema was its ever-changing, constructive, and unifying properties.

By drawing on the later work of Scheler and Husserl as well, Schilder saw this vaguely enveloping experience of the body—*through* the body—as the originary background of all awareness, not the awareness of some discrete “thing,” but of some “vague...happening within the body”⁶⁶ or indirect “awareness of relation.”⁶⁷ Consequently for Schilder, the “repression” of any experience always involved the body schema, or the background awareness of the body's relationship to individual experience. That was the reason repression was always, on some level, “organic” and embodied, yet equally subjective and “psychological.”⁶⁸ Even if the “imperception” of illness meant the repressed experience of some part of the body, such as a paralyzed

⁶⁵ Head and Holmes stressed the distinction between visual and postural awareness of the body. In patients who lacked the postural awareness, or body schema, sometimes visual awareness remained. This meant that patients could locate their body limb in space if allowed to look for it first. Whereas the mental image of the body from moment to moment was left intact, they could not connect these moments in a coherent succession, or “schema” of mental images. In effect, they could not find their own body by “feeling” it from inside. They had to “look” for it as if outside themselves. Head and Holmes, “Sensory Disturbances from Cerebral Lesions,” 185–188; Heller-Roazen, *The Inner Touch*; Lasègue, “De l’anesthésie et de l’ataxie hystériques”; Pierre Bonnier, *Vertige* (Chartres: Imprimerie Durand, 1893); Pierre Bonnier, “L’aschématie,” *Revue Neurologique* 13 (1905): 605–9.

⁶⁶ Head and Holmes, “Sensory Disturbances from Cerebral Lesions,” 181.

⁶⁷ Schilder was also fond of citing the work of Würzburg philosopher Narziss Ach (1871-1946), who stressed this point of indirect, “image-less” awareness of relation, which he called “*Bewusstheit*.” Edwin Garrigues Boring, *A History of Experimental Psychology* (New York: Appleton-Century-Crofts, 1950), 406.

⁶⁸ Paul Schilder, “Health as a Psychic Experience,” *Arch Neurol Psychiatry* 37, no. 6 (June 1, 1937): 1322–37.

limb, that experience did not totally disappear like a lesion of awareness. Instead, it remained in the background of the body.

Although Schilder's use of "organic repression" reflected his unusual professional allegiances, it also testified to his extremely original, if sometimes overtly obscure, medical thought. With his concept of organic repression allied to that of the body schema, he tried to fuse together cerebral localization theory, phenomenological philosophy, and psychoanalytic psychiatry. Ultimately, he urged for a new way of understanding disorders of the mind as, at once, more biological and more personal.

A New Anna O.

Kurt Goldstein is typically remembered as a neuropsychologist in the United States from the 1940s-50s.⁶⁹ What is often forgotten is that he trained first as a psychiatrist. Part of the reason this fact is overlooked is the historical shift in the meaning of psychiatry since Goldstein studied it in the first decade of the twentieth century. In those years, clinical psychiatry in the German-speaking world was still wedded to neuropathology, even though the relationship was growing increasingly strained.⁷⁰ Indeed, Goldstein's clinical training began under Carl Wernicke, one of the

⁶⁹ A. R. Luria, "Kurt Goldstein and Neuropsychology," *Neuropsychologia* 4, no. 4 (1966): 311–13; H.L. Teuber, "Kurt Goldstein's Role in the Development of Neuropsychology," *Neuropsychologia* 4, no. 4 (December 1966): 299–310.

⁷⁰ The Berlin psychiatrist Karl Bonhoeffer noted that the high incidence of head injuries during the war helped further the cause of neurologists who wanted permanent independence from psychiatry, despite the fact that most of their patient populations overlapped. Bonhoeffer, "Psychiatrie und Neurologie," 97.

foremost leaders of the old “double discipline” of psychiatry and neurology.⁷¹ Shortly after graduating from the University of Breslau in 1903, he assisted at the psychiatric clinic of the University of Königsberg until 1914 when he moved to Frankfurt and founded the “Institute for Research into the Consequences of Brain Injuries” in 1916.⁷² Within only a few weeks of its official opening, one of the first patients to arrive was Johann Schneider, who would become Goldstein’s most important case of his entire career—indeed one of the most important cases in the history of neurology.⁷³

As described at the beginning of the chapter, Johann Schneider was a soldier of the Imperial Germany Army, who in the summer of 1915 was injured in the back of his head by pieces of shrapnel from a mine explosion.⁷⁴ On December 29, 1915, Schneider

⁷¹ Part of the reason it is important to stress Goldstein’s initial role as a clinical psychiatrist is that he used it himself to account for his later ability, effectively as a “neurologist,” to detect and analyze some of the more minute psychological disturbances of patients with brain injuries from the war. Goldstein wrote that he learned this type of analysis from Wernicke, who taught him the importance of “psychological interpretation of the symptoms of nervous disease.” In other words, he paid careful attention to each patient’s behavior and the meaning of their words in addition to knowing how to dissect their brain. Kurt Goldstein, “Kurt Goldstein,” in *A History of Psychology in Autobiography*, ed. Edwin Garrigues Boring, vol. 5, The Century Psychology Series (Washington, D.C.: American Psychological Association, 1967), 148.

⁷² Harrington, *Reenchanted Science: Holism in German Culture from Wilhelm II to Hitler*, 145; Uta Noppeney, “Kurt Goldstein - A Philosophical Scientist,” *Journal of the History of the Neurosciences* 10 (2001): 67–78; Frank Stahnisch and Thomas Hoffman, “Kurt Goldstein and the Neurology of Movement during the Interwar Years: Physiological Experimentation, Clinical Psychology and Early Rehabilitation,” in *Was Bewegt Uns? Menschen Im Spannungsfeld Zwischen Mobilität Und Beschleunigung*, ed. Christian Hoffstadt (Bochum, Germany: Projektverlag, 2010), 283–311.

⁷³ That is not to say it was an uncontroversial case. Harrington, *Reenchanted Science: Holism in German Culture from Wilhelm II to Hitler*, 147; Goldenberg, “Goldstein and Gelb’s Case Schn.: A Classic Case in Neuropsychology?”; Marotta and Behrmann, “Patient Schn.”

⁷⁴ The nature of head wounds and their “behavioral sequelae” drastically changed during the war as the methods of warfare, in particular the kinds of weaponry, changed. Penetrating wounds of the brain became more common because more soldiers survived them, in part due to new methods of clinical treatment but in large part due to the “self-sterilizing” nature of the projectiles, due to the heat generated from their speed of entry as they seared the tissue. Jennifer Gurd, Udo Kischka, and John Marshall, *Handbook of Clinical Neuropsychology* (Oxford: Oxford University Press, 2010), 4–5.

underwent surgery to remove the fragments of metal from his brain tissue in the occipital lobes. The operation was considered a success, and after a few more weeks convalescing, he was transferred to a new clinic for the rehabilitation of patients with similar brain injuries. There, the case of “Schn.” was born.⁷⁵

Goldstein and his collaborator, the research psychologist Adh mar Gelb (1887-1936) who was one of the pioneers of Gestalt psychology, ran Schneider through countless series of tests to monitor his recovery.⁷⁶ In particular, they used a device called a “tachistoscope,” which flashed images at precisely calibrated speeds in order to measure subjects’ reaction times.⁷⁷ Gelb had modified the instrument specifically for these clinical purposes so that both he and Goldstein could watch the image at the same time they projected for the patient to see.⁷⁸ For months, they observed Schneider’s reactions and found very little unusual or remarkable. He took longer to recognize the images and generally was a very slow reader, but that was all they observed, at first.⁷⁹

Then one day, Goldstein and Gelb noticed Schneider always moved his fingers or his head, ever so slightly, as he slowly read the projected letters and words. If they tried

⁷⁵ Goldstein and Gelb initially withheld the full name of the patient, abbreviating his case history to “Schn.” Goldstein and Gelb, “Psychologische Analysen hirnpathologischer Falle auf Grund von Untersuchungen Hirnverletzter”; Goldenberg, “Goldstein and Gelb’s Case Schn.: A Classic Case in Neuropsychology?”.

⁷⁶ Ash, *Gestalt Psychology in German Culture*, 275; Spiegelberg, *Phenomenology in Psychology and Psychiatry*.

⁷⁷ Ruth Benschop, “What Is a Tachistoscope? Historical Explorations of an Instrument,” *Science in Context* 11, no. 1 (1998): 23–50; Jimena Canales, *A Tenth of a Second : A History* (Chicago: University of Chicago Press, 2009).

⁷⁸ Ash, *Gestalt Psychology in German Culture*, 275.

⁷⁹ Goldstein and Gelb, “Analysis of a Case of Figural Blindness,” 316.

to prevent him from moving either his head or hands, however, suddenly he said he could not read anything. Apparently, he used his fingers to trace in the air the shapes of the letters and words, and he was able to “translate” the movements of his hands, or head, in order to recognize, or “read,” the words.⁸⁰

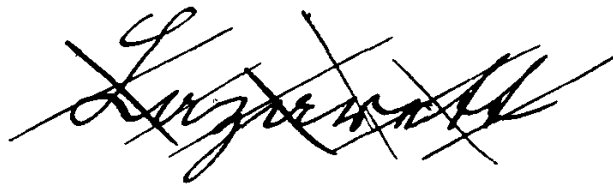


Figure 1. Schneider was unable to recognize the word “Lazarett” (“base hospital”) because of the strike-through lines, which prevented him from being able to distinguish letters by tracing their lines.⁸¹

Whereas normal individuals, according to Goldstein and Gelb, read and recognize words because they “visually *grasped* the *whole*,” Schneider lacked that ability. He could no longer “experience compactly organized visual impressions” as “unitary, self-contained wholes.” In other words, as a result of the injury to the back of his head, he became “word-blind.” And, to a great extent, he was now “mind-blind” too. He could see but he did not recognize what he saw, not by the normal process of “Gestalt seeing,” or seeing things “whole.”⁸² Such language was significant. It cued Goldstein and Gelb’s close involvement with the new experimental enterprise known as

⁸⁰ “If no ‘tracing’ movements were permitted, he invariably declared that he did not know what had been shown him.” *Ibid.*, 319.

⁸¹ Goldstein and Gelb, “Psychologische Analysen hirnpathologischer Fälle auf Grund von Untersuchungen Hirnverletzter,” 23.

⁸² Goldstein and Gelb, “Analysis of a Case of Figural Blindness,” 320.

“Gestalt psychology.”⁸³ For them and for other Gestalt researchers, such as Max Wertheimer and Wolfgang Köhler, this way of describing vision and visual disorders pointed to a *whole* new way of studying the mind and, ultimately, critiquing the foundations of knowledge.⁸⁴

However, there was one other aspect of Schneider’s condition that was just as subtle and equally significant as his “figural blindness.” Apparently, he never realized what he was doing as he traced the letters in the air with parts of his body. Even when Goldstein pointed it out to him, Schneider remained skeptical and unconvinced.

An especially interesting aspect of the case was the patient's own ignorance of using this method. Even after our discovery we found it difficult to persuade him that his procedure was not the customary one. He showed very clearly that he considered it inevitable for people to 'read' in this way.⁸⁵

Schneider believed that his way of “reading” was normal. And, in some ways, it was—for *him*. This was the only way he was able to read after his brain injury. By moving his head or his fingers, he managed to circumvent his deficit by “seeing” through the movements of his body. But crucially, he did not do so deliberately or with any explicit awareness. That was in large part what made it feel normal to him—because he did not *feel* it in the

⁸³ Furthermore, Goldstein fostered his relationship with Gestalt theorists throughout the 1920s by serving on the editorial board of its flagship journal *Psychologische Forschung*. Goldstein only wrote later, looking back, “I was impressed by the demonstrations of Wertheimer and the Gestalt psychologists...I tried to apply this principle to the study of the behavior of my patients....But later I became increasingly aware of the difference between the Gestalt theory and my own organismic concept. So I think it is not justified that I am often considered a ‘Gestaltist.’” Kurt Goldstein, “Notes on the Development of My Concepts,” in *Selected Papers: Ausgewählte Schriften*, ed. Aron Gurwitsch, Else M. Goldstein Haudek, and William E. Haudek (1959;: Springer, 1971), 10; Teuber, “Kurt Goldstein’s Role in the Development of Neuropsychology,” 301; Ash, *Gestalt Psychology in German Culture*, 281–283; Harrington, *Reenchanted Science: Holism in German Culture from Wilhelm II to Hitler*, 152–153.

⁸⁴ It is also significant that Ludwik Fleck likely drew from the work of Gestalt theorists to formulate his own theory of knowledge [*Erkenntnistheorie*] Fleck, *Genesis and Development of a Scientific Fact*.

⁸⁵ Gelb and Goldstein 1918 in Ellis 1938, 317.

first place. Goldstein reasoned, therefore, that Schneider's "ignorance" was a very important part of his recovery and rehabilitation.

In the late 1920s, Goldstein developed what he called the "organismic" approach to the study of behavior. It was "indefensible" and "disastrous," he complained, to try to isolate behaviors into abstractions and then generalize them as signs of pathology for all people.⁸⁶ Pathological behaviors could never be understood without considering the individual's unique circumstances. And so with the case of "Schn." His unawareness of his inability to read was due to the fact that his "whole organism" had adapted to his limitation, *even without him knowing*. In an effort to recover, he effectively hid from himself his own effort. That was his particular way, through his "whole organism," to re-establish a *new normal*, a new kind of *health*. It was his way of responding to the limitations created by his brain injury without causing him undue distress from the painful awareness of his constant struggle.

The *awareness* of illness was not always a sign of health. Goldstein witnessed scores of soldiers who were all too aware of their injuries and disabilities. This awareness often created for them further problems that extended beyond the scope of what local brain damage could have caused alone. Certain individuals, for example, who initially showed only occasional trouble speaking might cease trying to talk altogether. Despite the limited area of injury to the brain, they became utterly unresponsive and despondent. For others, paralysis might spread over their entire body, despite their

⁸⁶ Kurt Goldstein, "Das Symptom, seine Entstehung und Bedeutung für unsere Auffassung vom Bau und von der Funktion des Nervensystems," *Archiv für Psychiatrie* 76 (1925): 85–86; Kurt Goldstein, *Human Nature in the Light of Psychopathology* (Cambridge, Mass: Harvard Univ. Press, 1940), 152.

lacking sufficient nerve damage. Instead of a series of isolated symptoms caused by isolated brain or nerve lesions, therefore, Goldstein argued that these patients were overcome by a total “catastrophic reaction,” or “shock.” But this shock, he stressed, was entirely *biological*. It began in the brain and sent ripples throughout the entire body. It affected the “whole organism.”⁸⁷

Basic to preventing catastrophe, therefore, was avoiding having to acknowledge it. Goldstein believed the key to Schneider's recovery was his self-imposed ignorance. Because he did not sense that his head wound had forever changed him, he did not suffer an existential crisis.⁸⁸ Although he would never be the same, never “cured” or returned to his former state of health, even so, his “organism” somehow managed to “conceal” from him his incapacity, without his consent or conscious intervention. In Goldstein's view, “[T]hrough the unawareness of the defect, the mind is protected from catastrophic shocks, which the awareness would induce.”⁸⁹ It was not the patient who

⁸⁷ Goldstein's concept of “biological” was quite encompassing, however. He insisted on a type of renovated *Naturphilosophie* which considered the forces of nature as purposive and at times beneficent. Goldstein, “Das Symptom, seine Entstehung und Bedeutung für unsere Auffassung vom Bau und von der Funktion des Nervensystems,” 108; Kurt Goldstein, “Beobachtungen über die Veränderungen des Gesamtverhaltens bei Gehirnschädigung,” *Monatsschrift für Psychiatrie und Neurologie* 68 (1928): 224–225, 228; Goldstein, *The Organism*, 17–18, 22ff.

⁸⁸ “The catastrophic condition and anxiety can be understood only as a reaction of the personality to the danger to which he is exposed by the impossibility of realizing his essential capacities, due to the failure. The observations brought us to characterize anxiety in general as the *subjective experience of being in danger of losing 'existence.'*” Goldstein, “Notes on the Development of My Concepts,” 5–6.

⁸⁹ Goldstein, “Beobachtungen über die Veränderungen des Gesamtverhaltens bei Gehirnschädigung,” 242.

had “actively avoided” the catastrophe, but his organism, through the “goodness of nature,” that “passively protected” him from it.⁹⁰

The “*organism*” was one of Goldstein's favorite words. On the one hand, he used it to critique classical localization theory for its strangle-hold on psychiatry.⁹¹ The organism—not just the brain—was the locus of an individual's actions. In this sense, it resembled the person. On the other hand, he invoked it to critique purely psychological descriptions.⁹² To comprehend the overall change in a patient was only possible “biologically,” in terms of the “life-expressions of the organism.”⁹³ As a result, the “organism” conveyed an ambiguous but powerful meaning, subsuming both the organic and the personal, existential aspects of behavior.

Following the war, Goldstein continued to reflect on Schneider's particular form of unawareness. The case became in many ways his touchstone for interpreting the cases of many future patients with brain disease and injuries. It prompted him to rethink a number of assumptions in medicine, such as the relationship between symptom and

⁹⁰ “Sie vermeiden nach Möglichkeit alle Situationen, in denen katastrophale Reaktionen auftreten können. Allerdings ist der Ausdruck: die Kranken *vermeiden* diese Situation, eigentlich nicht ganz richtig...Die gefährliche Situation wird also weniger aktiv vermieden, als daß der Kranke passiv von ihr abgeschlossen wird.” Ibid., 229; Goldstein, *The Organism*, 51.

⁹¹ Goldstein, “Beobachtungen über die Veränderungen des Gesamtverhaltens bei Gehirnschädigung,” 218–220; David Ludwig, “Language and Human Nature: Kurt Goldstein’s Neurolinguistic Foundation of a Holistic Philosophy,” *Journal of the History of the Behavioral Sciences* 48, no. 1 (Winter 2012): 40–54.

⁹² In equal measure, he criticized the anti-localizationists for being “too general” and grossly neglecting localization as alleged “brain mythology.” Goldstein, “Beobachtungen über die Veränderungen des Gesamtverhaltens bei Gehirnschädigung,” 218.

⁹³ “[D]aß die Veränderungen des Gesamtverhaltens nicht nur psychologisch zu erfassen sind, ja rein psychologisch überhaupt eigentlich gar nicht völlig erfaßbar sind, sondern als Lebenserscheinungen, als Lebensäußerungen des Organismus begriffen werden müssen” Ibid.

recovery, and above all, the concepts of disease and health.⁹⁴ Accordingly, the historian of science Anne Harrington dubs Schneider the “Anna O. of holistic neurology.”⁹⁵ For each patient, unawareness was the key to understanding their recovery. It was now a “normal reaction” that “saved” them from debilitating despair.⁹⁶ Contrary to Freud’s view of Anna O., however, Goldstein believed that for patients like Johann Schneider, unawareness was not the problem but a part of the solution.

The Case of Merleau-Ponty

Schilder and Goldstein both left Europe for the United States—Schilder in 1928 when he was no longer assured of his succession to Wagner-Jauregg, and Goldstein in 1934 when he was forced to escape Nazi persecution.⁹⁷ Neither experienced the same degree of recognition and reception in America that they enjoyed in the early years following the First World War.⁹⁸ While they struggled to adapt to their new “normal” in their new country, a young French philosopher, Maurice Merleau-Ponty, eagerly adapted their ideas to his own. Much in the same way that Schilder and Goldstein

⁹⁴ Kurt Goldstein, “The Idea of Disease and Therapy,” *Review of Religion* 14 (1949): 229–40; Kurt Goldstein, “The Concept of Health, Disease and Therapy: Basic Ideas for an Organismic Psychotherapy,” *American Journal of Psychotherapy* 8, no. 4 (1954): 745–64.

⁹⁵ Harrington, *Reenchanted Science: Holism in German Culture from Wilhelm II to Hitler*, 147.

⁹⁶ Goldstein, “Beobachtungen über die Veränderungen des Gesamtverhaltens bei Gehirnschädigung,” 241; Goldstein, *Der Aufbau des Organismus. Einführung in die Biologie unter besonderer Berücksichtigung der Erfahrungen am kranken Menschen*, 26; Goldstein, *The Organism*, 51.

⁹⁷ Harrington, *Reenchanted Science: Holism in German Culture from Wilhelm II to Hitler*, 164–165; Hale, *The Rise and Crisis of Psychoanalysis in the United States*, 121–123.

⁹⁸ Harrington, *Reenchanted Science: Holism in German Culture from Wilhelm II to Hitler*, 173; Hale, *The Rise and Crisis of Psychoanalysis in the United States*, 123.

readily assimilated phenomenological and existential philosophy into their clinical writing, Merleau-Ponty reciprocally relied on their clinical case studies to build his own philosophy of behavior and perception.⁹⁹

In the late 1920s, Merleau-Ponty attended lectures by Edmund Husserl in Paris and steeped himself in the research of Gestalt psychologists. In 1937, he was introduced to Kurt Goldstein's ideas through the philosopher and former student of Goldstein, Aron Gurwitsch. In 1939, he read French neurologist Jean Lhermitte's *L'Image de notre corps*, which was heavily indebted to Schilder. Together, Goldstein and Schilder, transmitted via Gurwitsch and Lhermitte, exerted a tremendous impact on Merleau-Ponty's philosophical corpus, especially his signature emphasis on perception and the body.¹⁰⁰

The pivot of Merleau-Ponty's entire phenomenological philosophy was the lived body, indeed, he called it "the pivot of the world."¹⁰¹ He wanted ultimately to describe the contact between body and world as the creation of "perception." One of the best ways to see this connection, he thought, was to examine a case in which it became unhinged, specifically, the case of anosognosia.

⁹⁹ Herbert Spiegelberg, *The Phenomenological Movement: A Historical Introduction*, *Phaenomenologica* 5-6 (Hague: Nijhoff, 1960), 516–557; Spiegelberg, *Phenomenology in Psychology and Psychiatry*, chap. 12, 13; Harrington, *Reenchanting Science: Holism in German Culture from Wilhelm II to Hitler*, 158; Douwe Tiemersma, "'Body-Image' and 'Body-Schema' in the Existential Phenomenology of Merleau-Ponty," *Journal of the British Society for Phenomenology* 13, no. 3 (1982): 246–53.

¹⁰⁰ Gary Gutting, *French Philosophy in the Twentieth Century* (Cambridge University Press, 2001), 181–186; Carman, "The Body in Husserl and Merleau-Ponty," 206; Lester E. Embree, *The Collected Works of Aron Gurwitsch (1901-1973): Volume I: Constitutive Phenomenology in Historical Perspective* (Springer, 2009), 47; J. J. Lhermitte, *L'image du moi corporel et ses déformations pathologiques* (Paris: G. Doin éd., 1937); Jean Lhermitte, *L'image de notre corps* (Paris: Editions de la Nouvelle revue critique, 1939).

¹⁰¹ Merleau-Ponty, *Phenomenology of Perception*, 2002, 94.

Merleau-Ponty appreciated the generative ambiguity of the diagnosis, much like Schilder and Goldstein. On the one hand, it was the result of a “blind,” impersonal lesion that affected physiological mechanisms of the brain. On the other hand, it was the expression of a very directed kind of “blindness” or selective ignorance that appeared quite purposeful and particular to the individual’s situation. The problem was how to combine these two aspects, each of which was a fairly accurate description on their own, but which, if taken together, became deeply paradoxical.

Merleau-Ponty confessed at first, “it is difficult to see what ground could be common to ‘physiological facts’ which are in space and ‘psychic facts’ which are nowhere.”¹⁰² Typically, philosophers overlay these two “components” in “parallel” without connecting them. However, Merleau-Ponty wanted to “contrive some meeting-point for them” that would integrate the “third person processes and the personal acts” into a “common middle term.”¹⁰³ For this “middle term,” he invoked Schilder’s phrase, “organic repression.” It signified, he thought, the kind of “un-Cartesian” way of thinking through the intellectual impasse imposed on the body and mind. It pointed a middle way toward a new common ground.¹⁰⁴

Merleau-Ponty was convinced that anosognosia involved more than a brain disease. But he also did not think it was entirely “psychic” either. There was more to it than either lesion or repression could explain. Instead, the only way to come nearer

¹⁰² Ibid.

¹⁰³ Ibid., 89.

¹⁰⁴ “[O]rganic repression.’ These un-Cartesian terms force us to form the idea of an organic thought through which the relation of the ‘psychic’ to the ‘physiological’ becomes conceivable.” Ibid.

understanding anosognosia was by taking into account the patient's entire "situation."¹⁰⁵ Reminiscent of Goldstein's reference to "whole behavior" and the "whole organism," Merleau-Ponty meant by the "situation" the individual's "being-in-the-world" (*être au monde*). This was his way around the contradiction. "Being in the world," a modified version of Heidegger's *In-der-Welt-sein*, meant being open to the world, but neither passively receiving naïve impressions nor completely actively constructing *a priori* mental schemata. Instead, Merleau-Ponty meant for it to signal something in between empiricism and intellectualism, a "movement to and fro of existence," in which one approaches the world of perception neither empty-handed nor all-knowing but indeterminately, dimly aware.¹⁰⁶

This was also Merleau-Ponty's way of explaining the phenomenon of anosognosia. Goldstein had suggested that the organism already "knew" about the defect in order to compensate for it and to hide it from the patient's awareness. However, he did not mean to imply that the organism was exactly "conscious" in doing so. The quality of the awareness was indeterminate and in-between. It was neither fully present nor completely absent. That was how Merleau-Ponty approached the question of a patient's anosognosic behavior:

In reality the anosognosic is not simply ignorant of the existence of his paralysed limb: he can evade his deficiency only because he knows where he risks encountering it...[W]e turn aside from those areas of our life in which we might meet this nothingness, but this very fact necessitates that we intuit them. In the same way the anosognosic leaves his paralysed arm out of account in order not

¹⁰⁵ Ibid., 92.

¹⁰⁶ Ibid., 101.

to have to feel his handicap, but this means that he has a preconscious knowledge of it.¹⁰⁷

And so, instead of declaring the patient conscious or unconscious, blindly unaware or intentionally in denial, the philosopher opted for “preconscious,” or the as-yet-undefined, about-to-be state of un/awareness.¹⁰⁸

This was his answer to overcoming the “Cartesian” dualism that those like Babinski perpetuated. By recognizing the “pre-personal” and “pre-objective” dimensions of perceptual awareness, Merleau-Ponty believed he could “effect the union of the ‘psychic’ and the ‘physiological.’”¹⁰⁹ But the result was a very paradoxical portrait of anosognosia: “The patient therefore realizes his disability precisely in so far as he is ignorant of it, and is ignorant of it precisely to the extent that he knows of it.” That, Merleau-Ponty concluded, “is the paradox of all being in the world.”¹¹⁰ It was normal, therefore, not pathological, to be ignorant, or not explicitly aware, of one’s body. After all, that was the business of the body, to cover its own tracks, to keep itself hidden, as the “unperceived term in the centre of the world.”¹¹¹ That was why Merleau-Ponty

¹⁰⁷ Ibid., 95.

¹⁰⁸ Ibid., 6–7, 35–36. The German word “*Bewusstwerden*” approximates this processual, coming-to-be quality of not-yet but-almost consciousness.

¹⁰⁹ Ibid., 92.

¹¹⁰ Ibid., 95; Maurice Merleau-Ponty, *Phenomenology of Perception*, trans. Donald A. Landes (New York: Routledge, 2013), 84; Merleau-Ponty, *Phénoménologie de la perception*, 111–112.

¹¹¹ Merleau-Ponty, *Phenomenology of Perception*, 2002, 94.

could say, “nothing is more difficult than to have a sense for precisely *what we see*,” for, “perception hides itself from itself.”¹¹²

∞

After World War I, the history of the unawareness of illness took a sharp turn away from the fixation on anatomy. The soldiers-turned-patients offered living proof of the inadequacy of rigid localization by virtue of their remarkable feats of recovery. Doctors like Schilder and Goldstein recognized the patient's behavior as more than the outcome of the brain alone. In particular, they viewed the unawareness of illness as potentially adaptive and personally meaningful. In their minds, it was not a pure deficiency or a straightforward disease but a complex, covert strategy of coping. Sometimes, the loss of self-awareness was the price of self-preservation. Both physicians were drawn to the phenomenon of anosognosia because they were able to mold it in service of their own search to re-discover and re-define the meaning of medicine and the idea of health. In that sense, their focus on the loss of self-perception reflected a desire to re-assert their own. Although the project of revitalizing medicine took on different meaning after World War II, Goldstein's and Schilder's efforts were not lost on all, as the case of Merleau-Ponty attests. In the next chapter, we will see how their hybrid and holistic ideas spread across the Atlantic and helped shape American medicine in the latter half of the twentieth century.

¹¹² Merleau-Ponty, *Phénoménologie de la perception*, 85; Merleau-Ponty, *Phenomenology of Perception*, 2002, 67; Merleau-Ponty, *Phenomenology of Perception*, 2013, 59; Sean Kelly, “What Do We See (When We Do)?,” in *Reading Merleau-Ponty: On Phenomenology of Perception*, ed. Thomas Baldwin (New York: Routledge, 2007), 27.

Chapter 4

Filling In the Patient's View:

Denial Syndromes and American Neuropsychiatry of the Mid-Twentieth Century

"It is precisely those aforementioned moments where the utmost need to communicate coincides with the utmost speechlessness."¹

On March 14, 1949, a thirty-eight year old woman was taken to Mount Sinai Hospital in New York after several weeks of intense headaches and vomiting. Her husband told the doctors that she complained of dizziness and the feeling of pressure inside her head for over a year but assumed it was the result of "emotional tension."² Her condition deteriorated in recent weeks, however. Her husband noticed a "change in behavior" during which she became more "forgetful" and often showed "apparent unconcern," whereas before she had been "compulsive" and "worrisome."³ He explained that his wife had always been a "devoted mother," but recently she had become "easy-going and relaxed," so much so that one day he found their young child "playing with razor blades" as his wife looked on oblivious to the danger.⁴

The physician who examined the woman was neuropsychiatrist Edwin Weinstein (1909-1998). He reported that "F.M." (the patient's initials) showed a slight hemiparesis, or weakness, on the left side of her body as well as a positive Babinski response in her

¹ Peter Handke, *Wunschloses Unglück* (Manchester: Manchester University Press, 1993), 49.

² Edwin A. Weinstein and Robert L. Kahn, "The Syndrome of Anosognosia," *Archives of Neurology and Psychiatry* 64 (1950): 781.

³ *Ibid.*

⁴ *Ibid.*

left plantar tendons. In addition, there was swelling, or papilledema, in the head of the optic nerve in her right eye. According to her electroencephalogram, there was diminished activity from the right frontal electrodes on her scalp. Then, four days after her admission, her left leg began to jerk in clonic convulsion. After a week in the hospital, she finally underwent a craniotomy, during which a “dark bluish vascular tumor” was discovered in the right temporal lobe and was later confirmed to be a “spongioblastoma,” or tumor made of neuroepithelial cells.⁵

F.M.’s troubles only seemed to worsen after the operation. She suffered a complete paralysis in her left arm and extreme weakness in her left leg. Moreover, Weinstein noted, “The patient denied there was anything the matter with her left arm or leg...[and] also denied that an operation had been performed.”⁶ Repeatedly when asked to try to raise her left arm, she moved her left leg instead. When Weinstein pointed out to her the mistake, she exclaimed, “Oh, some people call it an arm; some a leg. What’s the difference!”⁷ Then, he lifted her left arm in front of her face and asked her to identify it, to which she answered that it belonged to him instead. Even when shown her shaven head in a mirror, she denied having had surgery, complaining, “It’s ridiculous. Why, an operation would be the last thing...Why are people bothering me about a nonexistent operation?”⁸ Gradually, she grew “bitter” and “irascible,” accusing

⁵ Ibid.

⁶ Ibid., 782.

⁷ Ibid.

⁸ Ibid.

her husband of trying to confine her to a mental institution. She called the hospital “Mount Cyanide” and accused the nurses of trying to poison her. On another occasion, she tried to fondle the breasts of one of the nurses while making “vulgar remarks.”⁹

A week and a half after her surgery, she finally admitted that her left arm was weak, but she continued to deny having had surgery—saying only that she had “a cancer,” all the while joking about the “bulge” on her head. On April, 25, 1949, she was discharged to another hospital as “a chronic patient.”¹⁰ With that, Weinstein concluded his discussion of her case, adding only a final, tantalizing remark:

Prior to coming to the hospital, the patient had expressed a great fear of having to have an operation: “It will make me soft brained, and I’ll end up in an asylum.”¹¹

He did not say whether he thought her fear had come true.

In 1950, Weinstein and the research psychologist Robert Kahn (1918-) described the case of “F.M.” along with twenty-one others in a paper titled, “The Syndrome of Anosognosia.”¹² Over the next decade, Weinstein and Kahn devoted nearly all of their research to anosognosia, which culminated in the first book ever dedicated exclusively to the disorder, *Denial of Illness: Symbolic and Physiological Aspects*.¹³ The two men openly embraced the ideas of both Paul Schilder and Kurt Goldstein, each of whom had recently emigrated to the United States. Building from their descriptions of anosognosia

⁹ Ibid.

¹⁰ Ibid., 783.

¹¹ Ibid.

¹² Weinstein and Kahn, “The Syndrome of Anosognosia.”

¹³ Weinstein and Kahn, *Denial of Illness: Symbolic and Physiological Aspects*.

as a “wish” or “drive to be well,” Weinstein and Kahn further argued that it occurred only in patients who already possessed “compulsive, perfectionistic” personalities,¹⁴ whose friends and relatives had long described as being afraid of illness and associating it with “sin,” “disgrace,” and “imperfection.”¹⁵

Beyond some anonymous “organic urge” to be well, therefore, Weinstein and Kahn “personalized” anosognosia. In fact, they argued that nearly every “organic” injury and disease affecting the brain ultimately depended on “personality differences”¹⁶ and “pre-existing social and cultural factors.”¹⁷ Anosognosia was not specific to an area of damage in the brain, nor to a specific individual, but to a specific pattern of relationships in the culture and society.

This chapter explores Weinstein and Kahn’s research on the denial of illness in the middle of the twentieth century. It focuses in particular on their efforts to expand the diagnostic criteria of anosognosia and subsume it under “syndromes of denial.” The discussion then opens onto the broader stage in which Weinstein and Kahn performed their research, specifically addressing the “culture-and-personality” movement of the 1930s and Weinstein’s personal experience as an army neuropsychiatrist in World War II. Finally, the chapter examines the historiographical question of just what was

¹⁴ Edwin A. Weinstein and Robert L. Kahn, “Personality Factors in Denial of Illness,” *AMA Arch Neurol Psychiatry* 69, no. 3 (March 1, 1953): 356.

¹⁵ Weinstein and Kahn, *Denial of Illness: Symbolic and Physiological Aspects*, 73195; Weinstein and Kahn, “Personality Factors in Denial of Illness,” 359.

¹⁶ Weinstein, Kahn, and Slote, “Withdrawal, Inattention, and Pain Asymbolia,” 235.

¹⁷ Weinstein and Kahn, *Denial of Illness: Symbolic and Physiological Aspects*, 83.

“neuropsychiatry” immediately following the war. I argue that by closely tracking Weinstein’s early medical career, particularly his wide-ranging approach to the study of anosognosia, we can better appreciate the degrees of professional fusion and confusion in mid-century American psychiatry and neurology.

A Unifying Concept

Not long after their emigration to the United States, Schilder and Goldstein sparked the young Weinstein’s early interest in anosognosia.¹⁸ Contrary to Gabriel Anton and Joseph Babinski, who emphasized the *exclusively* organic, focal, and localized nature of the disorder, Schilder and Goldstein each maintained that focal lesions alone could not explain anosognosia. Instead, doctors needed to consider the patient’s entire behavior [*Gesamtverhalten*] in order to understand how the unawareness of illness might serve to protect the individual and deflect the frightening recognition of an injury or disability. Beyond the dictates of cerebral localization, therefore, Schilder and Goldstein each argued that anosognosia had more to do with the patient’s deep-seated

¹⁸ In fact, Weinstein’s first internship in psychiatry at the New York State Psychiatric Institute from 1936 to 1937 overlapped with Goldstein’s stint there. Also during that period, Schilder worked in New York as the clinical director of psychiatry at the Bellevue Hospital, having permanently left Vienna in 1929. Thus, once Weinstein began to study the denial of illness in earnest by 1938, he was already quite familiar with the ideas of both Schilder and Goldstein. Harrington, *Reenchanting Science: Holism in German Culture from Wilhelm II to Hitler*, 165; Langer, “Paul Ferdinand Schilder: Leben und Werk,” 92.

“drive” or “wish” to be well, preserve the “feeling of integrity of one’s body,”¹⁹ and avoid “catastrophe.”²⁰

Crucially, neither Schilder nor Goldstein abandoned their belief in the doctrine of cerebral localization. Rather, their versions of “holistic” medicine were more geared toward integrating, in Schilder’s words, the “brain and personality,” or in Goldstein’s words, the “organism and environment.”²¹ That was not always the case with other proponents of holistic science and medicine, however. Some physicians and physiologists, not to mention many philosophers and psychologists, rejected the doctrine of cerebral localization. Foremost among them in the United States at the time was Harvard psychologist Karl Lashley (1890-1958), who by the end of the 1920s was convinced that the cerebral cortex was “equipotential” in function and not amenable to localization.²²

For the next two decades, Lashley’s principle of “equipotentiality” dominated the textbooks of physiological psychology and the new subdiscipline of “neuropsychology.” By the time Weinstein finished medical school in 1935 and Kahn

¹⁹ Schilder, *Das Körperschema: Ein Beitrag zur Lehre vom Bewusstsein des eigenen Körpers*, 28.

²⁰ E.A. Weinstein and M. Cole, “Concepts of Anosognosia,” in *Problems of Dynamic Neurology, an International Volume: Studies on the Higher Functions of the Human Nervous System*, ed. Lipman Halpern (Jerusalem: Jerusalem Post Press, 1963), 256.

²¹ See previous chapter for an expanded discussion. Schilder, *Brain and Personality: Studies in the Psychological Aspects of Cerebral Neuropathology and the Neuropsychiatric Aspects of the Motility of Schizophrenics*; Goldstein, *The Organism*; Ludwig, “Language and Human Nature: Kurt Goldstein’s Neurolinguistic Foundation of a Holistic Philosophy.”

²² Nadine M. Weidman, *Constructing Scientific Psychology: Karl Lashley’s Mind-Brain Debates* (Cambridge University Press, 1999), 52–58; Harrington, “The Brain and Behavioral Sciences,” 520; Karl S. Lashley, *Brain Mechanisms and Intelligence: A Quantitative Study of Injuries to the Brain*, Behavior Research Fund. Monographs (Chicago: The University of Chicago Press, 1929).

earned his Ph.D. in psychology in 1953, both were well aware of Lashley's critique. Although Weinstein stayed on after medical school at Northwestern University to work for the physiologist Stephen Ranson (1880-1942), studying the effect of lesions on the hypothalamus and the third ventricle, he remained convinced of Lashley's basic teaching that higher mental function in general and the cortex in particular could never be localized.²³ That was why he and Kahn rejected the prevailing view in the United States, articulated by the Los Angeles-based neurologists, Johannes Nielsen, Karl von Hagen, and Elinor Ives, that anosognosia was a "disturbance of the body-scheme [sic]" whose "sole cause" was a lesion in either the internal capsule or the parietal lobe of the right, cerebral hemisphere.²⁴ Lesions alone, no matter where they were located in the brain, could not explain denial.

²³ George P. Prigatano and Edwin Weinstein, "Edwin A. Weinstein's Contributions to Neuropsychological Rehabilitation," *Neuropsychological Rehabilitation: An International Journal* 6, no. 4 (1996): 306; Aura Edward Severinghaus, ed., *Neurology: A Medical Discipline Takes Stock*, DHEW Publication, no. (NIH) 72-175 (Bethesda, Md: National Institutes of Health, 1971), 17; H.W. Magoun, "The Role of Research Institutes in the Advancement of Neuroscience: Ranson's Institute of Neurology 1928-1942," in *The Neurosciences, Paths of Discovery*, ed. F.O. Schmitt et al. (MIT Press, 1975), 514-27.

²⁴ The earliest clinical cases of anosognosia in the United States were reported by Nielsen, von Hagen, and Ives. They focused their discussion of anosognosia with reference to the "body scheme" [sic], especially citing Paul Schilder and his co-workers from Vienna, Otto Pötzl and Josef Gerstmann (the latter whose emigration to America Schilder later sponsored). Historian Anne Harrington has examined in detail the history of hemispheric asymmetry and the emergence of so-called "right-brain" disorders such as anosognosia. Elinor R. Ives and Johannes M. Nielsen, "Disturbance of Body Scheme. Delusion of Absence of Part of Body in Two Cases with Autopsy Verification of the Lesions," *Bulletin of the Los Angeles Neurological Society* 2 (1937): 120-25; Karl O. von Hagen and Elinor R. Ives, "Anosognosia (Babinski), Imperception of Hemiplegia. Report of Six Cases, One with Autopsy," *Bulletin of the Los Angeles Neurological Society* 2 (1937): 95-103; Karl O. von Hagen and Elinor R. Ives, "Two Autopsied Cases of Anosognosia," *Bulletin of the Los Angeles Neurological Society* 4 (1939): 41-44; J.M. Nielsen and R.B. Raney, "Symptomatology of Tumors of the Third Ventricle," *Bulletin of the Los Angeles Neurological Society* 4 (1939): 1-7; Josef Gerstmann and Paul Schilder, "Studien über Bewegungsstörungen," *Zeitschrift fuer die gesamte Neurologie und Psychiatrie (Neurologisches Centralblatt)* 58 (1920): 266-75; Josef Gerstmann, "Fingeragnosie: eine umschriebene Störung der Orientierung am eigenen Körper," *Wiener klinische Wochenschrift* 37 (1924): 1010-12; Pötzl, "Über Störungen der Selbstwahrnehmung bei linkseitiger Hemiplegie"; Lazaros C. Triarhou, "Josef Gerstmann (1887-1969)," *Journal of Neurology* 255, no. 4 (2008): 614-15; Harrington, *Medicine, Mind, and the Double Brain*, 274-275.

In 1949, Weinstein, Kahn, and their mentor, Sidney Tarachow (1908-1965),²⁵ presented their first collaborative study of the denial of illness at the annual conference of the American Neurological Association.²⁶ In a clear nod to Goldstein, who served as the sole discussant at the conference, they argued that the denial of illness represented “a drive to health” and the “attempt to attain the unfulfilled wish of not being ill.”²⁷ Despite all twenty-four patients having verifiable brain tumors, they deemed that none of these tumors “caused” the denial but merely created the “conditions” for a new “milieu of brain function”—not a “loss of function” but an “altered mode of interaction.”²⁸ Gone was the emphasis on hemisphere specificity, and for that matter, organic specificity.

Also absent from Weinstein and Kahn’s discussions was the typical symptom specificity, that is, the focus of the patient’s unawareness or denial of, say, blindness but not back pain. According to Weinstein and Kahn, their patients denied multiple aspects

²⁵ Weinstein and Kahn, *Denial of Illness: Symbolic and Physiological Aspects*, v; Sylvan Keiser and William A. Console, “Sidney Tarachow, 1908–1965,” *Journal of the American Psychoanalytic Association* 14, no. 4 (1966): 858; J. Frosch, “Sidney Tarachow: 1908-1965,” *The International Journal of Psycho-Analysis* 48, no. 2 (1967): 321–22; William C. Manson, “Abram Kardiner and the Neo-Freudian Alternative in Culture and Personality,” in *Malinowski, Rivers, Benedict and Others: Essays on Culture and Personality*, ed. George W. Stocking (Univ of Wisconsin Press, 1986); J. Frosch, “The New York Psychoanalytic Civil War,” *Journal of the American Psychoanalytic Association* 39, no. 4 (1991): 1045, 1048.

²⁶ This is the only time Tarachow ever collaborated with Weinstein and Kahn, although they credit him for having guided them in the early stages of their research on the denial of illness. Weinstein, Kahn, and Tarachow, “Denial of Illness in Brain Tumor”; Weinstein and Kahn, *Denial of Illness: Symbolic and Physiological Aspects*; Keiser and Console, “Sidney Tarachow, 1908–1965,” 858.

²⁷ Weinstein, Kahn, and Tarachow, “Denial of Illness in Brain Tumor,” 69.

²⁸ Weinstein and Kahn, “The Syndrome of Anosognosia,” 791; E.A. Weinstein and R.L. Kahn, “Patterns of Disorientation in Organic Brain Disease,” *Journal of Neuropathology and Clinical Neurology* 1, no. 3 (1951): 224; Weinstein and Kahn, “Personality Factors in Denial of Illness,” 356, 367.

about their illness, not just hemiplegia, and especially not just left-sided hemiplegia.²⁹ In addition, they denied being in a hospital, having a surgery, being incontinent, suffering burns, being impotent, having menstruated, and a whole host of other aspects not necessarily related to any brain trauma.³⁰ Therefore, in addition to the long-documented cases of the denial of blindness and hemiplegia, Weinstein and Kahn maintained that patients with the “anosognosic delusion” denied “whatever he feels is seriously wrong with him,” which might span a wide range of “inadequacies and traumatic experiences.”³¹ Accordingly, the denial of illness “never occurred as an isolated entity.” It always involved “more than a single defect.” It was “always an aspect of a more generalized disturbance.”³² And that disturbance fell within a vast spectrum of behaviors, some of which were easily missed while others were so conspicuous that most doctors mistook them for a general form of disorientation, “labeled ‘confusion.’”³³

Weinstein and Kahn virtually exploded the aetiological and behavioral specificity of anosognosia, and in doing so they drastically expanded the diagnostic criteria of just exactly what “denial” entailed. Rather than confining themselves to a patient’s verbal

²⁹ In the very opening lines of their book, Weinstein and Kahn stressed that anosognosia was not confined to left-sided hemiplegia, and more importantly was not circumscribed in the right, “non-dominant” hemisphere alone. Weinstein and Kahn, *Denial of Illness: Symbolic and Physiological Aspects*.

³⁰ Weinstein, Kahn, and Tarachow, “Denial of Illness in Brain Tumor”; Weinstein and Kahn, “The Syndrome of Anosognosia,” 776–778; Weinstein and Kahn, *Denial of Illness: Symbolic and Physiological Aspects*, 5, 10, 20, 85, 130–131.

³¹ Weinstein and Kahn, “The Syndrome of Anosognosia,” 773, 789.

³² *Ibid.*, 774–775, 789.

³³ *Ibid.*, 789; David McK. Rioch, “Psychopathological and Neuropathological Aspects of Consciousness,” in *Brain Mechanisms and Consciousness: a Symposium*, ed. Edgar Douglas Adrian and J. F. Delafresnaye (Oxford: Blackwell Scientific Publications, 1954), 474.

denial alone, they believed that any patient who misnamed the hospital, say, “Mount Sinai restaurant” or “Mount Sinus Hospital,” or relocated it, claiming that it was closer to his own neighborhood, exhibited certain “patterns of disorientation” that fit within what they called the “syndromes of denial.”³⁴ Patients who joked darkly about their situation, for example, calling Mount Sinai, “Mount Cyanide,” or patients who frequently used clichés such as, “Once you admit you’re sick, you are licked,” were also grouped under the denial syndrome. Even patients who were withdrawn and depressed or who refused to cooperate in physical therapy were likewise suspected of “implicit denial.”³⁵

While Weinstein and Kahn expanded the criteria for the “denial of illness,” they also re-interpreted its personal significance to the patient. They believed that it represented more than the patient’s “drive to be well” or avoidance from having to confront the reality of being sick. They argued that it was also the only way such patient could communicate her feelings about her predicament. By calling a paralyzed limb a “dummy” or “an old piece of equipment that doesn’t work,”³⁶ the patient deployed symbolic language as “vehicles for the expression of his needs and feelings.”³⁷ These symbolic aspects, they stressed, were just as important as physiological aspects. It was

³⁴ Weinstein and Kahn, “Patterns of Disorientation in Organic Brain Disease,” 223–224.

³⁵ Some withdrawal even resembled “the clinical picture of hysterical hemianesthesia, as they did not feel pinprick, thermal, touch, and vibratory stimuli.” Weinstein, Kahn, and Slote, “Withdrawal, Inattention, and Pain Asymbolia,” 238–239.

³⁶ Weinstein and Kahn, “Personality Factors in Denial of Illness,” 358–359.

³⁷ Edwin A. Weinstein, Robert L. Kahn, and Leroy A. Sugarman, “Phenomenon of Reduplication,” *AMA Arch Neurol Psychiatry* 67, no. 6 (June 1, 1952): 812.

just that the physiological features were sometimes too psychologically painful to communicate other than by indirect “symbolic modalities.”³⁸ In other words, even the *look* of unawareness was a “symbolic” way of communicating tacit understanding: “In the very manifestation of ‘unawareness’ of his incapacity,” they wrote, “there is indicated a very definite awareness of the traumatic implications.”³⁹ Even denial was a form of confession.

Still, the ultimate reason patients denied illness had to do with *who* they were, which emerged from the kinds of social interactions, or “interpersonal patterns,” in which they participated. To investigate such “interpersonal relations,” Weinstein and Kahn devised elaborate questionnaires and interviews for the patients’ relatives, friends, and co-workers, in order to assess what “personality type” best characterized the patient before the onset of illness.⁴⁰ Consequently, they discovered that those with anosognosia were “compulsive, rigid, worrisome, over-conscientious, domineering individuals,” whom friends and families often described as “extremely guilty over having any illness or imperfection.”⁴¹ Such patients were the “types” who preferred to be busy,

³⁸ Edwin Weinstein, Robert Kahn, and Sidney Malitz, “Confabulation as a Social Process,” *Psychiatry* 19, no. 4 (1956): 395; Edwin A. Weinstein and Robert L. Kahn, “Symbolic Reorganization in Brain Injuries,” in *American Handbook of Psychiatry*, ed. Silvano Arieti, vol. 1 (New York: Basic Books, 1959), 966; Edwin A. Weinstein, “Symbolic Aspects of Thalamic Pain,” *The Yale Journal of Biology and Medicine* 28, no. 3–4 (December 1955): 465–70; Weinstein and Kahn, *Denial of Illness: Symbolic and Physiological Aspects*, 97; Weinstein, “Symbolic Neurology and Psychoanalysis.”

³⁹ Weinstein, Kahn, and Slote, “Withdrawal, Inattention, and Pain Asymbolia,” 247.

⁴⁰ Weinstein and Kahn, “The Syndrome of Anosognosia,” 780; Weinstein and Kahn, “Personality Factors in Denial of Illness,” 357; Weinstein, Kahn, and Slote, “Withdrawal, Inattention, and Pain Asymbolia,” 237–238.

⁴¹ Weinstein, Kahn, and Tarachow, “Denial of Illness in Brain Tumor,” 68.

and “put off going to doctors,” rather than attract attention for any perceived idleness or laziness.⁴² Health and work were tied up for them:

For such people, health and work were ethical values. Through social relationships involving health and work, they gained a sense of “being.” When ill or unable to work they were isolated and, through lack of a relatedness, did not derive such a feeling of self.

These patients, Weinstein and Kahn declared, were driven to “perfection and superiority” out of a background of “considerable insecurity.”⁴³ Even before their illness, they were given to frequent moralizing and were preoccupied with cleanliness. Above all, they valued the feeling of “prestige in the eyes of others” and believed that any admission of weakness or acknowledgement of sickness detracted from their social status.⁴⁴ In other words, their fear, not their brain, determined their denial.⁴⁵

But really, who were “they”? Weinstein and Kahn, in their quest to define the particular “personality type,” typically neglected the particularities of any one individual person. Instead, with their charts and checklists, they usually included only the barest of anecdotal details about a particular patient, such as, “One man (case 3) would insist...” or “a patient might refuse...” or “often, a patient will deny...” or “Another claimed

⁴² Weinstein, Kahn, and Slotte, “Withdrawal, Inattention, and Pain Asymbolia,” 241.

⁴³ Weinstein and Kahn, “The Syndrome of Anosognosia,” 780.

⁴⁴ Weinstein and Kahn, “Personality Factors in Denial of Illness,” 359.

⁴⁵ Later Weinstein, working with Marvin Cole, pointed out that even Babinski had written that one of his patients “expressed a fear of paralysis” before she became anosognosic for hemiplegia. Here is the phrase from Babinski: “Ce qui contrastait avec la conservation apparente de l'intelligence de cette malade, c'est qu'elle semblait ignorer l'existence de l'hémiplégie presque complète dont elle était atteinte et qu'elle avait cependant redoutée pendant plusieurs années.” Weinstein and Cole, “Concepts of Anosognosia,” 266; Babinski, “Contribution à l'étude des troubles mentaux dans l'hémiplégie organique (anosognosie),” 845.

that..."⁴⁶ Unlike any other doctor who studied the unawareness of illness, Weinstein amassed literally hundreds of cases of patients, and with Kahn's help, scoured each one for their "symbolic and physiological aspects." Yet when it came time to write a particular patient's "history," despite the touted emphasis on "personality," they usually limited their discussion to a few perfunctory asides, such as F.M.'s comment to her husband about her fear of having surgery. Ironically, Weinstein and Kahn's interest in the patient's personality, indeed in the patient's needs and feelings, seems to have served as a "vehicle for the expression" of their own. That is, despite their professed interest in the patient's view, they treated it more like a codified abstraction generated by form-questionnaires and serial boxes checked "yes" or "no" to tally into "types" of personalities, not persons.⁴⁷ It was "the patient's view" as a prescribed and form-fitted expectation rather than any sustained, individual experience.

Together Weinstein and Kahn believed that this kind of abstract categorization was adequate to understand anosognosia as a "unifying concept."⁴⁸ But they were far from the first to approach the study of illness and behavior in such way. In the next section, we explore some of the important precedents that helped shape Weinstein and

⁴⁶ Weinstein and Kahn, "Symbolic Reorganization in Brain Injuries."

⁴⁷ British sociologist of medicine David Armstrong identifies the 1940s and 1950s as precisely a time when the patient's view became more visibly and systematically incorporated into the case history, where the new "schema" of the "extended history" included things like marital, occupational, and social history of the individual patient. Somewhat relatedly, in the 1980s, psychiatrist and anthropologist Arthur Kleinman argued for more a deliberate and sensitive clinical attention to the patient's experience of illness, but which arguably degenerated in the hands of some practitioners into yet another checklist of codified "feelings." Armstrong, "The Patient's View," 739–741; Kleinman, *The Illness Narratives*.

⁴⁸ Weinstein and Kahn, *Denial of Illness: Symbolic and Physiological Aspects*, v.

Kahn's understanding of what the "personality" was and why it mattered to their new concepts of anosognosia.⁴⁹

Culture and Combat Fatigue

In 1936, a group of psychiatrists and anthropologists led by Harry Stack Sullivan (1892-1949) and Edward Sapir (1884-1939) founded the Washington School of Psychiatry in Washington, D.C. The school was established for the stated goal of "postdoctoral training of psychiatrists and social scientists" in "techniques for personality study" and "participant observation."⁵⁰ Ten years earlier, Sullivan first met Sapir in Chicago after the death of Sapir's wife. According to one scholar, their meeting was the beginning of not only a friendship but also "a remarkable interdisciplinary cross-fertilization."⁵¹ After Sapir moved to Yale in 1931, he began offering a seminar centered around "culture and personality," incorporating some of the ideas of Sullivan's psychoanalytic psychiatry into his own research on the symbolic functions of language.⁵² Meanwhile, Sullivan increasingly emphasized the "personal-cultural" aspects of mental

⁴⁹ Weinstein and Cole, "Concepts of Anosognosia."

⁵⁰ Kvarnes, "The Washington School of Psychiatry--Past Dreams and Present Realities"; Douglas Noble and Donald L. Burnham, *History of the Washington Psychoanalytic Society and the Washington Psychoanalytic Institute* (Washington, 1969); Harry Stack Sullivan, "A Note on the Implications of Psychiatry, the Study of Interpersonal Relations, for Investigations in the Social Sciences," *American Journal of Sociology*, 1937, 861.

⁵¹ Manson, "Abram Kardiner and the Neo-Freudian Alternative in Culture and Personality," 77; Helen Swick Perry, *Psychiatrist of America: The Life of Harry Stack Sullivan* (Belknap Press, 1982), 242–245.

⁵² Manson, "Abram Kardiner and the Neo-Freudian Alternative in Culture and Personality," 77; Edward Sapir, "Why Cultural Anthropology Needs the Psychiatrist. 1938," *Psychiatry* 64, no. 1 (2001): 2–10; Laurence J. Kirmayer, "Sapir's Vision of Culture and Personality," *Psychiatry* 64, no. 1 (2001): 23–31; Edward Sapir, "Symbolism," *Encyclopaedia of the Social Sciences*: (New York: Macmillan, 1934).

illness and the impact of “interpersonal relations” among the patient, his family, friends, physicians, and, indeed, all of society.⁵³ With Sapir’s encouragement, Sullivan argued for the “fusion” of psychiatry and the social sciences.⁵⁴

Together, Sullivan and Sapir, along with anthropologist Ruth Benedict (1887-1948) and psychoanalyst Karen Horney (1885-1952), among others, taught at the Washington School in its early years and in 1938 founded the journal *Psychiatry: Interpersonal and Biological Processes*.⁵⁵ Although it was not until the late 1950s that Edwin Weinstein joined the Washington School, after the deaths of both Sullivan and Sapir, he later recounted that already by the 1930s, their work on symbolic language and interpersonal relations was a major influence on his early ideas about the denial of illness.⁵⁶ For that matter, by the 1940s, their work and generally that of the other “culture-and-personality” theorists, such as Margaret Mead (1901-1978) and Gregory Bateson (1904-1980), influenced a number of American neuropsychiatrists and clinical psychologists.⁵⁷

⁵³ Sullivan, “A Note on the Implications of Psychiatry, the Study of Interpersonal Relations, for Investigations in the Social Sciences”; Harry Stack Sullivan, “Psychiatry: Introduction to the Study of Interpersonal Relations (part 1),” *Psychiatry: Journal for the Study of Interpersonal Processes* 1, no. 1 (February 1938): 121–34; Harry Stack Sullivan, *The Fusion of Psychiatry and Social Science*, 1st ed. (New York: Norton, 1964).

⁵⁴ Sullivan, *The Fusion of Psychiatry and Social Science*.

⁵⁵ Hale, *The Rise and Crisis of Psychoanalysis in the United States*, 177; Kvarnes, “The Washington School of Psychiatry--Past Dreams and Present Realities,” 296.

⁵⁶ Prigatano and Weinstein, “Edwin A. Weinstein’s Contributions to Neuropsychological Rehabilitation,” 306.

⁵⁷ Hale, *The Rise and Crisis of Psychoanalysis in the United States*; Gerald N. Grob, “World War II and American Psychiatry,” *Psychohistory Review* 19 (1990): 41–69.

Part of the reason for the success of Sullivan's social-psychoanalytic psychiatry had to do with his proximity to the nation's capital when the United States entered World War II. In 1942, the United States Armed Services tasked Sullivan with devising the interview methods to screen for potential mental instability among all of country's candidate recruits. Out of fifteen million men interviewed, nearly two million or 12% were declared unfit for service based on Sullivan's notoriously open-ended questions.⁵⁸ Although the military leaders did not conceal their disappointment with Sullivan's rate of rejection, they hoped that at least such screening process would limit the number of "neuropsychiatric casualties."

In the end, their hopes were dashed. The number of war-related psychoneuroses proved to be 11% higher in World War II than World War I.⁵⁹ Many suspected that Sullivan's screening success was "oversold," while others complained about the inadequate training of combat psychiatrists.⁶⁰ As for the latter, Major Edwin Weinstein, Chief of the Neuropsychiatric Unit of the United States Fifth Army, was in a position to shed some light from his own personal experiences in the war.⁶¹

⁵⁸ Hale, *The Rise and Crisis of Psychoanalysis in the United States*, 188; Grob, "World War II and American Psychiatry," 60–61; Leon Eisenberg and Laurence B Guttmacher, "Were We All Asleep at the Switch? A Personal Reminiscence of Psychiatry from 1940 to 2010," *Acta Psychiatrica Scandinavica* 122, no. 2 (August 1, 2010): 95.

⁵⁹ Hale, *The Rise and Crisis of Psychoanalysis in the United States*, 203.

⁶⁰ W.C. Menninger, "Psychiatric Experience in the War, 1941-1946," *American Journal of Psychiatry* 103, no. 5 (1947): 582.

⁶¹ Edwin A. Weinstein, "The Fifth U.S. Army Neuropsychiatric Center--'601st,'" in *Neuropsychiatry in World War II*, ed. Leonard D Heaton et al., vol. 2, 2 vols. (Washington, D.C: Office of the Surgeon General, Dept. of the Army, 1973), 127–41.

Almost as soon as the United States had soldiers on the ground in Tunisia, the trauma and shock of early bombardments and later combat engagement took a considerable toll on the mental resilience of troops. Up to 35% of all casualties in the North African campaign were neuropsychiatric.⁶² Weinstein, who was stationed in Tunisia and afterwards in Italy, recalled feeling helpless at the incoming flood of patients to the base hospital. He was far from alone. Brigadier General William Menninger (1899-1966) oversaw the training of all army neuropsychiatrists (as they were still called since the days of World War I) and admitted that he and his colleagues had virtually forgotten “all the lessons that we learned in the last war.”⁶³ Another psychiatrist similarly reflected that it was not until more than two years into the war that the U.S. Army achieved the “effective levels of operation” among its combat psychiatrists compared to those of the first world war.⁶⁴

What went wrong? In part, the blame shifted from Sullivan to Menninger, who failed to appreciate the aetiology of combat neuroses. Instead of “internal psychic factors” and the individual patient’s psychodynamic idiosyncrasies, so-called “combat fatigue” depended much more on situational and social stresses.⁶⁵ That was in part, however, why Sullivan’s screening was unsuccessful. He tried to tailor it to specific “types” of individuals, not taking into account some of the implications of his own

⁶² Hale, *The Rise and Crisis of Psychoanalysis in the United States*, 189.

⁶³ Menninger, “Psychiatric Experience in the War, 1941-1946,” 583.

⁶⁴ Albert J. Glass et al., eds., *Neuropsychiatry in World War II*, vol. 2 (Washington, D.C: Office of the Surgeon General, Dept. of the Army, 1973), xviii.

⁶⁵ Grob, “World War II and American Psychiatry,” 65; F.R. Hanson, “The Factor of Fatigue in the Neuroses of Combat,” *Bulletin of the United States Army Medical Department* 9 (November 1949): 147–50.

teaching with Sapir, namely that *anyone* could break down with “combat exhaustion” if “group ties” and “interpersonal relations” were sufficiently strained.⁶⁶ The aetiological emphasis slowly shifted, therefore, from what Weinstein later characterized as “principles of individual, psychobiologically based psychodynamics” to those of “social psychiatry” with a better understanding of the “combat society.”⁶⁷

One of the most conspicuous signs of such a shift was in the creation of “forward centers.” Instead of relying on base section hospitals, which required withdrawing the soldier often hundreds of miles from his combat unit, forward centers allowed psychiatrists to see patients for quicker assessments and without disrupting what they believed were salubrious social ties with the patient’s comrades.⁶⁸ Weinstein later referred to a certain “base hospital syndrome,” in which a patient’s condition appeared to worsen the farther they were removed and longer they stayed away from their unit.⁶⁹ It was “not surprising,” Weinstein maintained, that “many somatic symptoms develop” because such patients were left feeling guilty and remorseful for leaving their comrades. It was “clear,” he continued, why such a patient was “reluctant to talk about his battle experiences” because he was further “divorced from the ordering principles of his way of life.”⁷⁰ The farther patients were removed the more “inaccessible” they became.

⁶⁶ Grob, “World War II and American Psychiatry,” 52, 61; Menninger, “Psychiatric Experience in the War, 1941-1946,” 580–581.

⁶⁷ Weinstein, “The Fifth U.S. Army Neuropsychiatric Center--‘601st,’” 127, 132.

⁶⁸ Hale, *The Rise and Crisis of Psychoanalysis in the United States*, 192.

⁶⁹ Weinstein, “The Fifth U.S. Army Neuropsychiatric Center--‘601st,’” 133.

⁷⁰ *Ibid.*

However, forward centers were not implemented until the end of 1943.⁷¹ In the meantime, Weinstein and his fellow neuropsychiatrists desperately searched for other methods to “access” their patients.

One of the earliest methods was the use of barbiturates like sodium amytal and sodium pentothal in combination with the type of interviewing techniques taught by Harry Stack Sullivan, based on his understanding of psychoanalysis. The neurologist and psychoanalyst Roy Grinker (1900-1990) and his former student, the psychiatrist John Spiegel (1911-1991), were among the first in the United States Army to administer such “barbiturate interviews” in the hopes of aiding soldiers to recall traumatic experiences from combat. Grinker and Spiegel dubbed it “narcosynthesis,” or alternately “narcoanalysis,” with the implication that the drugs might accelerate the effects of psychoanalysis and its goal of “synthesis” or the re-integration of formerly repressed memories of trauma.⁷²

However, Weinstein remained skeptical. He and others wondered whether the barbiturate injections more often “aggravated rather than ameliorated symptoms.”⁷³ The so-called “truth drugs” or “truth serum” did not so much help soldiers to “recover” forgotten memories as compel them to “confabulate” fictional episodes that might

⁷¹ Ibid., 127.

⁷² Hale, *The Rise and Crisis of Psychoanalysis in the United States*, 193; Roy R. Grinker, *War Neuroses in North Africa: The Tunisian Campaign, January-May 1943* (New York: Prepared and distributed for the Air Surgeon, Army Air Forces, by the Josiah Macy, Jr. Foundation, 1943); Roy Richard Grinker and John Paul Spiegel, *Men under Stress* (McGraw-Hill, 1963); Harrington, *The Cure Within*, 152–153; Alison Winter, *Memory: Fragments of a Modern History* (University of Chicago Press, 2012), 58–63.

⁷³ Weinstein, “The Fifth U.S. Army Neuropsychiatric Center--‘601st,’” 137–138.

resemble a real event. One of the more refractory disorders that exposed this active confabulation was called “Old Sergeant’s syndrome.” The direct antithesis of malingering, soldiers with this syndrome denied anything wrong with them, even when they were conspicuously injured, for instance, no longer able to walk.⁷⁴ According to Weinstein, the injury was demoralizing: “He was now a patient, not a soldier. He had exchanged his fighting clothes for a pair of pajamas.”⁷⁵ Even after these “rusty iron men”⁷⁶ finally admitted their infirmity, they still refused to comply with interviews from the medical staff. But if Weinstein gave them an injection of sodium amytal, then they would often revert to *denying* their injury all over again.⁷⁷ It was as if the drug, Weinstein mused, “altered the milieu of brain function,” such that the latent “denial personality” re-emerged and compelled patients to “fill in” with confabulated excuses for the infirmity they did not want to acknowledge.

⁷⁴ R. Sobel, “The Old Sergeant Syndrome,” *Psychiatry* 10, no. 3 (August 1947): 315–21; R. Sobel, “Anxiety-Depressive Reactions After Prolonged Combat Experience, the Old Sergeant Syndrome,” *Bulletin of the United States Army Medical Department* 9 (November 1949): 137–46; Edwin A. Weinstein and Calvin S. Drayer, “A Dynamic Approach to the Problem of Combat-Induced Anxiety. Combat Psychiatry: Experiences in the North African and Mediterranean Theaters of Operation, American Ground Forces, World War II,” ed. Frederick R. Hanson, *The Bulletin of the U.S. Army Medical Department* 9 (November 1949): 12–25; Weinstein, “The Fifth U.S. Army Neuropsychiatric Center--‘601st,’” 133–134.

⁷⁵ Weinstein and Drayer, “A Dynamic Approach to the Problem of Combat-Induced Anxiety. Combat Psychiatry: Experiences in the North African and Mediterranean Theaters of Operation, American Ground Forces, World War II,” 14–15.

⁷⁶ “Medicine: Old Sergeant Syndrome,” *Time*, February 13, 1950.

⁷⁷ Edwin A. Weinstein et al., “The Diagnostic Use of Amobarbital Sodium (‘Amytal Sodium’) in Brain Disease,” *The American Journal of Psychiatry* 109 (1953): 889–94; Weinstein and Malitz, “Changes in Symbolic Expression with Amytal Sodium”; Edwin A. Weinstein et al., “Serial Administration of the Amytal Test for Brain Disease: Its Diagnostic and Prognostic Value,” *Archives of Neurology and Psychiatry* 71 (1954): 217–26.

The success of so-called “narcoanalysis” was mixed, therefore. In fact, the success of wartime neuropsychiatric practices in general, including the implementation of forward centers and combat rotation, was similarly disputed. Although some historians of psychiatry, such as Gerald Grob, identify the war as a “watershed for American psychiatry,” what Grob credits as its main influence, that is, the combat psychiatrists “successfully treating soldiers manifesting psychiatric symptoms,” may be called into question.⁷⁸ Another historian of psychiatry, Andrew Scull, argues that this “success” was rather engineered on the part of leading psychiatrists like William Menninger who “put their own spin on what they had accomplished.”⁷⁹ Like Grinker and Spiegel, they tried to leverage psychiatry’s success by affiliation with their “confrères” in internal medicine, which Menninger recognized as “one of the richest benefits of the war” for civilian psychiatry.⁸⁰ And so, apart from the questionable therapeutic success during the war, psychiatry’s professional success *after* the war had more to do with spin-doctors like Menninger, according to Scull. But perhaps the perception of success was not only actively conjured but also passively assumed through the persistent though elusive rubric, “neuropsychiatry.” In the next and final section, we briefly consider the contested history of this hybrid term in relation to Weinstein’s career in the middle of the twentieth century.

⁷⁸ Grob, “The Ambivalent Character of American Psychiatry,” 516; Grob, “World War II and American Psychiatry,” 46; W. P. Wilson, C. E. Wells, and P. J. Irigaray, “Should Psychiatry and Neurology Integrate?,” *American Journal of Psychiatry* 128, no. 5 (1971): 619.

⁷⁹ Andrew Scull, “The Mental Health Sector and the Social Sciences in Post-World War II USA. Part 1: Total War and Its Aftermath,” *History of Psychiatry* 22, no. 1 (March 1, 2011): 8.

⁸⁰ Menninger, “Psychiatric Experience in the War, 1941-1946,” 582.

The Genus of Neuropsychiatry

After the war, Weinstein returned to Mount Sinai Hospital as an “attending associate neurologist” from 1947 until 1956. During this period, he also served as a “consultant neuropsychiatrist” to both the Walter Reed Medical Center and the National Institutes of Health in Washington, D.C. In later years, he additionally called himself a “psychiatrist.” It is reasonable to ask, therefore, exactly which was he, a neurologist, a psychiatrist, or both, a “neuropsychiatrist”?

In fact, the term “neuropsychiatrist” did not necessarily signify that one was both a neurologist and psychiatrist.⁸¹ Originally, it was a designation used only in the military in an effort to present a united front and overcome professional rivalry during the First World War.⁸² At the beginning of the twentieth century, Swiss émigré and professor of psychiatry at Johns Hopkins University, Adolf Meyer (1866-1950), claimed to have invented the term based on what he later called his “pious wish” to see neurologists and psychiatrists “pool their domains,” partly in the mold of German-speaking traditions from the late nineteenth century, but partly also out of a “holistic” vision of medicine, not unlike that espoused by Kurt Goldstein.⁸³ Meyer wrote in 1922,

⁸¹ “Neuroanatomy means neurological anatomy; neurophysiology means neurological physiology. Does neuropsychiatry mean neurological psychiatry? I think not.” Z. M. Lebensohn, “Neurology and Psychiatry: Separable or Inseparable?,” *The Medical Annals of the District of Columbia* 34, no. 11 (1965): 515.

⁸² Pressman, *Last Resort*, 23.

⁸³ Walter Freeman, Franklin G. Ebaugh, and David A. Boyd, “The Founding of the American Board of Psychiatry and Neurology, Inc.,” *American Journal of Psychiatry* 115, no. 9 (1959): quoted on 773; Glass et al., *Neuropsychiatry in World War II*, 2:97–98; J. Ramsay Hunt, “The Domain of Neuropsychiatry and the Training of the Neuropsychiatrist,” *Archives of Neurology and Psychiatry* 31, no. 5 (May 1, 1934): 1081.

We want neuropsychiatrists—not merely neurologists and not merely psychologists, but primarily physicians able to study the entire organism and its functions and behavior and more especially the share of the nervous system and of the general problems of adaptation.⁸⁴

According to the “much lamented”⁸⁵ American neurologist Pearce Bailey (1902-1976), Meyer’s wish for neuropsychiatry eventually lost momentum and was only resuscitated once the United States entered World War II.⁸⁶

The official “death knell” of neuropsychiatry came in 1933 when a group of neurologists and psychiatrists gathered in New York to discuss the creation of a joint-board of examiners responsible for certifying specialists in neurology and psychiatry.⁸⁷ For the next year and a half, they debated a range of issues, almost all of which pointed back to one decisive question: should the Board *require* every student to specialize in *both* neurology and psychiatry, as Adolf Meyer envisioned, or should it allow them the *choice* of specialty in *either* neurology or psychiatry. Meyer, though chair of the committee, was outnumbered. As some delegates later reflected, “their union and mutual collaboration were on the basis of declaring a difference between the

⁸⁴ Adolf Meyer, “Inter-Relations of the Domain of Neuropsychiatry,” *Archives of Neurology & Psychiatry* 8, no. 2 (1922): 119.

⁸⁵ *Ibid.*, 111.

⁸⁶ Pearce Bailey, “National Institute of Neurological Diseases and Blindness: Origins, Founding and Early Years (1950-1959),” in *The Nervous System: A Three-Volume Work Commemorating the 25th Anniversary of the National Institute of Neurological and Communicative Disorders and Stroke*, ed. Donald Bayley Tower (New York: Raven Press, 1975), xxii.

⁸⁷ Marc H. Hollender, *The American Board of Psychiatry and Neurology: The First Fifty Years* (Deerfield, IL: American Board of Psychiatry and Neurology, 1991); Freeman, Ebaugh, and Boyd, “The Founding of the American Board of Psychiatry and Neurology, Inc.”

specialties.”⁸⁸ After a year and a half of deliberation, the board finally agreed to unite on the basis of that difference and formed the “American Board of Psychiatry and Neurology.”⁸⁹ On June 7, 1935, they held the first certifying meeting at which they granted twenty-two certificates in psychiatry and neurology together and twelve in psychiatry alone. Weinstein graduated that year and therefore would have been among the first generation of board-certified dual specialists in “neurology and psychiatry”—not “neuropsychiatry.”⁹⁰

Was Weinstein a “neuropsychiatrist”? In the sense that he was certified as both a neurologist and psychiatrist, yes. But also by virtue of having served in the military during the war, he became a neuropsychiatrist *de facto*. As Bailey pointed out, with notable chagrin, the war gave new wings to neuropsychiatry. When psychiatrist William Menninger assumed leadership of the Division of Neurology and Psychiatry in the United States Army, he changed its official name to the “Division of Neuropsychiatry,” dating back to the *de facto* precedent under Colonels Thomas Salmon (1876-1927) and Pearce Bailey, Sr. (1865-1922) in the first world war. However, the neuropsychiatry was not the same. Thanks to Meyer in Baltimore and his counterpart in Washington, William

⁸⁸ Freeman, Ebaugh, and Boyd, “The Founding of the American Board of Psychiatry and Neurology, Inc.,” 774.

⁸⁹ Even the name was a wedge issue. “There was cautious agreement on each point, with the neurologists and psychiatrists searching for the hidden implication of each proposal. Dr. Casamajor found himself unable to understand the alphabetic idiocy of the psychiatrists who wished to name the organization ‘Psychiatry and Neurology.’ There was dispute concerning which should take precedence: the alphabet or the numerical constituency.” *Ibid.*, 775.

⁹⁰ By 1938, Stevens calculates there were 1,656 physicians who practiced neurology and psychiatry as a “combined specialty.” She does not indicate who among them were board-certified. Rosemary Stevens, *American Medicine and the Public Interest: A History of Specialization* (1971; repr., Berkeley: University of California Press, 1998), 223.

Alanson White (1870-1937), the two helped steer the next generation of leaders like Menninger and Harry Stack Sullivan toward an “eclectic” embrace of both somatic and psychoanalytic forms of treatment as well as both neurological and psychiatric training.⁹¹ Thus, combat psychiatrists like Weinstein were encouraged to try whatever methods seemed to work, whether it was “narcoanalysis,” the “bonds of comradeship,” or simply “hot food and clean clothes.”⁹² It was less the plurality of treatment that rankled neurologists like Bailey, Jr. and more the manner of training that he viewed as ultimately subversive of neurology’s “prestige and authority.”⁹³ The “neuropsychiatric movement,” he complained, “did more to set neurology back in its bid for professional autonomy than any other single development in its history.”⁹⁴

To set matters straight, Bailey, who was chief of the Veterans Administration’s Neuropsychiatric Division, changed its name in 1947 to the “Psychiatry and Neurology Service,” which he justified as “giving to the neurology program greater exposure and enhancing its prestige.”⁹⁵ In addition, by the time the draft bill for a “National

⁹¹ Although Sullivan was an outspoken critic of many somatic techniques, especially Freeman's lobotomy procedure, he himself practiced a somatic type of therapy, using alcohol to induce in patients "continuous mild intoxication" for up to a week at a time. Hale, *The Rise and Crisis of Psychoanalysis in the United States*, 176.

⁹² Grob, “World War II and American Psychiatry,” quoted on 58–59.

⁹³ Bailey, “National Institute of Neurological Diseases and Blindness: Origins, Founding and Early Years (1950-1959),” xxiv.

⁹⁴ The irony was that Bailey’s father, as Chief of Psychiatry under the U.S. Surgeon General, oversaw the creation of the so-called “neuropsychiatry movement” when he became Colonel of the Division of Neurology and Psychiatry in the U.S. Army Medical Department during WWI. Pearce Bailey, “The Past, Present and Future of Neurology in the United States,” *Neurology* 1, no. 1 (February 1951): 5.

⁹⁵ Bailey, “National Institute of Neurological Diseases and Blindness: Origins, Founding and Early Years (1950-1959),” xxiii.

Neuropsychiatric Institute” was signed in July 1946, Congress had changed its name to the National Institute of Mental Health, later established in 1949.⁹⁶ A year later, President Truman signed into law the establishment of the National Institute of Neurological Diseases and Blindness, of which Bailey became its first director in 1951. And by the end of the decade, the *Archives of Neurology and Psychiatry* split into the *Archives of Neurology* and the *Archives of Psychiatry*.⁹⁷ Over the course of the 1950s, Derek Denny-Brown, the British-American neurologist at Harvard Medical School, wryly observed, “the genus ‘neuropsychiatrist’ gradually became scarce.”⁹⁸ Edwin Weinstein, at the height of his career and the peak of his research on the denial of illness, belonged to a dying breed.⁹⁹

In the decades following Weinstein and Kahn’s publication of *Denial of Illness*, their research attracted growing attention, though less frequently of the flattering kind.

⁹⁶ J. L. Brand, “Neurology and Psychiatry,” *The Education of American Physicians*, Ed. RL Numbers 226 (1980): 243.

⁹⁷ Gerald N. Grob, “The Attack of Psychiatric Legitimacy in the 1960s: Rhetoric and Reality,” *Journal of the History of the Behavioral Sciences* 47, no. 4 (Fall 2011): 410–411.

⁹⁸ Derek Denny-Brown, “The First Hundred Years: The Centennial of the American Neurological Association,” *Archives of Neurology* 32, no. 5 (May 1, 1975): 279.

⁹⁹ Historian of medicine Nancy Tomes is right to point out that the characteristic blurring between the identities of neurologist, psychiatrist, and neuropsychiatrist made it “much harder to maintain a clean story line,” especially when writing about a twentieth-century physician like Edwin Weinstein. From the perspective of others, like Richard Noll, the label “neuropsychiatrist” served as a “blanket term” to conceal the awkward fact that neurologists and psychiatrists simply did not know where to draw their lines of demarcation or how to settle their “jurisdictional disputes.” These disputes, Noll suggests, “largely ceased” after proper board-certified licensing procedures were established in 1934. However, as the next chapter will elaborate, they were far from over. The question of what to call, or where to “put,” someone like Weinstein may profitably be “put” back on ourselves. The need to name, in our words not theirs, potentially exposes the discrepancies and limits between our attempts to understand and the doctors’ own. In some ways, the categories “somatic” and “dynamic,” or “organicist” and “psychoanalytic,” break down in Weinstein’s discussions of the denial of illness. Tomes, “Beyond the ‘Two Psychiatries,’” 776; Richard Noll, *American Madness: The Rise and Fall of Dementia Praecox* (Cambridge: Harvard University Press, 2011), 17.

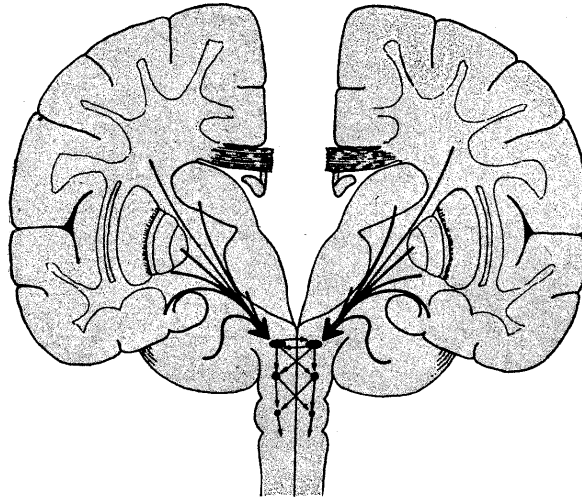
In fact, despite hundreds of citations of their work, their hybrid symbolic and physiological interpretation was more often pilloried than praised. Not only, then, did Weinstein belong to an increasingly marginalized medical specialty, but he also came to represent, albeit in certain caricatured form, the extremes of eclectic psychoanalytic theory applied to neurological disease. Moreover, according to this account, he was blamed for having led future researchers of anosognosia astray by downgrading the importance of anatomical studies in inflated favor of personality factors.¹⁰⁰ Not until the mid-1970s, when a number of new neurologists rose to leadership, did the reign of cerebral localization theory make a resurgence in the United States. This fable of revival through the stark shift from eclectic neuropsychiatry to “behavioral neurology” is the subject of the next and final chapter.

¹⁰⁰ Prigatano, *Advances in the Study of Anosognosia*, 8.

Chapter 5

**From Denial to Disconnection:
Norman Geschwind and the Rise of Behavioral Neurology**

"It is hard not to say 'the patient' and yet it is clear that this terminology is misleading."¹



2

On March 2, 1961, a forty-one year-old police officer entered the Boston Veterans Administration Hospital, complaining of headaches and nausea. The headaches usually lasted for several hours, and pain was especially intense over his left eye. The patient's wife pointed out that she and other family members thought he had been acting strange for some time. He often appeared indifferent and apathetic, confusing dates and people, which led to tensions at work. During the initial exam, the patient, designated "P.J.K.," looked alert and exhibited no disorientation, but he often repeated

¹ Geschwind, "Disconnexion Syndromes in Animals and Man. II," 637.

² Figure 1. The "cerebral prongs" of the right and left hemispheres in the experimentally-severed animal brain. Roger W. Sperry, "Cerebral Organization and Behavior," *Science* 133, no. 3466 (1961): 1754.

the questions posed to him as if he did not understand them. According to the examiners, the neurologist Norman Geschwind (1926-1984) and psychologist Edith Kaplan (1924-2009), he “exhibited inappropriate jocularity against a background of general apathy” and displayed “no insight into his illness,” appearing generally “unconcerned about it.”³

Two weeks later, P.J.K. was given an arteriogram which indicated a mass in the left frontal area of his brain. Soon afterwards, he underwent a craniotomy operation, during which a large part of his left frontal lobe was “amputated” along with a tumor later identified as a glioblastoma. Following the surgery, P.J.K. was noticeably weak on his right side, but over the next few months this gradually improved. His trouble with language persisted, however. On May 22, 1961, Kaplan noticed that he could not write with his left hand. He was, after all, right-handed, but his difficulty with the non-dominant left hand was so severe that he could not even type his own name. When he was shown his errors, he was “quite astonished” though unable to correct them.⁴

The image shows a handwritten transcription of the phrase "To come early was impossible." written with the left hand. The handwriting is highly stylized and difficult to read, with many loops and flourishes. The text is written in black ink on a white background.

Figure 2. P.J.K.’s transcription with his left hand of the phrase, “To come early was impossible.”⁵

Geschwind and Kaplan decided to test P.J.K.’s left hand abilities further with a new series of tests, specifically designed to assess the functional differences between

³ Geschwind and Kaplan, “A Human Cerebral Deconnection Syndrome,” 676.

⁴ Ibid., 678.

⁵ Ibid., 679.

the right and left cerebral hemispheres of the brain. They had him close his eyes and then placed an object in his left hand. Next, they asked him to gesture silently how he would use the object, and then to describe its function in his own words. When they put a hammer in his left hand, he made the correct hammering motion, but then said, “I would use this to comb my hair with it.”⁶ When they gave him a key, he turned it in mid-air as if unlocking a door, then said he would use it for “erasing a blackboard.”⁷ With a pair of scissors, he made the proper cutting motion and said, “I’d use that to light a cigarette with.”⁸ No matter what object he held, he could no longer verbally describe its function—not until he opened his eyes.

After several more months in the hospital, Geschwind noted that while P.J.K. was “oriented in all spheres,” he “still had little insight into his illness.”⁹ The illness Geschwind and Kaplan named, “a human anterior disconnection syndrome,” the first of its kind.¹⁰ They believed P.J.K. suffered from “callosal lesions,” or discrete damage in the corpus callosum, a cerebral structure that connected the right and left hemispheres.¹¹ As a result of these lesions, activity in each hemisphere was “disconnected” from the

⁶ Ibid., 678.

⁷ Ibid.

⁸ Ibid.

⁹ Ibid., 677.

¹⁰ This was the initial name they gave it in their first presentation to the Boston Society of Psychiatry and Neurology on December 14, 1961. When they published the report in *Neurology* on October 1, 1962, they omitted “anterior.” In later years, Geschwind alternately referred to the “disconnection” as “disconnexion” and “disconnection.” Geschwind and Kaplan, “A Human Cerebral Disconnection Syndrome”; Geschwind, “Disconnexion Syndromes in Animals and Man. I.”; Norman Geschwind, *Selected Papers on Language and the Brain* (Dordrecht ; Boston: Reidel, 1974), 13.

¹¹ Geschwind and Kaplan, “A Human Cerebral Disconnection Syndrome,” 684.

other.¹² That was why P.J.K. could not verbally identify objects held in his left hand. As long as his eyes were closed, the tactile sensations from his hand never reached his language-dominant left cerebral hemisphere. He made the correct motions with the objects because he was still able to recognize them via intact connections with his contralateral, non-dominant, right cerebral hemisphere. But he could no longer articulate this tacit, tactile form of recognition because his dominant, left cerebral hemisphere was effectively “cut off” from the sensory signals coming from the left side of his body.¹³ As a result, he behaved as if he had “2 nearly isolated half-brains, functioning independently.”¹⁴ His perceptual and linguistic capacities were functionally “split,” making him do one thing and say another. On September 4, 1961, he was discharged from the hospital, having “improved markedly.” Nine months later, though, at age forty-two, he was dead.¹⁵

This chapter analyzes the impact of Norman Geschwind's theory of “disconnection syndromes” with respect to the history of anosognosia. Broadly, it tracks Geschwind's career as an American neurologist in the 1960s and 1970s to the subsequent creation of a subspecialty he called “behavioral neurology.” In particular, it shows how Geschwind's theory of disconnection syndromes starkly diverged from his

¹² Ibid., 682.

¹³ Geschwind, “Disconnexion Syndromes in Animals and Man. I,” 276.

¹⁴ Geschwind and Kaplan, “A Human Cerebral Deconnection Syndrome,” 675.

¹⁵ Anonymous, “Random Reports: Human Split-Brain Syndromes,” 1013; Geschwind, “Random Reports.”

predecessor Edwin Weinstein's idea of the "denial personality," replacing the emphasis on the patient's perspective with a renewed interest in the localization of brain lesions. Then, with this contrast in mind, the chapter explores neurology's shifting allegiances, away from social psychiatry and toward cognitive psychology, precisely at a time when psychiatrists were under increasing social criticism. Finally, I argue that Geschwind ultimately sought to undermine the legacy of holistic medical theorists like Kurt Goldstein by resorting to a strong version of revisionist history bent on reclaiming for behavioral neurology the so-called "classical" theorists of localizationism.

Split-Brain Syndromes

The idea of P.J.K.'s "disconnection syndrome" did not originate with Geschwind and Kaplan. In 1953, physiologists Roger Sperry (1913-1994) and Ronald Myers of the University of Chicago surgically resected the corpus callosum and optic chiasm in a number of research animals to test for its role in learning. With both connecting structures destroyed, there was no way for information to pass between the animal's cerebral hemispheres. Sperry and Myers proceeded to train the animals in tasks involving the visual discrimination of objects, with one of its eyes covered. After they switched the cover to the animal's other eye, it appeared as if the animal had never learned the task. The "untrained" eye no longer received any input from the side of the brain corresponding to the "trained" eye. As a result, Sperry and Myers argued that the

corpus callosum played a major role in the “transfer of learning” between the cerebral hemispheres.¹⁶

Their argument, conspicuously in favor of cerebral localization, overturned the received wisdom of Sperry’s former mentor at Harvard, Karl Lashley, whose theory of equipotentiality mandated explicitly against the idea of any one cerebral structure possessing unique, specialized function. Undeterred, however, Sperry and his team, later at the California Institute of Technology, continued to generate evidence from their “split-brain” research on animals in support of the theory that the corpus callosum did, in fact, play a special role in higher mental functions, namely, coordinating those functions between the right and left cerebral hemispheres. In 1961, Sperry wrote a brief article in *Science*, entitled, “Cerebral Organization and Behavior.”¹⁷ He argued that as a result of severing the corpus callosum, “each of the twin half brains,” or cerebral hemispheres, became, “in a sense, pretty much a whole brain.”¹⁸ Each half, therefore, functioned as a new whole. Consequently, Sperry speculated, “In these respects it is as if the animals had two separate brains.”¹⁹ Each hemisphere appeared to be “unaware of what is experienced in the other.”²⁰ Each possessed its own “independent mental

¹⁶ R. E. Myers and R. W. Sperry, “Interocular Transfer of a Visual Form Discrimination Habit in Cats after Section of the Optic Chiasm and Corpus Callosum,” *Anatomical Record* 115, no. 35 (1953): 70–71; R. E. Myers and R. W. Sperry, “Interhemispheric Communication through the Corpus Callosum: Mnemonic Carry-over between the Hemispheres,” *Archives of Neurology and Psychiatry* 80, no. 3 (1958): 298; Malcolm Piercy, “The Effects of Cerebral Lesions on Intellectual Function: A Review of Current Research Trends,” *The British Journal of Psychiatry* 110, no. 466 (1964): 324.

¹⁷ Sperry, “Cerebral Organization and Behavior.”

¹⁸ *Ibid.*, 1752–1753.

¹⁹ *Ibid.*, 1749.

sphere or cognitive system,” representing “qualitatively different cerebral prongs.”²¹

What remained to be seen, though, was whether Sperry’s findings applied to humans as well. That was Geschwind and Kaplan’s contribution.

Since Sperry and Myers’ paper in 1953, Geschwind later recalled that the “tide of interest in the callosum began to turn.”²² By the time P.J.K. entered the V.A. hospital in Boston in March of 1961, both Geschwind and Kaplan were aware of Sperry’s continuing animal experiments at Caltech. In fact, they wrote, “It was Sperry’s work which alerted us to the possibility of disconnection syndromes in man.”²³ However, Sperry’s work did more than “alert” them. They adopted his more speculative streak as well. Where Sperry theorized, “[I]t is as if the animals had two separate brains,” Geschwind and Kaplan similarly suspected of P.J.K., “[H]e behaved as if his two cerebral hemispheres were functioning nearly autonomously.”²⁴ The critical difference between Geschwind’s patient and Sperry’s experiment was that P.J.K.’s corpus callosum was never surgically altered or resected as Sperry had done to his research animals. Thus, Geschwind and Kaplan lacked any anatomical evidence of the functional “disconnection.” This may have been the reason they imitated Sperry’s suggestive speculation, since they could not replicate his experiment.

²⁰ Ibid.

²¹ Ibid., 1749, 1752.

²² Geschwind, “Disconnexion Syndromes in Animals and Man. I,” 240.

²³ Geschwind and Kaplan, “A Human Cerebral Disconnection Syndrome,” 675.

²⁴ Ibid., 683.

After P.J.K.'s death, Geschwind and Kaplan briefly described the findings from his autopsy in a footnote. Significantly, the corpus callosum did not reveal any visible lesions, although near the anterior region it was "markedly thinned."²⁵ Thus, there was no clear-cut evidence of any functional "deconnection," although to some commentators, P.J.K.'s symptoms were "sufficiently concordant" with Sperry's animal research to "compel attention."²⁶ However, more cases were needed for further testing. Not long after the death of P.J.K., on February 6, 1962, two neurosurgeons, Joseph Bogen and Philip Vogel, from Loma Linda University in Los Angeles, operated on a patient named "W.J." who suffered from severe epilepsy.²⁷ They surgically severed his corpus callosum and the anterior commissure connecting the right and left cerebral hemispheres in the hope of eliminating, or at least minimizing, his seizures. After W.J. recovered from the commissurotomy, one of Sperry's graduate students, Michael Gazzaniga, conducted a battery of psychological tests on him, the results of which closely matched many of Geschwind and Kaplan's observations of P.J.K.²⁸

²⁵ Ibid., 684.

²⁶ Piercy, "The Effects of Cerebral Lesions on Intellectual Function," 318.

²⁷ J. E. Bogen and P. J. Vogel, "Cerebral Commissurotomy in Man," *Bulletin of the Los Angeles Neurological Society* 27, no. 4 (1962): 169–72.

²⁸ M.S. Gazzaniga, J.E. Bogen, and R.W. Sperry, "Some Functional Effects of Sectioning the Cerebral Commissures in Man," *Proceedings of the National Academy of Sciences of the United States of America* 48, no. 10 (1962): 1765; M.S. Gazzaniga, J.E. Bogen, and R.W. Sperry, "Laterality Effects in Somesthesia Following Cerebral Commissurotomy in Man," *Neuropsychologia* 1, no. 3 (1963): 209–15; M.S. Gazzaniga, J.E. Bogen, and R.W. Sperry, "Observations on Visual Perception after Disconnexion of the Cerebral Hemispheres in Man," *Brain: A Journal of Neurology* 88, no. 2 (June 1965): 221–36.

In response to Sperry and Gazzaniga's stimulating research as well as his own work with Kaplan, Geschwind published a seminal two-part article in *Brain* in 1965, unveiling an elaborate new theoretical explanation of nearly all the disorders of higher mental functions, including the aphasias, agnosias, and apraxias, based on what he called, in the paper's title, "Disconnexion Syndromes in Animals and Man."²⁹ According to many contemporaries, Geschwind's "DSAM" represented a "renaissance of anatomical reasoning in neuropsychology."³⁰ It "revived [Theodor] Meynert's attention to anatomy and corticocortical connections" and "reinvented disconnectionism."³¹ Although some dismissed Geschwind's lengthy paper as a mere literature review, he intended for his discussion of historical case literature to help reform the current neurology of higher mental functions, or "behavioral neurology." Because Geschwind's article was so important to neurologists for the next two decades, it will help to review some of his basic claims before proceeding to examine how he applied the theory specifically to the case of anosognosia.

²⁹ As early as 1961, Geschwind began brooding over a general theory of higher mental disorders. While at a conference in New York that year, the senior British neuropsychologist Oliver Zangwill patiently listened while the eager Geschwind expounded on his initial ideas about disconnection. At the end, Zangwill invited Geschwind to submit his theory in a paper to the journal *Brain*. It would be a some years until Geschwind was finally ready, but in the meantime he pored over nineteenth-century case literature as well as the scientific papers issuing from Sperry's lab. By the time he was finished, the paper had to be published in two parts for its uncommon length, not to mention its ranging breadth, from animal physiology to clinical neurology to the philosophy of mind. Geschwind, "Disconnexion Syndromes in Animals and Man. I"; Geschwind, "Disconnexion Syndromes in Animals and Man. II."

³⁰ Klaus Poeck, "Modern Trends in Neuropsychology," in *Brain & Behavior Research in Clinical Neuropsychology*, ed. Arthur Lester Benton (New Brunswick, NJ: AldineTransaction, 1969), 8.

³¹ Orrin Devinsky, "Disconnexion Syndromes," in *Behavioral Neurology and the Legacy of Norman Geschwind*, ed. S.C. Schachter and Orrin Devinsky (Philadelphia: Lippincott-Raven, 1997), 117; Absher and Benson, "Disconnection Syndromes," 862.

The key to understanding disconnection syndromes and their effect on higher mental functions was to understand the anatomical structures of the brain. First and foremost, Geschwind deferred to “Flechsigs Rule,” or the teaching of German anatomist and psychiatrist Paul Flechsig that all the primary sensory areas in the cortex send and receive signals via short bundles of fibers called “association pathways.”³² The collection of these pathways make up “association areas,” and these areas are what communicate between the primary sensory area and several other regions in the cortex through “cortico-cortical” or “transcortical” connections. When lesions appear along these fiber tracts, sometimes it leads to the disruption, if not outright “disconnection,” of the cortico-cortical pathways, resulting in what Geschwind called “disconnexion syndromes,” which he equated to the “‘transcortical’ or ‘conduction’ syndromes of older authors [Carl Wernicke].”³³ This may have been partly the reason some readers confused Geschwind's paper with a literature review. One colleague joked, “So, Norman, you discovered that neurons have axons. What's new?”³⁴ The anatomical description of transcortical pathways and association areas was not new, but Geschwind's use of the historical case literature in combination with Sperry's split-brain research was.

³² Actually, this “rule” appears to have first derived from Theodor Meynert, not Flechsig. It is unclear why Geschwind chose to credit the Leipzig doctor over the Vienna professor.

³³ Geschwind, “Disconnexion Syndromes in Animals and Man. I,” 243.

³⁴ Marco Catani and Marsel Mesulam, “What Is a Disconnection Syndrome?,” *Cortex* 44, no. 8 (September 2008): quoted on 911.

Geschwind always had a special interest in languages (he was said to speak eight³⁵), and language offered him the first clue that P.J.K. suffered a form of “cerebral disconnection” or “split-brain syndrome.”³⁶ Unlike Sperry and Myers’ “callosum-sectioned” animals, one could interview human patients and have them try to describe their experiences. Based on verbal testimony, particularly when it contradicted the patient’s non-verbal behavior, one could reasonably infer a disconnect, not only between the person’s words and deeds, but anatomically, between the association areas and speech areas located in the left cerebral hemisphere. The belief that the function for speech was located in the left, and therefore “dominant,” cerebral hemisphere dominated the brain sciences for over a century.³⁷ But the idea that speech functions could be cut off from the right, “non-dominant,” cerebral hemisphere owed partly to Geschwind’s clever combination of the nineteenth-century theorists like Meynert and Wernicke with the new experimental findings from Sperry’s group. Ultimately, Geschwind ascribed a wide range of mental disorders sheerly to the fact that patients lacked the anatomical structures to connect their experiences accumulated in one half of the brain to the language confined in the other half. As a result, the left half

³⁵ S.C. Schachter and O. Devinsky, *Behavioral Neurology and the Legacy of Norman Geschwind* (Lippincott-Raven, 1997), xiv.

³⁶ Geschwind preferred the term “disconnection syndrome” but Sperry used the term “‘split-brain’ syndrome.” R. W. Sperry, M. S. Gazzaniga, and J. E. Bogen, “Interhemispheric Relationships: The Neocortical Commissures; Syndromes of Hemisphere Disconnection,” *Handbook of Clinical Neurology* 4 (1969): 273.

³⁷ Harrington, *Medicine, Mind, and the Double Brain*, passim.

was left to guessing in the dark as to what went on in the right half, while the right half was silently sequestered in a “curious form of psychic imprisonment.”³⁸

What may have looked like a problem of perception was more fundamentally a problem of communication and connections in the brain. It may have seemed that P.J.K. did not recognize the hammer in his left hand because of what he said: “I would use this to comb my hair with it.” According to Geschwind, however, this was merely a “confabulatory response” generated by one half of the brain, the language-dominant left hemisphere. Without any information to work with, because the sensations in his left hand only went as far as his reticent right hemisphere, P.J.K. was forced to resort to a “verbal filling in” or “confabulatory completion.”³⁹ Of course, it also depended on what “recognition” actually meant in patients with a disconnection syndrome like P.J.K.. Even though he was unable to identify the hammer verbally, he still seemed to recognize it by making the correct hammering motions. The question remained, did he or did he not recognize the objects in his hand? According to Geschwind, the question, “Did the patient recognize?” was flawed. In cases of disconnection syndromes, the question was no longer about the patient: “The ‘patient’ who speaks to you is not the

³⁸ Norman Geschwind, “Wings: A Neurologist at the Theater,” *New England Journal of Medicine* 300, no. 10 (1979): 570.

³⁹ The term “confabulatory completion” was a modification of the so-called “completion effect” in perception, which was first described by Walther Poppelreuter in 1917 and which Oliver Zangwill, one of Geschwind’s mentors, applied to the study of anosognosia as a perceptual filling-in of body-awareness in 1963. Geschwind, “Disconnexion Syndromes in Animals and Man. II,” 590, 600; Walther Poppelreuter, *Die psychischen Schädigungen durch Kopfschuss im Kriege 1914/17: Die Herabsetzung der körperlichen Leistungsfähigkeit und des Arbeitswillens durch Hirnverletzung im Vergleich zu Normalen und Psychogenen*, vol. 2 (Leipzig: Leopold Voss, 1917); Oliver L. Zangwill, “The Completion Effect in Hemianopia and Its Relation to Anosognosia,” in *Problems of Dynamic Neurology, an International Volume: Studies on the Higher Functions of the Human Nervous System*, ed. Lipman Halpern (Jerusalem: Jerusalem Post Press, 1963), 274–82.

‘patient’ who is perceiving—they are, in fact, separate.”⁴⁰ Whatever the patient said, or his left hemisphere “said,” was important only in so far as it revealed that the patient himself did not really know what he was saying. He was merely covering up, or confabulating, for his lack of awareness.

Of all the types of disconnection syndromes, Geschwind believed, anosognosia took this confabulation to “an even more extreme.”⁴¹ A patient who denied blindness might protest that the light was poor or that he needed glasses, but his attempt to make such excuses only drew more attention to his lack of sight and insight. Indeed, if there was any lesson from disconnection syndromes that Geschwind stressed above all, it was the fact that “the patient’s ‘explanation’ of his disability” was never to be relied upon with any regularity.⁴² Rather than “giving us any very useful insight into his illness,” he observed, “we do not attempt to take seriously this patient’s protestations.”⁴³ So-called “introspections,” or the patient’s self-reports of his experiences, were basically useless and “ineffective”⁴⁴:

One of the most important implications is that the “introspections” of the patient as to his disability may be of little or no use to the examiner. The patient cannot “introspect” about the activities of a piece of brain which has no connexion to the speech area. What he tells you is of little value in elucidating the mechanism and may indeed be actively misleading.⁴⁵

⁴⁰ Geschwind, “Disconnexion Syndromes in Animals and Man. II,” 590.

⁴¹ *Ibid.*

⁴² *Ibid.*, 591.

⁴³ *Ibid.*, 590–591.

⁴⁴ *Ibid.*, 638.

⁴⁵ *Ibid.*, 590.

Geschwind was not speaking of anosognosia alone. By the end of the “DSAM,” he pivoted from a critique of introspection to launch an even larger critique of, what he labeled in subheadings, “The Unity of Consciousness” and “The Whole Man.” But before taking up a discussion of these broad themes, it will help first to explore what led Geschwind to them, specifically by focusing on his early engagement with Edwin Weinstein.

The Borderlands between Neurology and Psychiatry

Because of Weinstein’s hybrid status as one of the few remaining neuropsychiatrists in the 1960s, his emphatically “symbolic-physiological” or “personal-cultural” approach was frequently conflated with psychiatry—and conveniently so. According to one recent appraisal by the psychologist George Prigatano, Weinstein “moved the field of the study of anosognosia out of neurology into psychiatry,” causing “a loss of interest in studying [its] neuropsychological basis.”⁴⁶ However, it was never that decisive or abrupt. Even Geschwind chided fellow neurologists at the time for such a “naïve misinterpretation” of Weinstein’s work that assumed he was “advancing a purely psychiatric explanation of the syndromes of denial.”⁴⁷ Nevertheless, such “misinterpretation” had its functions. It helped to re-position Weinstein’s research in the history of anosognosia as an aberration and intrusion by a psychiatrist rather than a continuation and contribution from a fellow neurologist. Geschwind knew better than

⁴⁶ Prigatano, “Historical Observations Relevant to the Study of Anosognosia,” 8.

⁴⁷ Geschwind, *Selected Papers on Language and the Brain*, 73.

to re-interpret Weinstein's legacy that way. Still, ironically, it was Geschwind's reinterpretation of anosognosia and his "friendly argument" with Weinstein in the early 1960s which eventually led to such a revisionist history of Weinstein and others who had studied anosognosia.

In December 1962, at the annual conference in New York of the Association for Research in Nervous and Mental Disease, Geschwind, as chief of neurology at the Boston V.A. hospital, was invited by Weinstein and David McK. Rioch, the chairman of the program committee, to speak on aphasia.⁴⁸ With his colleague Davis Howes, a professor of neuropsychology at the Boston University School of Medicine, Geschwind began the presentation, "Quantitative Studies of Aphasic Language," by underlining the difference between their approach and that of Weinstein.⁴⁹ Although Geschwind later admitted that he "leaned heavily on the work of Weinstein,"⁵⁰ in the presentation with Howes, they stressed that the patient's language did not necessarily represent some "personally meaningful code" as Weinstein claimed,⁵¹ but rather a "random disturbance" equivalent to a "mathematical form" and amenable to a "probability distribution."⁵² In other words, the patient's words did not need to be combed for symbolic

⁴⁸ Rioch was the director of the Division of Neuropsychiatry at the Walter Reed Army Institute of Research. Robert Galambos, "Robert Galambos," in *The History of Neuroscience in Autobiography*, ed. Larry R. Squire (Oxford: Oxford University Press, 2008), 193.

⁴⁹ Howes and Geschwind, "Quantitative Studies of Aphasic Language."

⁵⁰ Geschwind, "Disconnexion Syndromes in Animals and Man. II," 597n1.

⁵¹ Edwin A. Weinstein and N.J.A. Keller, "Linguistic Patterns of Misnaming in Brain Injury," *Neuropsychologia* 1, no. 2 (August 1963): 83.

⁵² Howes and Geschwind, "Quantitative Studies of Aphasic Language," 229, 233.

meaning to understand her condition. Her words could simply be counted and

categorized based on their frequency. Geschwind and Howes explained:

Our approach differs in some respects from those usually followed. We have ignored questions about the meaning of the aphasic patient's utterances, feeling that an objective analysis of meaning is not within the reach of present techniques and is better deferred until we understand the actual behavior that constitutes language.⁵³

Whereas Weinstein reiterated that patients employed “metaphorical language” to preserve their “stereotyped cultural role” according to “preexisting cultural values,”⁵⁴ Geschwind and Howes “deferred” such interpretative approach in favor of a more quantitative and “objective analysis.” Significantly, the difference between Weinstein and Geschwind involved more than the study of aphasic disorders. It represented a widening fissure between Weinstein’s version of “symbolic neurology” and Geschwind’s brand of “behavioral neurology.”

At the eighty-eighth annual meeting of the American Neurological Association in Atlantic City, on the morning of June 12, 1963, Weinstein and colleagues presented on “Anosognosia and Aphasia.”⁵⁵ Geschwind was also there and was asked to prepare a response. Weinstein, now chief of psychiatric research at Walter Reed Medical Center and a faculty member at the Washington School of Psychiatry, opened with his familiar argument that the anosognosic patient’s denial of illness was a symbolic expression and

⁵³ Ibid., 229.

⁵⁴ Edwin A. Weinstein, “Affections of Speech with Lesions of the Nondominant Hemisphere,” in *Disorders of Communication. Proceedings of the Association. December 7 and 8, 1962, New York, NY*, ed. David McK Rioch and Edwin A. Weinstein (Williams & Wilkins, 1964), 224–225.

⁵⁵ Weinstein, Cole, and Mitchell, “Anosognosia and Aphasia.”

admission of it. The reason cases of anosognosia rarely coincided with cases of aphasia was not because, as commonly assumed, anosognosia resulted from right-hemispheric lesions, while aphasia resulted from left-hemispheric lesions. Anosognosia, Weinstein argued, was “not a matter of right brain versus left brain, per se.”⁵⁶ Instead, the reason for its higher correlation with right-hemispheric lesions was because patients with left-sided lesions usually suffered some form of aphasia, rendering them unable to express their denial in any explicit verbal manner. In effect, the aphasia “silenced” the anosognosia.

Then came Geschwind’s chance to reply. He began modestly by confessing that he was astonished to learn of Weinstein’s cases because he had never observed such behavior among his own patients. With polite deference, he added, “I hope to go back to my patients again to see whether I can confirm Dr. Weinstein’s findings.”⁵⁷ But Geschwind did not stop there. He intimated that Weinstein risked reading too much into his patients’ words and actions, even though he kept his critique of this senior colleague aimed carefully wide of his target:

The problem of anosognosia is a very complicated one. The reason, I think, is that, like any part of neurology which overlaps with psychiatry, it is likely to bring out emotional responses in both neurologists and psychiatrists...We must be very careful not to make the error of either being hyperpsychiatric and interpreting all of these phenomena in terms of the patient’s attempt to deal with the illness, or of being hyperneurological and trying to make the denial in the man with the myocardial infarction also result from a coincident and unexpected lesion in some denial center of the brain.⁵⁸

⁵⁶ *Ibid.*, 173; Harrington, *Medicine, Mind, and the Double Brain*, 274–275.

⁵⁷ Weinstein, Cole, and Mitchell, “Anosognosia and Aphasia,” Geschwind on 174.

⁵⁸ *Ibid.*

With faintly concealed sarcasm, Geschwind warned against diagnosing patients with anosognosia who merely disagreed with their doctors and sought a second opinion. He criticized such interpretation because it made normal behavior into a brain disease. At the other extreme, he cautioned against twisting the patient's words into a cryptic communication of some personal plea. While the effects of brain disease may appear meaningful and motivated, in fact, it remained merely random. Sometimes, brain disease was just brain disease.⁵⁹ Ideally for Geschwind, psychiatrists needed to keep their "emotional responses" in check to avoid overinterpretation, and consequently, misdiagnosis.

Weinstein, however, never eliminated the possibility of anosognosia even in someone who denied his doctor's diagnosis of heart disease. He also rebutted Geschwind by arguing that anosognosic denial was "not dependent on a structural brain lesion."⁶⁰ Even a patient with heart disease and without conspicuous brain disease could exhibit signs of anosognosia. No matter what the condition, Weinstein believed, even normal language contained important "symbolic and physiological aspects," which could be parsed for "personally meaningful" clues. Thus, Weinstein sounded quite like the kind of doctor Geschwind ridiculed at each extreme, both the "hyperneurological" and "hyperpsychiatric." The difference was that for Weinstein these were not extremes.

⁵⁹ See also, Geschwind, "Wings: A Neurologist at the Theater."

⁶⁰ Weinstein, Cole, and Mitchell, "Anosognosia and Aphasia," 175.

There was nothing “hyper-” about either point of view, at least, not according to his version of neuropsychiatry.

Geschwind’s formulation of the “hyperpsychiatric” and “hyperneurological” extremes resonated, however, for many American neurologists, more so than Weinstein’s hybrid version. That was in large part because neurology and psychiatry continued to drift farther and farther apart after the Second World War making their differences appear to the other as extreme and excessive. In this respect, Geschwind’s and Weinstein’s contrasting views of anosognosia accentuated a larger debate about the appropriate zones of expertise divided between neurology and psychiatry. And that debate, in part, pointed back to each doctor’s professional experience and early training.

Like Weinstein, Geschwind served in World War II, but not as a neuropsychiatrist. Instead, he acted as an army interpreter for German prisoners of war.⁶¹ During his two years of service, he, like Weinstein, grew interested in certain types of combat behavior, especially when soldiers seemed to “forget their own safety” by attacking the enemy even when their commanding officers ordered them to “stay low.”⁶² Similar to the “Old Sergeant’s syndrome,” Geschwind was intrigued by the social psychology of self-sacrifice and self-denial. After the war, he returned to Harvard College where he concentrated in psychology under Karl Lashley. Thinking he wanted to

⁶¹ Orrin Devinsky, “Norman Geschwind: Influence on His Career and Comments on His Course on the Neurology of Behavior,” *Epilepsy and Behavior* 15, no. 4 (2009): 414.

⁶² Antonio R. Damasio and Albert M. Galaburda, “Norman Geschwind,” *Archives of Neurology* 42, no. 5 (May 1985): 500.

become a psychiatrist, he enrolled at Harvard Medical School in 1947. Although he later described himself as having initially “no strong interest in organic medicine,” he changed his mind after taking a course in neuroanatomy, where he first learned about the study of epilepsy and aphasia. Because psychiatry was “overwhelmingly concerned with psychotherapy” and psychology textbooks “overwhelmingly quoted such authors as Kurt Goldstein, who appeared to have argued very convincingly that there was little or no significant localization in the brain,”⁶³ Geschwind eventually shifted his interest from psychiatry to neurology. In 1955, when Weinstein and Kahn published *Denial of Illness*, Geschwind became chief resident in neurology at the Boston City Hospital under British-American neurologist Derek Denny-Brown.

Meanwhile in the early 1950s, Denny-Brown, also acting as professor of neurology at Harvard Medical School, tirelessly promoted the British model of neurology as an autonomous specialty, independent from psychiatry. Contra his colleague Karl Lashley’s vision of “coalescence” between neurology and psychiatry,⁶⁴ Denny-Brown worked with Pearce Bailey, the director of the new National Institute of Neurological Disease and Blindness, to align “neurologic medicine” more closely with internal medicine throughout the United States. With the “cry ‘back to the medical wards and autopsy room,’” Denny-Brown helped lead young neurologists like

⁶³ Devinsky, “Norman Geschwind,” Geschwind quoted on 414.

⁶⁴ Karl S. Lashley, “Coalescence of Neurology and Psychology,” *Proceedings of the American Philosophical Society* 84, no. 4 (1941): 462.

Geschwind “back” to neuroanatomy, rather than neuropsychiatry.⁶⁵ And so, a year after joining Denny-Brown’s clinical staff, Geschwind took up a two-year position as research associate at the Massachusetts Institute of Technology, studying the physiology of the squid axon under Francis Schmitt, one of the very first to use the term “neurosciences” when he founded the Neurosciences Research Program in 1962.⁶⁶

Psychiatrists, for their part, became increasingly worried that they were misunderstood and misrepresented, especially among the younger generations of doctors and medical students. In 1958, a study appeared in the *Journal of Medical Education* analyzing medical students’ “personality factors” in their attitudes toward the “whole man approach.” The author, an academic psychiatrist in Philadelphia, quoted students who in their interviews called psychiatry, “a B.S.er’s paradise...full of couch doctors fleecing the public.”⁶⁷ Such students, according to the survey, were consistently more resistant to “person-oriented” approaches and less interested in course offerings in the social sciences. Furthermore, they were more likely to display “authoritarian personalities” and were “less able than others to take a psychiatrically oriented view of people.”⁶⁸ In response, the study proposed increasing the number of courses offered in

⁶⁵ D.E. Denny-Brown, “The Changing Pattern of Neurologic Medicine,” *New England Journal of Medicine* 246, no. 22 (1952): 840; Joel A. Vilensky, Sid Gilman, and Pandey R. Sinish, “Denny-Brown, Boston City Hospital, and the History of American Neurology,” *Perspectives in Biology and Medicine* 47, no. 4 (2004): 514.

⁶⁶ Francis Otto Schmitt and Norman Geschwind, “The Axon Surface.,” *Progress in Biophysics and Biophysical Chemistry* 8 (1957): 165–215; Francis Otto Schmitt et al., eds., *The Neurosciences, Paths of Discovery* (Cambridge, Mass: MIT Press, 1975).

⁶⁷ Seymour Parker, “Personality Factors among Medical Students as Related to Their Predisposition to View the Patient as a ‘Whole Man.’,” *Journal of Medical Education* 33 (1958): 741.

⁶⁸ *Ibid.*

“dynamic psychiatry” so as to underline how illness always related to the “wider context of the patient’s life” as well as to demonstrate that psychiatry was in “an active and continuous relationship with the other clinical disciplines.”⁶⁹ However, this sort of increased exposure of psychiatry in medical curricula was precisely what many senior neurologists opposed.

Soon they struck back, hard. Two neurologists, one from the College of Physicians and Surgeons at Columbia University and the other from the Neurological Institute of Presbyterian Hospital in New York, published an article with the title, “Psychiatric Symptoms Masking Brain Tumor.”⁷⁰ They documented over a hundred cases of misdiagnosis of brain tumors because the consulting psychiatrist failed to understand the symptoms. In some cases, the misdiagnosis proved fatal. Patients were treated to decades of psychotherapy and electric shock treatment for so-called “psychiatric symptoms,” some of which included depression, but all of which, according to the authors, were “masking” brain tumors. They wrote, not masking their contempt:

There is a pathetic, poignant ineffectiveness about doing psychotherapy in the hope of exorcising an expanding brain tumor. We have become so enchanted with emotional factors in the production of symptoms that we sometimes forget organic components.⁷¹

The burden of blame decisively shifted. Instead of the prospective neurologists’ “authoritarian personality,” it was the feckless, inept psychiatrists who failed their

⁶⁹ Ibid., 744.

⁷⁰ J. L. Pool and J. W. Correll, “Psychiatric Symptoms Masking Brain Tumor.,” *The Journal of the Medical Society of New Jersey* 55, no. 1 (1958): 4–9.

⁷¹ Ibid., 4; D. Frank Benson and Norman Geschwind, “Psychiatric Conditions Associated with Focal Lesions of the Central Nervous System,” *American Handbook of Psychiatry* 4 (1975): quoted on 235.

hapless patients with untreated brain tumors. Neurologists raised the stakes, vehemently opposing those psychiatrists who wanted to see more “person-oriented” clinical instruction in American medical schools. Clearly, the so-called “whole man approach” was not wholly trusted.

When in 1965 Geschwind wrote the monograph on disconnection syndromes that was said to have “launched” his career as well as “behavioral neurology as a neurological specialty,”⁷² he, too, concluded with a critique of the so-called “whole man.” “For the past forty years,” he wrote, “there have been schools of thought which have stressed the importance of thinking of the patient as a whole.”⁷³ This approach, he cautioned, was “actively misleading.”⁷⁴ Recounting his study with Edith Kaplan of the case of P.J.K., he pointed out the “many confusions” that arose while trying to treat him “as a whole” instead of “the exact opposite...to regard the patient as made of connected parts rather than as an indissoluble whole.”⁷⁵ Correlatively, he suggested that such non-holistic point of view might productively be applied to not only patients but healthy people as well:

It should be pointed out that the usefulness of sometimes considering animals or humans not as a unit but as a union of loosely joined wholes need not apply only to disease states although probably it will find its greatest use in that situation. Probably even in the normal person parts of the brain are so weakly connected as to make their interaction difficult...Perhaps an adult man is more

⁷² Damasio and Galaburda, “Norman Geschwind,” 502; Devinsky, “Disconnexion Syndromes,” 115.

⁷³ Geschwind, “Disconnexion Syndromes in Animals and Man. II,” 637.

⁷⁴ Ibid.

⁷⁵ Ibid.

unitary than a chimpanzee, but perhaps total unity is never obtainable because of the necessary separation of some structures.⁷⁶

In the end, Geschwind believed that the split-brain experiments by Sperry's team and his own clinical study of disconnection syndromes suggested that "there are better criteria of consciousness" and urged for "the necessity of re-evaluating the idea of the unity of consciousness if it is to be at all useful."⁷⁷ But his critique was not purely philosophical. He, too, was concerned that a preoccupation with treating the patient "as a whole" could lead to dire consequences, including fatalities from misdiagnosis.⁷⁸ And here again, his critique was not purely diagnosis-directed or nosological but also partly pedagogical and professional. It was a critical volley aimed at the very legitimacy of psychiatric expertise, a legitimacy which happened to be already broadly under attack by the mid-1960s.⁷⁹

"The dichotomy of medical disciplines is not oriented as much for patients as it is for physicians," wrote one American doctor concerned about the dearth of "talent" in the "borderlands between neurology and psychiatry."⁸⁰ Left abandoned and allegedly

⁷⁶ Ibid., 637–638.

⁷⁷ Ibid., 638.

⁷⁸ Norman Geschwind, "Non-Aphasic Disorders of Speech," *International Journal of Neurology* 4, no. 3 (1964): 207–208.

⁷⁹ Rosenberg, "The Crisis in Psychiatric Legitimacy," passim; Hale, *The Rise and Crisis of Psychoanalysis in the United States*, 302; Grob, "The Attack of Psychiatric Legitimacy in the 1960s."

⁸⁰ Aring, "Observations on Multiple Sclerosis and Conversion Hysteria," 673; Lebensohn, "Neurology and Psychiatry"; Wilson, Wells, and Irigaray, "Should Psychiatry and Neurology Integrate?," 619; Lipowski and Kiriakos, "Borderlands between Neurology and Psychiatry," 135; Geschwind, "The Borderland of Neurology and Psychiatry: Some Common Misconceptions," 2–3; Benson and Geschwind, "Psychiatric Conditions Associated with Focal Lesions of the Central Nervous System," 224; D. Frank Benson and Dietrich Blumer, eds., *Psychiatric Aspects of Neurologic Disease, Volume 2*, Seminars in Psychiatry (New

neglected in such “borderland,” or “no-man’s-land” as Geschwind liked to call it,⁸¹ were a number of “neurologically silent” but “psychiatrically noisy” disorders,⁸² one of the most exemplary of which was anosognosia. Yet despite its “noise” or psychiatric-seeming symptoms, Geschwind, like many of his colleagues, remained convinced that this particular tract of the “borderlands” belonged to neurology, specifically, the “neurology of behavior.”⁸³ Whereas earlier Weinstein argued that anosognosia represented a “bridge” of communication between neurology and psychiatry, indeed *neuropsychiatry*, Geschwind ultimately saw it as a “borderland” for the reclamation and demarcation of a new field he called “behavioral neurology.” How he managed to “launch” this new field had as much to do with Geschwind’s study of history as his prowess in neurology.

Historical Neurology and Neurological History

As soon as Geschwind joined the Neurology Service of the Boston V.A. Hospital, his director, Fred Quadfasel, a former student of Kurt Goldstein and Karl Bonhoeffer in Berlin, immediately encouraged him to study their historical case literature along with

York, N.Y: Grune & Stratton, 1982); B.H. Price, R.D. Adams, and J.T. Coyle, “Neurology and Psychiatry: Closing the Great Divide,” *Neurology* 54, no. 1 (January 11, 2000): 9–10.

⁸¹ Geschwind, “The Borderland of Neurology and Psychiatry: Some Common Misconceptions,” 1.

⁸² R. Hunter, “Psychiatry and Neurology. Psychosyndrome or Brain Disease.,” *Proceedings of the Royal Society of Medicine* 66, no. 4 (1973): 361.

⁸³ D F Benson, “The History of Behavioral Neurology,” *Neurologic Clinics* 11, no. 1 (February 1993): 1; D. Frank Benson, “Neuropsychiatry and Behavioral Neurology: Past, Present, and Future,” *The Journal of Neuropsychiatry and Clinical Neurosciences* 8, no. 3 (1996): 355; Wilson, Wells, and Irigaray, “Should Psychiatry and Neurology Integrate?,” 618; Devinsky, “Norman Geschwind,” 415.

the “great classical neurologists” of the nineteenth century.⁸⁴ Expecting to discover a number of errors in the presumably obsolete writings, Geschwind was shocked to find “new” insights about “classical localizationist teachings.”⁸⁵ As a result, he resolved to study the case literature more closely:

I therefore decided to study the ideas of the classical ‘localizationist’ school by reading their own writings rather than by reading the interpretations of later hostile authors...[and] decide for myself whether the repudiation of the classical views was indeed justified.⁸⁶

Among the so-called “classic cases” Geschwind studied was an article written by the French neurologist Jules Déjerine in 1892, which Quadfasel had given him. It was entitled, “Contribution to the Pathological- and Clinical-Anatomical Study of the Different Varieties of Word Blindness.”⁸⁷ After reading it, Geschwind recalled, “I awoke, perhaps belatedly, to my own profound confusion.”⁸⁸ What he uncovered, he believed, was one of the first case histories of a disconnection syndrome.

⁸⁴ Geschwind, *Selected Papers on Language and the Brain*, 1.

⁸⁵ Norman Geschwind, “Anatomy and the Higher Functions of the Brain,” *Boston Studies in the Philosophy of Science* 4 (1969): 108.

⁸⁶ Geschwind, *Selected Papers on Language and the Brain*, 1.

⁸⁷ Joseph Jules Déjerine, “Contribution à l’étude anatomo-pathologique et clinique des différentes variétés de cécité verbale,” *Mémoire de la Société de Biologie* 4 (1892): 61–90; D.N. Bub, M. Arguin, and A.R. Lecours, “Jules Dejerine and His Interpretation of Pure Alexia,” *Brain and Language* 45, no. 4 (November 1993): 531–59.

⁸⁸ Norman Geschwind, “The Paradoxical Position of Kurt Goldstein in the History of Aphasia,” *Cortex* 1 (1964): 215.

Déjerine himself described his patient's case as one of "pure alexia without agraphia."⁸⁹ Also known as "word-blindness," this version of alexia was uncommonly focal because the patient could still write despite not knowing how to read. That is to say, he could see words, but he was effectively "blind" to them *as words*, seeing only "curves, angles, [and] dots,"⁹⁰ unless he traced those curves and lines with his finger, much like Goldstein described Schneider's strategy of "reading."⁹¹ Déjerine accounted for this unusual disturbance by hypothesizing a disconnection between the visual centers in the occipital cortex from the language areas in the left cerebral hemisphere. The patient suffered from right hemianopia, which restricted the right side of his field of vision and suggested contralateral damage in his left occipital cortex. This meant he could only see out of the left side of his visual field, using the right occipital cortex. Somehow, though, the connection between the right and the left was also damaged, which explained why the patient could see the words but not see them *as words*. After the autopsy, Déjerine confirmed areas of damage in the left occipital cortex and concluded that this damage alone was severe enough to have affected the connection between the right occipital cortex and the left cerebral hemisphere.

⁸⁹ Déjerine, "Contribution à l'étude anatomo-pathologique et clinique des différentes variétés de cécité verbale"; Jules Déjerine, "Sur un cas de cécité verbale avec agraphie, suivi d'autopsie," *CR Société du Biologie* 43 (1891): 197–201.

⁹⁰ These were not Déjerine's words, but the American poet, Walt Whitman's, who died that year. Walt Whitman, "Song of the Rolling Earth," in *Leaves of Grass*, 150th anniversary ed (Oxford ; New York: Oxford University Press, 2005).

⁹¹ Goldstein and Gelb, "Psychologische Analysen hirnpathologischer Fälle auf Grund von Untersuchungen Hirnverletzter," 23.

Seventy years later, Geschwind saw things differently. While Déjerine mentioned partial damage to the patient's corpus callosum, he did not attach much significance to it. Geschwind, however, was convinced that, for pure alexia to occur, there had to be not only destruction of the left visual cortex but also damage to the corpus callosum.⁹² Only this would account for the "specialized disconnection" between the right visual cortex and the language areas of the left hemisphere. And only this would hamper the patient's recognition of words. Despite the fact that Déjerine downplayed the "callosal lesion," Geschwind declared it essential to pure word-blindness. He seized on what he considered the historic significance of Déjerine's paper, drawing connections between it and Sperry's latest "split-brain" experiments, and began to look for evidence of "callosal lesions" among his own patients. In a matter of just a few weeks, he chanced upon the case of P.J.K.⁹³

Geschwind did not stop there. His interest in the historical case literature only waxed along with his skepticism toward the received "'standard' history" and its depicted "Dark Ages" of nineteenth-century neurology.⁹⁴ He continued to read the "classical neurologists," such as Jean-Martin Charcot and Carl Wernicke,⁹⁵ but he

⁹² Norman Geschwind, "The Anatomy of Acquired Disorders of Reading," in *Reading Disability: Progress and Research Needs in Dyslexia*, ed. John Money (Baltimore: Johns Hopkins Press, 1962), 119–120; Geschwind, *Selected Papers on Language and the Brain*, 7–8.

⁹³ Geschwind, "Disconnexion Syndromes in Animals and Man. I," 241.

⁹⁴ Geschwind, "The Paradoxical Position of Kurt Goldstein in the History of Aphasia," 215.

⁹⁵ Norman Geschwind, "Carl Wernicke, the Breslau School and the History of Aphasia," in *Brain Function: Speech, Language, and Communication*, ed. E.C. Carterette, vol. 3 (Berkeley: University of California Press, 1966), 1–16; Norman Geschwind, "Wernicke's Contribution to the Study of Aphasia," *Cortex* 3 (1967): 449–63; Geschwind, "The Work and Influence of Wernicke."

puzzled most over the “paradoxical” writings of the German neurologist (and psychiatrist) Kurt Goldstein.⁹⁶ Although he focused mainly on a monograph Goldstein wrote in 1927, entitled, *Localization in the Cerebral Cortex: Toward an Understanding of the Sick*,⁹⁷ Geschwind believed that Goldstein’s ideas were grossly misinterpreted and misappropriated by American psychologists and anti-localizationists like Karl Lashley. Despite the “active suppression” of “true knowledge” by “holists” like Lashley,⁹⁸ Goldstein, according to Geschwind, was “in fact a very classical localizer,” and he sought to set the historical record straight.⁹⁹ Although Goldstein sometimes gave in to “holist” ideas, Geschwind decided that it was because he basically led “the life of an intellectual double agent,” concealing his presumed “true” localizationist sympathies.¹⁰⁰

Besides the critique that Geschwind did not read a substantial portion of Goldstein’s writing to appreciate the nuances in his interpretation of localization theory,¹⁰¹ why did it matter to him? Probably because re-reading (and re-writing) history

⁹⁶ Geschwind, “The Paradoxical Position of Kurt Goldstein in the History of Aphasia.”

⁹⁷ Kurt Goldstein, “Die Lokalisation in der Grosshirnrinde: nach den Erfahrungen am kranken Menschen,” in *Handbuch der normalen und pathologischen Physiologie*, ed. A. Bethe and G. von Bergmann, vol. 10 (Berlin: Springer, 1927).

⁹⁸ Norman Geschwind, “Neurological Knowledge and Complex Behaviors,” *Cognitive Science* 4, no. 2 (1980): 193; Norman Geschwind, “The Neglect of Advances in the Neurology of Behavior,” in *The Encyclopaedia of Medical Ignorance: Exploring the Frontiers of Medical Knowledge*, 1984, 14.

⁹⁹ Norman Geschwind, “Disorders of Attention: A Frontier in Neuropsychology,” *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences* 298, no. 1089 (1982): 183.

¹⁰⁰ Geschwind, “The Neglect of Advances in the Neurology of Behavior,” 13.

¹⁰¹ Historian of science David Ludwig qualifies Goldstein’s holism-cum-localizationism “weak localization” (what I prefer to call “reformed localizationism”): “While it is true that Goldstein accepts correlations between aphasic symptoms and circumscribed brain areas, the main point of his holism is not what neural correlates can be found, but how these correlates have to be understood.” Ludwig, “Language and Human

offered him a way to reassert the growing independence of American neurology by “rediscovering” its venerable past out from “under the cloud...of psychiatry.”¹⁰²

Notwithstanding the fact that many of the “great neurologists” whose work Geschwind presumed to rehabilitate were also psychiatrists, their work represented to him a “useful past” to narrate a story of “revival” and mythical “resurrection” of the “golden age of neurology.”¹⁰³

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What Geschwind identified as the “hyperneurological” and “hyperpsychiatric” perspectives struck a nerve that ran deeper than different interpretations of anosognosia. His debate with Weinstein reflected the basic question of how doctors viewed their patients, that is, before trying either to diagnose or treat them, how to regard them and represent them, as bodies with damaged brains or individuals with idiosyncratic troubles. The debate between Geschwind and Weinstein was more than a reflection of the deepening division between neurology and psychiatry. It was a debate over the diagnostic relevance of the patient's experience. And it is a debate that echoes to the present.

Nature: Kurt Goldstein’s Neurolinguistic Foundation of a Holistic Philosophy,” 47–48. Goldstein wrote, “Analysis of the defects in lesions of this part does not at all allow us to relate a circumscribed defect to a circumscribed lesion, still less to localise a definite performance in a definite group of cells, etc.” Kurt Goldstein, “Remarks on Localisation,” *Confinia Neurologica* 7, no. 1–2 (1946): 26.

¹⁰² Aura Edward Severinghaus, “A Medical Discipline Takes Stock,” *Archives of Neurology* 17, no. 5 (1967): 463; Schachter and Devinsky, *Behavioral Neurology and the Legacy of Norman Geschwind*, xiv–xv.

¹⁰³ A. M. Galaburda, “Norman Geschwind 1926–1984,” *Neuropsychologia* 23, no. 3 (1985): 299; Caine and Joynt, “Neuropsychiatry . . . Again,” 326; V.S Ramachandran, “Behavioral Neurology: Reviving the Golden Age,” *Journal of the Neurological Sciences* 285, Supplement 1, no. 0 (October 2009): S3. Casper, Stephen T. “A Revisionist History of American Neurology.” *Brain* 133, no. 2 (February 1, 2010): 638–642.

Epilogue

Insight Out:

Re-thinking the Perception of an Illness of Perception

"The state of health is a state of unawareness."¹

This dissertation originated from the belief that there is a larger history of the denial of illness that stretches beyond the diagnosis itself. I argued that this history can be used like a special lens to examine in detail the formation and transformation of areas of medicine today called neurology and psychiatry. It can also be used to magnify the tightly woven mesh of ideas, both medical and philosophical, about the nature of consciousness and the meaning of illness. Above all, by tunneling into its historical particularities, I have tried to open up a new channel in the social and intellectual history of medicine.

Beginning in the middle of the nineteenth century with the debates on scientific materialism, I traced the emergence of some of the earliest formulations of the denial of illness, tying it to localization theory and the organic aetiology of mental illness. From there, I explored a series of key transitions in the study of what became known as "anosognosia" in the early decades of the twentieth century, especially after the First World War. Then, I tracked it across the Atlantic through the forced emigration of a select group of European psychiatrists and neurologists to the United States, where I studied the impact of theories of culture and personality on "neuropsychiatry" during and after the Second World War. Finally, I examined the increasingly strained relations

¹ Canguilhem, *The Normal and the Pathological*, 91–92.

between American neurologists and psychiatrists in the 1960s by focusing on the critique of personality-oriented approaches in favor of the definition of anosognosia as a “split-brain syndrome.”

Along the way, the discussion turned to questions about the division of medical expertise, particularly of neurology and psychiatry. I showed that their differences were never self-evident or stable. What was called “neurological” and what “psychiatric” depended on time and place, whether it was in Vienna or Paris at the end of the nineteenth century or whether it was in the United States during the 1930s or the 1980s. Both “when” and “where” made a difference in what neurology and psychiatry signified. These designations mattered because what doctors chose to call themselves and how they organized specialties helped determine how they diagnosed their patients and even *who* became their patients.

Medical specialists carved out nosological distinctions between diseases thought to involve the mind and/or the brain, but in doing so, they exposed “borderlands” which sprawled outside the designated boundaries. Even if the number of patients who straddled the borders, or fell between their cracks, was relatively small, the number was not all that mattered. The very nature of anosognosia, as one of the so-called “borderland disorders,” specially magnified the difficulties inherent in trying to rationalize and categorize behavior on the basis of either brain anomaly or personal idiosyncrasy—as if it could be so definitively confined to one or the other.

Regardless of the potential mismatch between the individual’s experience of illness and the doctor’s presumption of its absence, anosognosia the concept has

endured. Throughout the fitful expansion and contraction of neurology and psychiatry, it has continuously forced physicians to reflect on the definitions of health and disease in light of the limits of self-awareness.

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What is health? French neurosurgeon René Leriche once wrote, it is “life lived in the silence of the organs.”² Years later, German philosopher Hans-Georg Gadamer added, health is “that miraculous capacity we have to forget ourselves.”³ Silence and forgetting, of body and self. These definitions are largely negative. They emphasize absence. Health is the lack of feeling sick, the lack of disruption in one's body, the lack of self-awareness. *Or is it?*

Descriptions of anosognosia often sound like this version of health. It, too, entails a sort of self-forgetfulness, an organic silence, and a lack of feeling limitations. What is the difference, then? Where is the boundary and who draws it between health and illness? What does it have to do with experience and the other boundary, between consciousness and the unconscious?

One of the aims of this dissertation was to begin answering such questions, not in any absolute sense but in an *historical* one. That means I have tried to underline the instability of every answer to the definition of health and the limits of consciousness. I have tried to illuminate how these very definitions were shaped by physicians, whose careers often did not track with preconceived borders distinguishing “neurology” and

² Quoted in Canguilhem, *The Normal and the Pathological*, 243.

³ Gadamer, H. G. *The Enigma of Health*, 96.

“psychiatry.” Instead, their work sometimes had the effect of re-drawing such borders.

Treated separately, it would be a daunting task to undertake writing either a history of “illness” or a history of “awareness,” but that was never my intention. Instead, I sought to illuminate a unique point at which their histories converged, where one became unintelligible without the other, even in its absence.

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