Neuropsychodynamic Psychiatry

Heinz Boeker
Peter Hartwich
Georg Northoff
Editors

Springer

Neuropsychodynamic Psychiatry

Heinz Boeker • Peter Hartwich
Georg Northoff
Editors

Neuropsychodynamic Psychiatry

Springer

Editors
Heinz Boeker
Psychiatric University Hospital Zurich
Zurich
Switzerland

Georg Northoff
Mind, Brain Imaging, and Neuroethics
Institute of Mental Health Research
University of Ottawa
Ottawa
ON, Canada

Peter Hartwich
Hospital of Psychiatry-Psychotherapy-Psychosomatic
General Hospital Frankfurt
Teaching Hospital of the University
Frankfurt
Germany

ISBN 978-3-319-75111-5 ISBN 978-3-319-75112-2 (eBook)
https://doi.org/10.1007/978-3-319-75112-2

Library of Congress Control Number: 2018948668

This Springer imprint is published by the registered company Springer Nature Switzerland AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Preface

When reading the title of our book, *Neuropsychodynamic Psychiatry*, the reader may well feel curious, but also somewhat surprised: Is this simply another attempt to reduce complex phenomena like subjective experience, psychopathology, and psychodynamics to neuronal activation patterns and dysfunctions?

One of this book's key issues is in fact to encourage and promote the critical discussion surrounding neuroscientific knowledge in the context of psychiatry, psychotherapy, and particularly of psychodynamic psychiatry. We particularly wanted to address that psychodynamic models and mechanisms can well be integrated with neurobiological approaches without reducing the former to the latter. For that, as we claim, one needs to take a slightly different and novel look on both psychodynamics and neurobiology though. One needs to consider psychodynamic processes in the larger social context in concrete relation to the world—psychodynamic mechanisms like defense mechanisms are psychosocial configurations of our relation to the world. At the same time, one needs to free the brain from its encapsulation within the skull and put it into the context of the world—neuroscience thus needs to extend beyond the brain to the world (of which the body is part). This points out the world as common reference for both psychodynamic mechanisms and brain (Northoff 2011)—that, in turn, makes possible to easily integrate psychodynamics and neurobiology and to thereby account for the complexities one can see in daily clinical reality.

The book focuses on mental functions such as the self, the ego, the conscious and the unconscious, and the psychological mechanisms and functions underlying these. The self in psychiatric patients is considered in a relational perspective, and the relational connecting processes between the brain and the environment are examined. The empirical plausibility of this relational definition of the self and the brain presents a particular challenge.

As neurobiological scientific knowledge on psychiatric illness grows, numerous therapeutic implications will certainly come into play. In view of this, fundamental, epistemologically oriented research, for instance, whether evidence-based research can give adequate answers to important questions in clinical practice, should always be kept in mind.

Considering how neurobiological knowledge can potentially benefit psychiatry and psychotherapy (for instance, a stronger position among the general public and in government healthcare policies, breaking down stigmas and taboos about mental

illness, a better understanding of the etiology involved, and of how physiological and mental aspects of psychiatric disorders are linked, aiming at neurobiological variables in psychotherapeutic treatment, a better understanding of the interaction between various brain regions, and developing selective rules for indications using neurobiological predictors), the limits and possible risks that may be involved in neuroscientific approaches and, in the worst case, their one-dimensional application should not be overlooked. A neurobiological view of mental disorders and their treatment can always become problematic, as Fuchs (2006) puts it, if neurobiological approaches are no longer considered an alternative and complement to psychological models and procedures, but claim to have the absolute sovereignty of interpretation in human science.

Recent studies have shown a relationship between such reductionistic biological interpretations of psychiatric illness and the empathy shown by the clinician concerned: for example, Lebowitz and Ahn (2014) reported on unintentional negative consequences of exclusively biological models of illness depending on how the therapeutical relationship was built, on how the symptoms were perceived, interpreted, and dealt with, and also particularly depending on the therapist's empathy.

Psychiatrists, psychotherapists, and patients encounter one another as individual subjects or persons. The brain, however, is an "object." This obvious difference has considerable implications, in both a conceptual and an empirical perspective. Bennet and Hacker (2003) warn, for instance, about confusing individual subjects with their respective brains, as this would not consider the fundamental difference between persons and objects. Neuronal processes and mechanisms concern the brain and can be viewed as a necessary, but not sufficient condition for psychiatric treatment, and psychotherapy in particular, to be effective. This is because there are also other factors, such as interpersonal constellations and the cultural environment, that have to be considered. On the other hand, the effectiveness of psychiatric therapy and psychotherapy has to do with the personal level, which is implicitly related to the brain, but ultimately may not be identified with it in a conceptual perspective. Considering all these points, this book faces the particular challenge of trying to bridge the gap between the principal differences of a person's individual level and the brain's general level. One of the greatest methodological challenges of the future will be to develop experimental designs and analytical methods, which will make it possible to link individual and general features on a neuronal level. "First-person science" may thus be defined as a methodological strategy endeavoring to systematically link subjective experience with the observation of neuronal states (from a third-person perspective) (Northoff et al. 2006; Boeker et al. 2013). Like this, first-person neuroscience is different from general neuroscience, which is based on the observation of neuronal states, more or less independently of subjective experience. The complex task of investigating neuronal effects of psychiatric treatments, and especially those of psychotherapy, reflects in an almost paradigmatical way the complexity of the brain, resulting in greater insight and a better understanding of the general principles of neuronal organization (Boeker and Northoff 2010).

This book's chapters have been written by internationally renowned clinicians and researchers in the fields of psychiatry, psychotherapy, psychoanalysis, neuroscience, and other related disciplines. It is their contributions that have made it possible to adequately consider the broad spectrum of clinical-psychiatric, psychotherapeutic, psychoanalytical, and neuroscientific perspectives. Paradigms and methodological procedures in neuroscientific studies and results from fundamental research, as well as questions relevant for clinicians concerning the limited application of neuroscientific findings in clinical practice, are considered. This is presented within the broader framework of historical, epistemological, and philosophical discussions. This book has partly resulted from numerous discussions over many years with various professionals in psychiatric clinics and institutions. On behalf of everyone involved in these discussions, we would like to thank our colleagues from the "Therapy and Process Research" group at the University Hospital of Psychiatry, Zurich/Switzerland, from the Psychiatric University Hospital in Frankfurt/Main/Germany, from the Psychiatric University Hospital Charité, Humboldt University Berlin/Germany, Giessen/Germany, and Freiburg/Breisgau/Germany, and from the Hospital for Psychiatry and Psychotherapy in Göttingen-Tiefenbrunnen/Germany. Our cooperation with Professor Marianne Leuzinger-Bohleber's research group (Freud Institute Frankfurt/Germany and the Psychological Institute at the University of Kassel/Germany) was particularly fruitful. Many of the important findings discussed in this book were generated in close cooperation with the research groups at the ETH and University of Zurich/Switzerland (Professor Dr.rer.nat. P. Boesiger), the University Hospital of Psychiatry, Zurich, and the Humboldt University Berlin (Frau Dr. rer. nat. S. Grimm) and the University of Ottawa. Our stimulating discussions with lecturers and participants of various training courses in psychotherapy and psychoanalysis (Further Education in Psychiatry in North-East Switzerland, University of Zurich, Freud Institute Zurich, International Psychoanalytical University Berlin/IPU/Germany, Further Education in Analytical Therapy of Psychoses at the Academy of Psychoanalysis and Psychotherapy in Munich/Germany) and in various US-American, Canadian, and Chinese training and research institutes gave the editors of this book great inspiration.

Furthermore, we would like to thank Springer Publishers and especially Renate Scheddin for her encouragement right from the start, and Debika Bose (Project Coordinator for Springer Science) and Ejaz Ahmad (Assistant Project Coordinator) for their excellent, constructive cooperation.

We would particularly like to express our gratitude to Dawn Eckelhart for her valuable assistance in the preparation, coordination, and organization of the book's material and for making use of her talent to create order in chaos.

Zurich, Switzerland
Frankfurt, Germany
Ottawa, ON, Canada
May 2017

Heinz Boeker
Peter Hartwich
Georg Northoff

References

Bennet MR, Hacker PMS. Philosophical foundations of neuroscience. Oxford, UK: Blackwell; 2003.

Boeker H, Northoff G. Die Entkopplung des Selbst in der Depression: Empirische Befunde und neuropsychodynamische Hypothesen. Psyche – Z Psychoanal. 2010;64:934–76.

Boeker H, Richter R, Himmighoffen H, et al. Essentials of psychoanalytic process and change: how can we investigate the neural effects of psychodynamic psychotherapy in individualized neuro-imaging? Front Hum Neurosci. 2013;7:355.

Fuchs T. Difficult issues in neuroscience. Curr Opin Psychiatry. 2006;19:600–7.

Lebowitz MS, Ahn W. Effects of biological explanations for mental disorders on clinicians' empathy. PNAS. 2014;111(50):17786–90.

Northoff G. Neuropsychoanalysis in practice. Brain, self, and objects. Oxford, New York: Oxford University Press; 2011.

Northoff G, Boeker H, Bogert P. Subjektives Erleben und neuronale Integration im Gehirn: Benötigen wir eine Erste-Person-Neurowissenschaft? Fortschr Neurol Psychiat. 2006;74:627–33.

Contents

Introduction

1

Heinz Boeker, Peter Hartwich, and Georg Northoff

Abstract

Please check and confirm the hierarchy of the section headings are appropriate.

What Is "Neuropsychodynamic Psychiatry"?

This book, "Neuropsychodynamic Psychiatry", follows the tradition of psychodynamic psychiatry (Gabbard 2005, 2014), and discusses its fundamental principles, linking them with recent findings in neuroscientific research. The guiding principle is the concept of the "socially embedded brain" and the "relational self". Both psychopathological phenomena and psychic experience are considered in the context of social and interpersonal experiences. Hence, psychodynamic relationships are discussed in a neurobiological context. We are interested in the neuropsychosocial mechanisms underlying psychic experiences and social relationship as knowledge of the former might strongly impact how we can approach the latter in therapeutic terms. Hence "mechanism-based approach" integrating neuronal and psychic mechanisms is of central importance.

H. Boeker (✉)
Department of Psychiatry, Psychotherapy, and Psychosomatics,
Psychiatric University Hospital Zurich, University of Zurich, Zurich, Switzerland
e-mail: heinz.boeker@bli.uzh.ch

P. Hartwich
Department of Psychiatry, Psychotherapy, Psychosomatics,
General Hospital of Frankfurt am Main, Teaching Hospital University Frankfurt,
Frankfurt, Germany
e-mail: Prof.Hartwich@casytec.de

G. Northoff
Mind, Brain Imaging, and Neuroethics,
Institute of Mental Health Research, University of Ottawa, Ottawa, ON, Canada
e-mail: georg.northoff@theroyal.ca; www.georgnorthoff.com

H. Boeker et al. (eds.), *Neuropsychodynamic Psychiatry*,
https://doi.org/10.1007/978-3-319-75112-2_1

Accordingly, the essentials of neuropsychodynamic psychiatry may be defined as follows:

Neuropsychodynamic psychiatry is a diagnostic and therapeutic approach as well as a scientific model, encompassing unconscious conflicts and dilemmata, as well as distortions in intrapsychic structures and internalized object relationships, with a view to explaining, understanding, investigating, diagnosing and treating psychopathological phenomena. It focuses on the functionality and dysfunctionality of psychic and neuronal mechanisms and integrates these elements into the context of recent findings in neuroscience with a specific focus on the relational-social dimension of the brain and our self (cf. Böker et al. 2015).

As there is widespread literature in psychodynamics itself, we here define our psychodynamic starting point in clear terms. The starting point in the psychodynamic perspective is the three-dimensional model drawn up by Mentzos (1991, 2009). The three dimensions comprise defence mechanisms, personality structure and conflict. In this model, conflict in various stages of development should be understood as resulting from the interaction between defence mechanisms and structure. We aim to develop a model equivalent to the one designed by Mentzos on the neuronal level. Here, defence and compensation mechanisms on a psychic level correspond to neuronal mechanisms. These focus mainly on the interaction between intrinsic and extrinsic activity, and thus on how the brain and the outside world interplay with each other.

We here aim to extend Mentos' three-dimensional model from the psychological level to the neuronal and, more precisely, neuro-ecological level, that is, our brain's relation to and integration within the world, the world–brain relation as we will term it (Chap. 5 and Northoff 2016, 2017). Like Mentzos' three-dimensional model which was linked with various mental and psychiatric-syndromal disorders, different psychiatric syndromes are integrated into the three-dimensional neuropsychodynamic model. This neuropsychodynamic model thus forms the foundation of our book. As it forms the foundation of this very book, we illustrate our three-dimensional neuropsychodynamic model by the following figure (Fig. 1.1) to which we add a short explanation as given below which will be detailed and extended in Chap. 5.

This marble sculpture can be seen as a symbol of the spatial-temporal structure of the self, which represents what is described as self-continuity and can be found in the relationship between brain, body and environment as a continuous process in balancing out dynamic powers and structuring order.

Specifically, we suggest that the dimension of structure on the psychological level corresponds to the spatiotemporal organisation of the brain's spontaneous activity and its different layers. As it will become clear throughout the book, changes in different layers of the spontaneous activity's spatiotemporal features correspond to different psychiatric disorders. Conflict describes the encounter between internal and external demands on the psychological level which, as we assume, can be traced to the interaction between brain, i.e., its internal spontaneous activity, and external events in the world. As well manifest in the various symptoms, that very same interaction, i.e., rest–stimulus interaction (Northoff et al. 2010; Huang et al. 2017) is altered in several psychiatric disorders. Finally, defence mechanisms on the

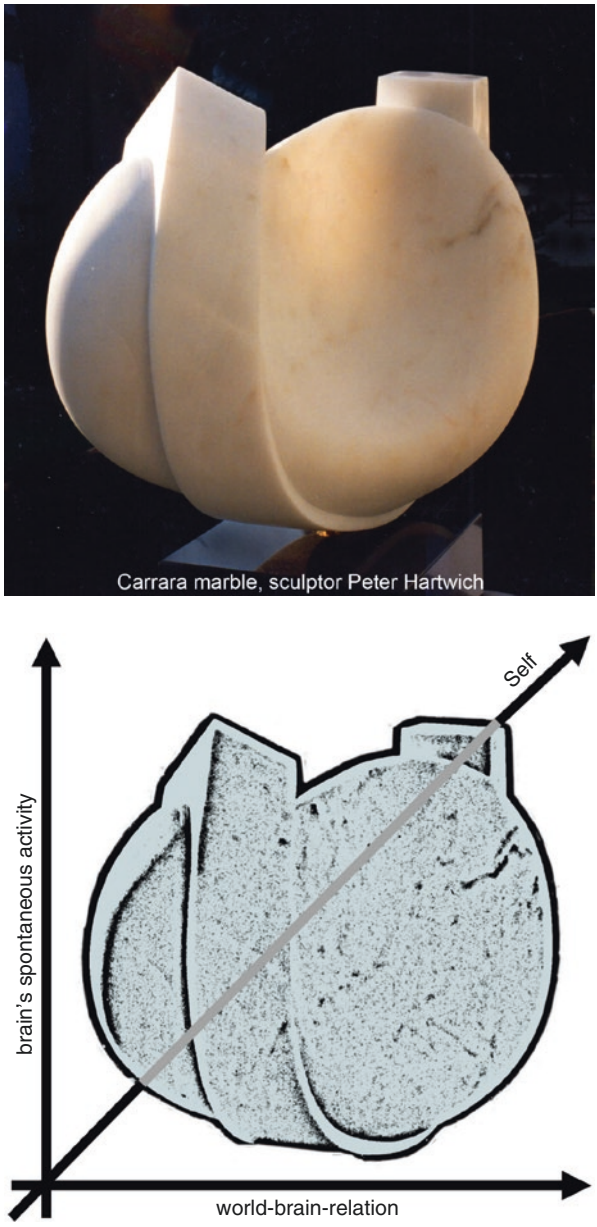

Fig. 1.1 Balance of dynamics and structure (Carrara-marble, sculptor Peter Hartwich, 2001)

psychological level supposedly correspond to the brain and its spontaneous activity's processing and elaboration of the various information about events in the world it receives. Hence, defence mechanisms reflect the spontaneous activity's capacity for dynamic change in response to internal and external information and events (Northoff 2011)—we will see that that very same capacity or predisposition is seriously altered in many psychiatric disorders.

1.1 The First Part of the Book: Neuropsychodynamic Foundations

In the book's first section, the neuropsychodynamic essentials outlined above are presented and discussed. As well as the historical development of fundamental psychoanalytical concepts (the unconscious, the self, psychic development and mental structure, emotions, conflicts and dilemmata, transference and counter-transference, attachment, mentalisation, defence and compensation mechanisms) and what role they play in modern neuroscience, the concept of the "socially embedded brain", the "relational self" and personality structure are addressed.

1.2 The Second Part of the Book: Neuropsychodynamics of Psychiatric Disorders

The book's second section focuses on the neuropsychodynamics of psychiatric disorders using a syndromal approach, including chapters on schizophrenia and other psychoses, depressive syndromes, manic and bipolar syndromes, anxiety disorders, obsessive-compulsive disorders, somatisation and body distress disorder, anorexia and bulimia, traumatogenic disturbances, personality disorders, dissociative syndromes, and last but not least, addiction.

Each chapter covers the following aspects:

- Clinical observations
- Psychodynamics
- Scientific results
- Neuropsychodynamic hypotheses
- Clinical and therapeutic implications

In specially marked boxes (with a grey background) specific viewpoints are discussed:

- Historical background
- Recent and current discussions
- Critical reflection
- Ideas for future research.

1.3 The Third Part of the Book: Neuropsychodynamic Perspectives

The book's third section focuses on neuropsychodynamic therapy, its established principles and their application in different therapeutic settings (in-patient, out-patient and day clinic). Each treatment intervention is put into the context of the neuropsychodynamic mechanisms mentioned above. Comparing the current state of

the art with neuropsychodynamic mechanisms, future therapeutic perspectives may be developed. The five aspects mentioned above (clinical observations, psychodynamics, scientific results, neuropsychodynamic hypotheses, clinical and therapeutic implication) are also covered in these chapters.

The third section of the book also presents future neuropsychodynamic perspectives: psychotherapy research in the context of neuroscience and ethical and neurophilosophical aspects are considered.

References

Böker H, Hartwich P, Northof G, editors. Neuropsychodynamische psychiatrie. Heidelberg: Springer; 2015.

Gabbard GO. Psychodynamic psychiatry in clinical practice. Washington DC: American Psychiatric Publishing Inc; 2005.

Gabbard GO. Psychodynamic psychiatry in clinical practice. 5th ed. Washington DC: American Psychiatric Publishing; 2014.

Huang Z, Zhang J, Longtin A, Dumont G, Duncan NW, Pokorny J, Qin P, Dai R, Ferri F, Weng X, Northoff G. Is there a nonadditive interaction between spontaneous and evoked activity? Phase-dependence and its relation to the temporal structure of scale-free brain activity. Cereb Cortex. 2017;27(2):1037–59. https://doi.org/10.1093/cercor/bhv288.

Mentzos S. Psychodynamische modelle in der psychiatrie. Göttingen: Vandenhoeck und Ruprecht; 1991.

Mentzos S. Lehrbuch der Psychodynamik. Die Funktion der Dysfunktionalität psychischer Störungen. Göttingen: Vandenhoeck und Ruprecht; 2009.

Northoff G. Neuropsychoanalysis in practice, self, object and brain. Oxford: Oxford University Press; 2011.

Northoff G, Qin P, Nakao T. Rest-stimulus interaction in the brain: a review. Trends Neurosci. 2010;33(6):277–84. https://doi.org/10.1016/j.tins.2010.02.006.

Northoff G. How do resting state changes in depression translate into psychopathological symptoms? From 'spatiotemporal correspondence' to 'spatiotemporal psychopathology'. Curr Opin Psychiatry. 2016;29:18–24.

Northoff G. The brain's spontaneous activity and its psychopathological symptoms–spatiotemporal binding and integration. Prog Neuro-Psychopharmacol Biol Psychiatry. 2017;80(Pt B):81–90. https://doi.org/10.1016/j.pnpbp.2017.03.019.

Part I

Neuropsychodynamic Foundations

Why Do We Need Psychopathology? From the Brain's Spontaneous Activity to "Spatiotemporal Psychopathology"

2

Georg Northoff

Abstract

The resurgence of biological psychiatry raises the question how and why we need psychopathology. Psychopathology has been well developed in the time where the brain was not yet explored; it has brought forth psychological approaches like cognitive psychopathology and experiential approaches like phenomenological psychopathology. Both psychological and experiential approaches suffer from a divide to the brain though, the divide between the brain and cognition as well as the divide between the brain and experience. I here suggest a novel form of psychopathology that focuses on spatiotemporal rather than cognitive or experiential features, i.e., spatiotemporal psychopathology. Thereby the brain's spontaneous activity plays a central role since it provides and shows various kinds of spatial and temporal features which, as I suppose, are organized cognition and are transformed into experience. I illustrate such spatiotemporal approach to psychopathological symptoms by the examples of depression and mania in bipolar disorder. I conclude that spatiotemporal psychopathology holds the promise to bridge the gap between the brain and symptoms including the divides between the brain and cognition/experience. Taken in this sense, spatiotemporal psychopathology will also be able to trace both psychological and experiential approaches to psychopathology to a commonly underlying basis, i.e., the spatiotemporal structure and features of the brain's spontaneous activity. Accordingly, we need psychopathology and, more specifically, "spatiotemporal psychopathology" to understand both the brain's neural activity and psychopathological symptoms and how the former translates into the latter.

G. Northoff
Mind, Brain Imaging, and Neuroethics,
Institute of Mental Health Research, University of Ottawa, Ottawa, ON, Canada
e-mail: georg.northoff@theroyal.ca; www.georgnorthoff.com

H. Boeker et al. (eds.), *Neuropsychodynamic Psychiatry*,
https://doi.org/10.1007/978-3-319-75112-2_2

This, in turn, opens a new understanding of psychodynamic mechanisms like defense mechanisms that can then be considered as different and specific "spatiotemporal configurations" as engineered on the basis of the brain' spontaneous activity and its spatiotemporal structure.

Neither the "brainless" psychiatry of the middle of the 20th century, nor the "mindless" variety of the past 30 years should be taken to represent the most we can achieve. The future should yield a synthesis. Panksepp (2004, p. 17)

2.1 Introduction

Neuroscience has made enormous progress in the last 20–30 years on all levels ranging from the genetic over the molecular to the regional and network level of neural activity. This has also affected psychiatry as in biological psychiatry. Various psychiatric disorders including schizophrenia, major depressive disorder (MDD), and bipolar disorder (BP) as well as others like addiction, personality disorders, etc. show molecular, genetic, regional, and network abnormalities in the brain. However, despite all progress in biological psychiatry, we still fall short in explaining the exact neuronal mechanisms of the various psychopathological symptoms. Specifically, biological psychiatry cannot yet explain how the brain's neuronal changes transform into the mind's alterations, the psychopathological symptoms.

Traditionally, the explanation and understanding of psychopathological symptoms has been the focus of psychopathology. Put in a nutshell, psychopathology concerns the empirical and theoretical framework in which symptoms, behavior, and experiences in psychiatric patients can be described, categorized, and classified (Parnas et al. 2008, 2013; Stanghellini 2009a, b; Stanghellini and Broome 2014). Different empirical and theoretical frameworks have been suggested in past and present approaches to psychopathology. However, how the different approaches to psychopathology (see below for details) are linked to the brain and its various neuronal mechanisms remains unclear.

Taken all together, we are facing a divide between biological psychiatry and psychopathology. The advocates of biological psychiatry tend to claim that all we need is the brain: the more we understand the brain and its abnormal changes in psychiatric disorders, the better we will understand and explain the psychopathological symptoms. This makes psychopathology as separate scientific discipline (Stanghellini and Broome 2014) meaningless and senseless and thus superfluous. Conversely advocates of psychopathology resist such interpretation. There is "more" to psychopathological symptoms than just the brain, and this "more" consists in the central role of experience or consciousness, i.e., the mind (Parnas et al. 2008, 2013; Stanghellini 2009a, b; Stanghellini and Broome 2014). Taken in this view, biological psychiatry remains as "mindless" as psychopathology is "brainless," to pick up our initial quote.

How can we resolve the divide between the brain and mind/symptoms and thus biological psychiatry and psychopathology? The aim in this paper is to show that a

novel approach, "spatiotemporal psychopathology," can bridge this divide by providing a "common currency" between the brain and symptoms—that "common currency" is supposed to consist in spatiotemporal features that transform abnormal neuronal activity in psychopathological symptoms.

2.2 "Spatiotemporal Psychopathology": Determination and Distinction

2.2.1 Psychological Approaches to Psychopathology

Roughly, one may want to divide between psychological and experiential approaches to psychopathology. Psychological approaches focus on specific psychological functions like cognitive or affective cognitive as in cognitive psychopathology (David and Halligan 2000; Halligan and David 2001), affective psychopathology (Panksepp 2004). With the development of neuroimaging, these approaches are now able to link the objectified changes in cognitive and affective functions onto the brain. However, such "mapping" of cognitive into neural functions leaves open how and why abnormal changes in the brain's neural activity are transformed into psychopathological symptoms.

How can cognitive and affective and cognitive functions more strongly link to the brain and its neuronal mechanisms? We would need to unravel a yet unclear "common currency" that allows to transform neuronal into psychological activity. To be clear, I am not raising the question which regions or networks in the brain are related to cognitive functions. Such "cognitive-neural mapping" has been well established in cognitive neuroscience that showed how cognitive functions like executive functions, attention, memory, etc. are related to specific regions or networks in the brain. This and the respective changes in those regions and networks have been well researched intensely over the last 10–20 years. Instead, I am raising the question why and how the brain's neuronal activity in those regions and networks transforms into cognitive (and affective functions) rather than remaining merely neuronal (and non-cognitive).

What is needed is a "common currency" between neural and cognitive functions—due to such yet unclear "common currency," neural activity translates into cognitive function basically by default. And it is this transformation or translation that seems to be altered in psychiatric disorders that can indeed be characterized by numerous cognitive deficits. I postulate that the spatial and temporal features of the brain's spontaneous activity provide such "common currency"—cognitive symptoms are spatiotemporal symptoms for which reason I speak of "spatiotemporal psychopathology."

2.2.2 "Common Currency" Between the Brain and Cognition

We are confronted with a divide between the brain on the one hand and cognition on the other. Biological psychiatry focuses on the brain while leaving out the mind and its experience. While psychological approaches to psychopathology focus on

cognitive functions and the relation of their contents to the brain, neither has yet provided a full-fledged explanation and understanding of psychopathological symptoms though. We are thus confronted with a divide between the brain and cognition.

Psychological approaches to psychopathology focus on contents, i.e., cognitive, affective, sensorimotor, and social contents and their related functions. The cognitive, affective, sensorimotor, and social contents are then "mapped" upon the brain and its various regions and networks—this is where psychological approaches to psychopathology converge with biological psychiatry. This neglects one central dimension of the contents though. The contents are organized and structured in a particular way, and this organization is mainly spatial and temporal. Spatiotemporal psychopathology as suggested here focuses on the temporal and spatial organization of the contents rather than the nature of the contents themselves, i.e., cognitive, affective, sensorimotor, or social.

Spatiotemporal psychopathology aims to unravel the spatiotemporal organization and structure within which the various kinds of contents are embedded hence the name "spatiotemporal psychopathology." Alterations in cognition in psychiatric disorders are consequently not related to specific contents, i.e., cognitive, affective, etc. Instead, abnormal cognition is related and traced to abnormal spatial and temporal organization within which the contents are embedded.

Let us give an empirical example. Duncan et al. (2015) recently demonstrated that early childhood traumatic experience is manifested in adulthood in the spatiotemporal patterns of the brain's spontaneous activity (as indexed by entropy) which, in turn, impacts subsequent stimulus-induced activity in relation to aversive stimuli. The early childhood traumata were thus encoded in terms of spatiotemporal features, i.e., entropy, rather than in terms of specific contents and cognitions. Sure, the very same spatiotemporal pattern impacts the contents and their subsequent cognition—however, it is clear that the latter has a spatiotemporal basis in the spatiotemporal features of the brain's spontaneous activity. Hence early childhood traumas are primarily a matter of spatiotemporal organization of the contents, i.e., life events, rather than being directly related one-to-one to the life event and its content itself.

Taken together, the spatiotemporal organization of the brain's spontaneous activity may provide the "currency" that translates directly into the cognitive level with the cognition of contents. Spatial and temporal features as manifest in both the brain's spontaneous activity and our cognition of contents may consequently provide the "common currency" between the brain and cognition. Changes in cognition as in psychiatric disorders may then be traced to alterations in the resting state's spatial and temporal features. This would link the psychological approaches to psychopathology even more tightly to the brain while, at the same time, providing a new view on the brain and especially its resting state, a spatiotemporal rather than cognitive view (Northoff 2016a).

2.2.3 Experiential Approaches to Psychopathology

In contrast to psychological approaches to psychopathology, experiential approaches focus on the subject's experience, i.e., subjective experience, of self, world, and

body rather than on objectified cognitive and affective functions. The hallmark experiential approach is phenomenological psychopathology takes the subject's experience of self, body, and world and thus the structure of its consciousness as explanatory framework for psychopathological symptoms (Fuchs 2007, 2013; Northoff 2016b; Parnas et al. 2008, 2013; Stanghellini 2009a, b; Stanghellini and Broome 2014).

Siblings of phenomenological psychopathology include existential psychopathological, which focuses on the existence as the deeper layer underlying experience, and the hermeneutical psychopathology that emphasizes the meaning of symptoms in a wider biographical and environmental context (Stanghellini 2009a, b). Despite the difference in focus or emphasis, the overall explanatory framework in all three approaches consists in experience or consciousness for which I reason I subsume under the "experiential approaches" to psychopathology.

Phenomenological psychiatry takes experience or consciousness itself as starting point and focuses on exploring first-person experiences in detail (Parnas et al. 2008, 2013; Stanghellini 2009a, b). Specifically, the focus is on the first-person experience of time and space as well as body, self, and world. The brain, in contrast, nowhere surfaces in experience, in particular, and phenomenology, in general, since it cannot be accessed in experience in first-person perspective but only in observation as in third-person perspective. The brain is thus excluded in experience of the own self, body, and world including time and space in particular and phenomenology in general. Such exclusion of the brain in experience or consciousness occurs by default, e.g., on methodological grounds, since the brain cannot be accessed in experience in first-person perspective. Importantly, this leaves the link to the brain open and renders the experiential approaches to psychopathology ultimately as "brainless" (as picking up our initial quote).

2.2.4 "Common Currency" Between the Brain and Experience

How can we close the gap between experience and brain? Closing this gap is central for psychiatry since we need to understand the processes that transform abnormal neuronal into phenomenal states which psychiatric patients experience in first-person perspective. How can we apprehend these transformative processes, e.g., neuronal-phenomenal transformation? For that we may want to search for a shared overlap or "common currency" between neuronal and phenomenal states that drives the transformation of the former into the latter.

The shared overlap or common currency between neuronal and phenomenal states, e.g., the brain and experience, may consist in spatiotemporal features. On the side of the brain, it is the spontaneous activity (rather than its stimulus-induced or task-evoked activity) that may be central in providing or constituting such spatiotemporal structure (see below for details). The brain's spontaneous activity shows certain spatiotemporal features, a particular spatial and temporal structure in its neural activity that surfaces in and is transformed into phenomenal state, e.g., experience (see (Northoff 2016a) for many examples). One would consequently expect a common, similar, or analogous spatiotemporal structure between the brain's spontaneous activity and the phenomenal features of experience.

Such common, similar, or analogous spatiotemporal structure between the brain and experience amounts to what I describe as "spatiotemporal correspondence." The concept of spatiotemporal correspondence means that the brain's spontaneous activity and the phenomenal features of experience show corresponding or analogous spatial and temporal features: the spatial and temporal configuration or structure of the neural activity in the brain's spontaneous activity surface in the spatial and temporal features within which the contents of experience (like specific objects or events including body, self, and world) are integrated and thus structured and organized.

For instance, a recent study of ours demonstrated that private self-consciousness is directly related to the temporal patterns of spontaneous or resting state activity across different frequency ranges (as indexed by what is described as "power law") (Huang et al. 2016). This suggests that mental features like self may be rooted in spatiotemporal features of the brain's spontaneous activity. The self as mental feature may then be characterized in spatiotemporal terms, that is, by specific spatiotemporal schemata or structure rather than by cognition of particular contents (see Fig. 4.1).

Unlike biological psychiatry that focuses on the brain itself independent of its respective ecological context, phenomenological psychiatry emphasizes the integration of experience including the subject of experience within the ecological context of the world. There is continuity between experience and world with such continuity often assumed to be mediated by the body, e.g., experience of the body as lived body (see, for instance, Northoff and Stanghellini 2016). Such continuity between subject and world is deemed central for making experience including the first-person perspective itself first and foremost possible.

2.3 Spatiotemporal Psychopathology: Depression and Bipolar Disorder

2.3.1 Spatiotemporal Psychopathology: Bipolar Disorder and Neuronal Variability

How about spatiotemporal changes in the resting state in bipolar disorder (BP)? Several resting state investigations observed changes in functional connectivity in the default mode network in bipolar disorder though the phases, i.e., depressed, euthymic, and manic, are rarely specified. Going beyond functional connectivity, we investigated neuronal variability in different resting state network in manic, euthymic, and depressed phases of BP as well as healthy subjects. Neuronal variability is measured by the root means square of the amplitude, in that it reflects the change in the amplitude over time and the degree to which these changes vary over time. Taken in this sense, neuronal variability can be considered a measure of the temporal structure and, more specifically, the temporal dynamics of the ongoing spontaneous activity.

We focused on neuronal variability (SD) in the main neural networks, default mode network (DMN), central executive network (CEN), salience network (SN), and sensorimotor network (SMN). Depressed BP patients showed significantly

decreased SD in the sensorimotor network, while their SD was significantly increased in the DMN. The other neural networks like SN and CEN did not show any SD changes. We then calculated the ratio or balance between DMN SD and SMN SD; this was tilted significantly toward the DMN SD at the expense of the SMN SD.

What does this mean? Neuronal variability may be linked to the initiation of internally directed cognition in DMN and movements/actions in SMN. The more often the neuronal variability change surpasses a certain threshold, the more often the respective regions internally, i.e., by itself independent of external stimuli, initiate either cognition or action. Let us be more specific regionally. The DMN has been associated with internally directed cognition as in spontaneous cognition and mind wandering. If now neuronal variability is abnormally high in the DMN, there is a higher likelihood that spontaneous thoughts will be initiated. This is exactly what one can observe in depressed BP where the patients suffer from increased spontaneous thought which are described as rumination.

How about the SMN? In that case, neuronal variability may be related to the spontaneous or internal initiation of movements and actions. If now neuronal variability in SMN is decreased, one would expect decreased internal initiation of movements and actions. This, again, is exactly what can be observed in depressed BP where patients often suffer from psychomotor retardation. Most interestingly, it seems that the balance between DMN SD and SMN SD is central since the balance correlated significantly positively with depressive symptoms (as measured with Hamilton depression scale): the more the SD balance was shifted toward the DMN at the expense of the SMN, the more and stronger depressive symptoms.

The reverse could be observed in the manic phase. Here SD was significantly lower in the DMN and abnormally high in the SMN; the balance between DMN SD and SMN SD is consequently tilted toward the SMN at the expense of the DMN. This is symptomatically manifested in increased internal initiation of movement/action as it is reflected in the well-known psychomotor agitation in mania. In contrast, internally directed cognition like spontaneous thought is no longer initiated internally as much—this is reflected in the fact that many manic patients say "that they do not think much or not at all" in the manic episode.

2.3.2 Spatiotemporal Psychopathology: From Neuronal Variability to Cognition and Experience

What do these findings tell us about the nature of psychopathological symptoms? There is still internal initiation of movements as related to SMN and internally directed cognition, i.e., spontaneous thought as based on DMN. However, the neuronal mechanism potentially underlying such internal initiation, i.e., neuronal variability, is expressed to an abnormal degree. It is either too high or too low which leads to either increased or decreased internal initiation of the respective function. That very same neuronal mechanism is temporal, i.e., SD, and spatial, i.e., in

different networks like DMN and SMN, and can therefore be considered "spatiotemporal mechanism" as I say (see Fig. 4.2).

Let me be more precise. The function, i.e., internal initiation of movements/action and internally directed cognition, is still intact by itself—the bipolar patients are still able to internally initiate them. This distinguishes psychiatric patients from neurological patients. In the latter, the region itself is lesioned which makes impossible the internal initiation of, for instance, movement and action as in Parkinson's disease. However, the function of internal initiation of movement/action and internally directed cognition is expressed in an abnormally high or low way due to some spatial and temporal abnormalities in the brain's spontaneous activity, i.e., SD in SMN and DMN. The resulting abnormalities in movement/action and internally directed cognition, i.e., the psychopathological symptoms, are thus based on and can be traced to spatiotemporal changes in the brain's spontaneous activity. Rather than being primarily motor, as in Parkinson's disease, the psychomotor changes in mania and depression are thus spatiotemporal at their very basis.

The same holds analogously for internally guided cognition. Unlike in neurological lesion patients, the bipolar patients can still initiate internally directed cognition like spontaneous thought. However, that very same internal initiation is temporally disorganized by the abnormal high neuronal variability in DMN in depression and the low SD in DMN in mania. The cognitive symptoms like rumination (or decreased thought) are consequently not primarily cognitive but rather spatiotemporal as they are related to spatiotemporal changes in the brain's spontaneous activity.

Taken together, the example of BP nicely demonstrates that cognitive and motor symptoms in both depression and mania are not related to primary dysfunction in either cognitive or motor functions. Instead, the basic function, i.e., cognitive or motor, is preserved by itself but abnormally organized in spatial and temporal terms. Therefore, the symptoms are spatiotemporal rather than cognitive and motor. What is described as cognitive in cognitive psychopathology is based on and can be traced to spatiotemporal abnormalities in the brain's spontaneous activity and thus spatiotemporal psychopathology.

The same holds for experience and phenomenological psychopathology. Depressed patients often experience their "inner time," i.e., the time of their own self, as extremely slow which, when taken as reference, lets them perceive the "outer time," i.e., the time in the environment, as extremely fast (Fuchs 2013). We measured neuronal variability in the neural network underlying "inner time," i.e., the somatosensory network (SS), and the one related to "outer time," i.e., primary sensory regions like visual cortex (VS). This yielded decreased SD in the SS and increased SD.

How are these findings related to the experience or perception of time? Neuronal variability indicates change in neural activity, and the more change there is, the faster the time. Decreased SD in SS thus indicates slower "inner time," while increased SD in VC reflects faster "outer time"—this corresponds exactly to the experience of time depressed patients report. The opposite SD pattern with increased SD in SS and decreased SD in VC was observed in manic

patients—this corresponds well to their experience of faster "inner time" and slower "outer time."

Taken together, these findings indicate how a temporal measure like neuronal variability is translated into experience or perception, i.e., the experience of the speed of time. Hence, experience of the speed of time may be traced to and be based on a corresponding neuronal measure that indicates the speed of the brain's time, i.e., neuronal variability. Hence, the change in the brain's time speed, i.e., its neuronal time as indexed by neuronal variability, is transformed into corresponding experience or perception, i.e., the experience of the speed of time. Experience of time and experience in general is thus spatiotemporal by itself and thereby based on the spatiotemporal features of the brain's spontaneous activity. Experiential approaches like phenomenological psychopathology are thus ultimately based on and can be traced to spatiotemporal features and hence spatiotemporal psychopathology.

Conclusion

How can we bridge the divide between the brain and cognition and hence between biological psychiatry and cognitive psychopathology? I demonstrated how cognitive changes like rumination in depression and decreased cognition in mania are related to abnormal expression of spatial and temporal mechanisms of the brain's spontaneous activity. Hence, I postulate that what is described as abnormal cognition in cognitive psychopathology is based on and can be traced to abnormal spatial and temporal organization of cognitive functions—this entails what I describe as "spatiotemporal psychopathology." Accordingly, I postulate that the spontaneous activity's spatial and temporal features provide the bridge between the brain and cognition. Therefore, spatiotemporal psychopathology provides the bridge between biological psychiatry on the one hand and cognitive psychopathology on the other.

How about the divide between the brain and experience and hence between biological psychiatry and phenomenological psychopathology? I showed how the abnormal experience of time in depression and mania may be based on abnormal temporal features like neuronal variability in the brain's spontaneous activity. Experience is thus based on spatiotemporal features—the spatiotemporal features of the brain's spontaneous activity transform into experience which thereby can be characterized as spatiotemporal. Hence, the spontaneous activity's spatiotemporal structure allows linking the brain and experience and can therefore bridge the divide between biological psychiatry and phenomenological psychopathology.

The initial question and title in this paper is: Why Do We Need Psychopathology? We need psychopathology to bridge the gap between the brain and cognition as well as the one between the brain and experience. This does not only provide common link between biological, cognitive, and experiential forms of psychopathology but also a novel, i.e., spatiotemporal, understanding of both the brain and symptoms. I postulate that spatiotemporal psychopathology as sketched here provides exactly that form of psychopathology that allows us to understand the brain and how its neural activity transform

into cognition and experience and subsequently the kind of symptoms we observe in our patients.

Why is spatiotemporal psychopathology relevant for neuropsychodynamic psychiatry? First and foremost, it opens a novel way of understanding psychodynamic mechanisms in a neurobiological context. For instance, defense mechanisms can then be understood in a spatiotemporal way as specific constellations of how time and space can (or cannot) be constructed by the brain's spontaneous activity in its relation to both body and world. In other terms, defense mechanisms are spatiotemporal mechanisms—this sheds a novel light on the defense mechanisms and other psychodynamic features. And, at the same time, the elaborate description of different defense mechanisms may also provide insight into the different kinds of possible spatiotemporal configurations as constructed on the basis of the brain's spontaneous activity. Hence, there may be true bilateral exchange between neuropsychodynamic psychiatry and spatiotemporal psychopathology.

References

David AS, Halligan PW. Cognitive neuropsychiatry: potential for progress. J Neuropsychiatry Clin Neurosci. 2000;12(4):506.

Duncan NW, Hayes DJ, Wiebking C, Brice T, Pietruska K, Chen D, et al. Negative childhood experiences alter a prefrontal-insular-motor cortical network in healthy adults: a preliminary multimodal rsfMRI-fMRI-MRS-dMRI study. Hum Brain Mapp. 2015;36:4622–37.

Fuchs T. The temporal structure of intentionality and its disturbance in schizophrenia. Psychopathology. 2007;40(4):229–35.

Fuchs T. Temporality and psychopathology. Phenomenol Cogn Sci. 2013;12(1):75–104.

Halligan PW, David AS. Cognitive neuropsychiatry: towards a scientific psychopathology. Nat Rev Neurosci. 2001;2(3):209–15.

Huang Z, Obara N, Davis HH IV, Pokorny J, Northoff G. The temporal structure of resting-state brain activity in the medial prefrontal cortex predicts self-consciousness. Neuropsychologia. 2016;82:161–70.

Northoff G, Stanghellini G. How to link brain and experience? Spatiotemporal psychopathology of the lived body. Front Hum Neurosci. 2016;10:172.

Northoff G. Spatiotemporal psychopathology I: no rest for the brain's resting state activity in depression? Spatiotemporal psychopathology of depressive symptoms. J Affect Disord. 2016a;190:854–66. https://doi.org/10.1016/j.jad.2015.05.007.

Northoff G. Spatiotemporal psychopathology II: how does a psychopathology of the brain's resting state look like? Spatiotemporal approach and the history of psychopathology. J Affect Disord. 2016b;190:867–79. https://doi.org/10.1016/j.jad.2015.05.008.

Panksepp J. Textbook of biological psychiatry. New York: Wiley; 2004.

Parnas J, Sass LA, Zahavi D. Recent developments in philosophy of psychopathology. Curr Opin Psychiatry. 2008;21(6):578–84.

Parnas J, Sass LA, Zahavi D. Rediscovering psychopathology: the epistemology and phenomenology of the psychiatric object. Schizophr Bull. 2013;39(2):270–7.

Stanghellini G. A hermeneutic framework for psychopathology. Psychopathology. 2009a;43(5):319–26.

Stanghellini G. The meanings of psychopathology. Curr Opin Psychiatry. 2009b;22(6):559–64.

Stanghellini G, Broome MR. Psychopathology as the basic science of psychiatry. Br J Psychiatry. 2014;205(3):169–70.

Psychoanalysis and Neuroscience: The Development of Neuropsychoanalysis

3

Heinz Boeker

Abstract

In his *Project for a Scientific Psychology*, published in 1895, Freud tried to connect psychoanalysis with the neuroscience of his day. Finally, he was forced to give up this endeavour because of the lack of diagnostic possibilities and empirical facts. Freud's paradigmatic change to psychoanalysis came about through his work on the mind-body problem and its central question of how the brain is able to generate subjective experience (consciousness) by means of existing anatomical structures and physiological functions.

This chapter focuses on the development of modern neuroscience with its diagnostic-technical repertoire and the possibility to gain insight into neuronal processing of mental processes such as emotional-cognitive interaction, which enabled to look for connections between what is known in both disciplines.

Lurija's neurodynamic approach, Kaplan-Solms and Solms' neuroanatomical methods, Damasio's *The Feeling of What Happens*, Panksepp's *Affective Neuroscience* and the discovery of the so-called mirror neurons (Rizzolatti, Galese) are milestones of the development of neuropsychoanalysis.

Finally, the further development of a "first-person neuroscience" (Northoff) may contribute suitable strategies to answer core questions on the relation between subjective experience and neuronal integration in the brain.

The apportioning of the determining factors of our life between the 'necessities' of our constitution and the 'chances' of our childhood may still be uncertain in detail; but in general it is no longer possible to doubt the importance precisely of the first years of our

H. Boeker
Department of Psychiatry, Psychotherapy, and Psychosomatics, Psychiatric Hospital, University of Zurich, Zurich, Switzerland
e-mail: heinz.boeker@bli.uzh.ch

H. Boeker et al. (eds.), *Neuropsychodynamic Psychiatry*,
https://doi.org/10.1007/978-3-319-75112-2_3

> childhood. We still show too little respect for Nature which (in the obscure words of Leonardo which recall Hamlet's lines) 'is full of countless causes ('ragioni') that never enter experience'.
>
> Everyone of us human beings corresponds to one of the countless experiments in which these 'ragioni' of nature force their way into experience.
>
> (Freud 1990, p. 137)

3.1 Introduction

This chapter will focus on challenges which ultimately led to the development of a "theory of the unconscious" by Freud and later psychoanalysts from a clinical perspective. The beginnings of the dialogue between psychoanalysis and neuroscience will be presented, starting with Freud's *Project for a Scientific Psychology*. Furthermore, the important role of the unconscious and the unconscious phantasy in modern-day psychoanalysis will be examined. In this context, connections with neuroscientific research strategies and concepts of "cognitive neuroscience" concerning unconscious information processing will also be mentioned. This historical review of how a "theory of the unconscious" was developed and rejected will show just how relevant this issue remains today.

3.2 Freud and the Demystification of the Unconscious

The concept of the unconscious and the theory of instincts are the cornerstones of a psychoanalytical understanding of man's psychic reality. They go hand in hand with each other, are dependent on each other and are merely two different viewpoints from which psychoanalysis considers and examines psychic reality. Together they form a theory of man's first and most urgent wishes, his phantasies and conflicts, expressed above all in his sexuality, aggression and striving for self-assertion (Müller-Pozzi 2002).

Freud is often credited as having discovered the unconscious: this is an honour he neither merited nor claimed for himself or for psychoanalysis. On the contrary, when Freud began exploring the unconscious origins of mental disorders, the consideration of the unconscious forces of the mind was a popular subject in literature and philosophy. To counterpoint one-sided rationalism, the romantic transfiguration and mystification of the dark and mysterious unconscious side of the psyche reached its late climax at the end of the nineteenth century (for instance, in the works of the philosophers Arthur Schopenhauer, Friedrich Nietzsche and Ludwig Klages and the authors Hugo von Hofmannsthal and Arthur Schnitzler).

Freud kept a critical distance from these cultural schools of thought. He denied himself, as he put it (Freud 1914b, pp. 15–16), "…the very great pleasure of reading the works of Nietzsche, with the deliberate object of not being hampered in working out the impressions received in psycho-analysis by any sort of anticipatory ideas. I had therefore to be prepared–and I am so, gladly—to forgo all claims to priority in the many instances in which laborious psychoanalytic investigation can merely confirm the truths which the philosopher recognized by intuition".

Prior to his psychoanalytical discoveries, Freud's career was apparently that of a young scientific researcher in the late nineteenth century. However, Freud lacked the financial means needed to pursue a purely scientific career. Yet he may well have succeeded in this endeavour, had he not fallen in love. Today, in the light of his partly published letters to his fiancé, it may inevitably be concluded—as Israël (1983) wrote—that his work and his private life were closely connected and that, indeed, without passion nothing can be discovered, invented or captured.

These circumstances in Freud's private life are worth mentioning, since, as we will see later, his examination of the unconscious was preceded by a far-reaching professional and, thus for Freud, personal crisis, namely, when he discovered that what some of his female patients told him did not concern the actual trauma itself but was an expression of their unconscious phantasies.

As a neurologist, Freud was confronted on a daily basis with the symptoms of hysteria, a common illness at that time. Hysteria was manifest externally by striking functional disturbances, such as paralysis, difficulties swallowing, visual disturbances, pain and impaired consciousness. For medical professionals in those days, a condition which could not be somatically explained and treated was considered a nuisance and a contradiction, so that the patient was often suspected of faking the illness.

Freud did not close his eyes to the "neurotic suffering" of these female patients. Based on knowledge from doctors who had at times used hypnosis to cure hysterical symptoms, he began, together with Josef Breuer, to investigate hysterical patients' subjective experience.

He looked for the causes of hysterical symptoms not in organic factors but in the patient's subjective experience and was finally convinced that hysterical symptoms should be viewed as the physical manifestation of mental reality. Thus, the symptoms have meaning, but are expressed in body language which cannot be directly understood. Freud believed that this strangely distorted language of the symptoms was connected to the unconscious dimension, which not even the patient herself can access. Freud realized that the hysterical patient suffers from her reminiscences. These are often memories of emotionally meaningful experiences involving significant persons from the patient's childhood. Reflected in these reminiscent memories are intensive wishes and phantasies which could not be lived out in earlier relationships and have been pushed aside into unconscious phantasies. The "unconscious phantasy"—in sharp contrast to the psychology of consciousness—became one of the most important concepts in psychoanalysis.

The foundation for the psychoanalytical concept of the unconscious was thus laid: what previously seemed incomprehensible and nonsensical now seemed to make sense as a derivative and as the manifestation of the unconscious. This is not only the case for psychopathology in the narrow sense but also for the "psychopathology of everyday life", i.e. the Freudian slips and symptomatic actions which, like wit, dreams, daydreams, phantasies and even being artistically creative, can be seen as the manifestation or as deriving from unconscious phantasies. With this interpretation, Freud "challenged the whole world" and summoned up "the greatest critical minds against psychoanalysis" (Freud 1915a, b, p. 287, 294).