



# Why Biological Psychiatry Hasn't Delivered Yet – and Why Neurology Knows

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It is increasingly recognized that neuroscience has not delivered the revolutionary clinical possibilities for psychiatry that had been promised. Explanations differ, however: some proponents emphasize the divide between biopsychosocial psychiatry and mechanistic neurology. Others rely on further basic experimental neuroscience as only the most elementary level of explanation will allow us to fully understand and treat mental disorders. From a clinical-neuropsychological perspective, I shall argue that both views are mistaken. Diagnosis and treatment of neurological diseases demands a biopsychosocial perspective similar to psychiatry. Acknowledging this might help to bring both disciplines together and improve clinical outcome.

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## INTRODUCTION

After more than 20 years of neuroscientific enthusiasm in diagnosing and treating mental disorders, a sobering atmosphere has gained ground. Questions are raised why neuroscience—along with other domains of biological psychiatry such as genetics or neurochemistry—has failed so far to deliver the promised clinical possibilities.<sup>1</sup> Explanations for this failure differ, however. One quite common position holds that neurological illnesses are causally dependent on specific brain mechanisms, in contrast to mental disorders.<sup>2</sup> An opposing view sees brain mechanisms as crucial in both cases, but argues that more basic experimental research is necessary in order to fully reveal them.<sup>3</sup> In the following, I shall argue that both explanations fail: although the putative divide between psychiatry and neurology is actually much smaller than suggested, both do not profit from a mechanistic view. At least not in the literal sense of ‘mechanism’ as a steady and repetitive, machine-like phenomenon, which can be fully isolated from an organ-

ismic context. The idea of mechanisms is a result of artificially isolating single aspects from an organism-environment context in laboratory experiments.<sup>4,5</sup> Clinical, ‘real-world’ neurology cannot neglect this context and therefore not dispense of an integrative, biopsychosocial perspective on brain and organism, similar to psychiatry.

## BIOLOGICAL PSYCHIATRY: THE CALL OF MECHANISM (AND SOME PSEUDO-PROBLEMS)

Current discussion of the divide between psychiatry and neurology profoundly suffers from ideological tendencies. Biological psychiatry (BP), especially in its most ambitious versions originating in the late 1990ies, characterized itself by an a priori commitment to basic levels (neuronal, neurochemical etc.) of explanation.<sup>6</sup> Accordingly, BP critics are being accused of not accepting the idea that all there is to the mind are just nerve impulses.<sup>7</sup> Following the epistemology of experimental neuroscience, psychosocial approaches are either discarded as unscientific<sup>7</sup> or reduced to fundamental mechanisms on a ‘hard’ (neuro)science base.<sup>3,7</sup> This view often goes along with a vigorous campaign against a supposed Cartesian mind-brain dualism of BP critics. This dualism is then countered by repeatedly pointing out correlations of structural or functional brain abnormalities with symptoms of mental disorders.<sup>8</sup> Unfortunately, dualism is often misinterpreted in psychiatric discourse

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as an independence between mind and brain.<sup>2,9,10</sup> It is therefore questionable whether traditional psychiatry's intrinsic dualism actually hampers clinical progress. Historically, psychiatry has long known all types of bodily treatments of mental illness, such as physical activity, bleeding, purging, baths, insulin shocks, electroconvulsion, psychosurgery etc. It seems a dead end to repeatedly point to brain-symptom associations in mental illnesses such as schizophrenia or depression in order to demonstrate that mind and body are interrelated.<sup>8,11</sup> A more fertile part of the debate concerns the question of why such findings have largely failed to revolutionize the diagnosis and treatment of mental disorders so far.

A common explanation relies on a contrast between psychiatry and neurology: whereas neuroscience has revealed 'brute mechanical' causal conditions in neurological disorders,<sup>2</sup> analogous causal connections do not exist in mental disorders. This view often defends the autonomy of psychiatry by claiming that mental illness necessitates an integrative view of the person with its subjective view and his/her entanglement with a physical and social environment. Neurological diseases, by contrast, are seen as existing just inside the brain and cognitive or affective symptoms as being produced inside the brain.<sup>2</sup> A holistic, personal approach in psychiatry is thus contrasted with a brain-centered view of neurology, where the machine metaphor for the brain straightforwardly applies. This view, however, is questionable, but not because a mechanistic (e.g. machine-like) view of the brain is adequate for psychiatry. As I shall argue, this view is inadequate for neurology as well, as revealed most evidently by clinical neuropsychology.<sup>6</sup>

## THE ROLE OF 'BRAIN MECHANISMS' IN CLINICAL NEURO(PSYCHO)LOGY

Despite prominent holistic proponents, a mechanistic view of the brain prospering in the 19th century has largely dominated neurology.<sup>6</sup> Historically, the concept of mechanism has been very powerful in singling out specific causal relationships in constructing machines. Transferring this idea to living organisms (or parts thereof), however, is largely metaphorical and quickly reaches its limits: In order to make them clinically useful, mechanisms have to be construed as independent from an organismic context.<sup>5</sup> Traditional neuro(psycho)logy aimed at identifying symptoms (such as problems in naming, repeating words, calculating etc.) and relating them to regional brain lesions. Apart from conceptual problems,<sup>4</sup> disregarding the context of the organism (or seeing it as merely additive) neglects all higher order influences onto those basic mechanisms.<sup>5</sup> To take a seminal example, aphasic patients may be able to use a word in everyday language but not in a clinical investigation when naming a picture. Thus, the brain still represents the word

(due to lesioned areas 'storing' it), but is unable to make it available in a specific context.<sup>4</sup>

Many established results from experimental neuroscience associating mental functions (anxiety, pain, working memory etc.) with brain regions are not reliably mirrored in lesioned brains. This is not only due to the fact that these functions can involve distributed networks of activity. Rather, lesioned brains may shift networks, even by recruiting additional areas normally not engaged in these functions.<sup>4</sup> Compensatory functions of the brain depend on a range of inextricably intertwined predictors.<sup>4,6</sup> There is an evolutionary value to organisms' inherent 'teleology', that is, their ability to establish and maintain an identity across development or damage and across changes in the environment. In case of a brain damage due to a neurological condition, clinical neuropsychologists normally see a sophisticated *mélange* of individual changes. These depend on the compensatory processes in the brain and organism, both unconscious and conscious, such as neuronal reorganization, tasks avoidance, compensation through (self-)training, deficit awareness, reduction of drive, resilience, real-life demands, life goals etc.<sup>6</sup> A brain-centered, mechanistic view of brain-function relationships is therefore inadequate to understand and treat cognitive or affective sequelae of neurological diseases. This is why experimental results isolating 'brain mechanisms' often do not translate into clinical practice as seen in numerous laboratory results that do not reach the phase of clinical trials.<sup>5</sup> Human organisms are intrinsically relational with respect to 1) their awareness for and interpretation of deficits and 2) their physical, social and cultural environment. This requires a biopsychosocial perspective which is apt to explain why organismic influences may easily alter or even suspend local mechanisms, as seen in the placebo, nocebo and lessebo effects in all major neurological diseases.<sup>12</sup> Suspension of putative mechanisms may be due to a variety of factors, intrapsychic (such as hope), interpersonal (such as trust and distrust) and sociocultural.<sup>12</sup> Finally, only a comprehensive, organism-environment perspective allows us to understand diseases as suffering, namely in terms of a subjective discrepancy between individual organismic resources on the one hand and individual environmental demands on the other (modulated by personal values and goals). This discrepancy is the actual target for any clinical intervention.

Unfortunately, due to its traditions, neurology has long stuck to the medical model with fatal consequences for treatment. As an example, it has long been resisting psychosocial approaches such as psychotherapy.<sup>6</sup> Meanwhile, neurologists increasingly acknowledge the following: 1) psychosocial interventions are effective in neurological diseases,<sup>6</sup> 2) comorbid or even underlying psychiatric symptoms in neurological disorders are often underdiagnosed by neurologists,<sup>13</sup> 3) placebo effects exist in a

range of neurological diseases despite established causal neuropathology<sup>12</sup> and 4) a biopsychosocial perspective for clinical neurology has proven adequate in studies on almost all neurological diseases, to a greater or lesser extent.<sup>6</sup>

## CONCLUSION: TOWARDS AN INTEGRATIVE, BIOPSYCHOSOCIAL VIEW OF BOTH PSYCHIATRY AND NEUROLOGY

Psychiatry and neurology are not easily distinguished, and neuropathology does not allow a sharp distinction, either.<sup>14</sup> Geriatric psychiatry best illustrates the entanglement of both disciplines: Sequelae of neurological diseases (such as stroke or neurodegeneration) often ‘melt’ with mental disorders (such as psychosis or depression) and effects of long-term medication, sometimes over a life course. Differences between neurology and psychiatry may be due to their historical origins and to different traditions of teaching, disease conceptualization and treatment. However, they are neither due to Cartesian dualism nor to the mechanistic-organismic dichotomy. Mental disorders are not ‘brainless’, but they call for an integrative, biopsychosocial view, as neurological diseases do.

The impression that biological mechanisms in the literal sense exist results from laboratory experiments of fundamental neuroscience, where an organismic context can be either eliminated or controlled for.<sup>4,5</sup> With its fatal adherence to ‘brain mechanisms’, BP further limits itself to overly fine-grained levels of explanation a priori, even dismissing others as unscientific.<sup>3,7</sup> Any level of explanation, however, must be chosen pragmatically, in order to encompass the maximal range of therapeutic interventions. Take depression as an example: from the patient’s description, from clinical observations and expert knowledge we derive hypotheses about causal relationships that might be effective in producing and maintaining the depressive symptoms. We may then derive therapeutic interventions from different levels of explanation: pharmacological (e.g. SSRIs), neurophysiological (e.g. electroconvulsive therapy), intrapsychic (e.g. dysfunctional cognitions), interpersonal (e.g. conflict management), social (e.g. employment situation) etc. depending on resources and comorbidities of the patient, his/her social situation, resources and competencies of the therapists, availability of treatments etc. Such a pragmatic, multidimensional approach is common for actual clinical practice in psychiatry.

Contrary to the view of many BP proponents, there is no reason to assume that more fine-grained levels of explanation are more adequate per se. We do not need to know particle physics in order to build safe cars, and in the same way, the effectiveness of psychotherapy does not have to be demonstrated

on a neuronal or even molecular level. The adoption of such a reductionist view had long been identified as a general problem of the medical model of disease by Engel<sup>15</sup> in favor of his biopsychosocial, organismic approach.<sup>6</sup> Such a perspective would allow for a non-literal and non-reductionist understanding of cause-effect-relationships on any level of explanation- and therapeutic intervention.<sup>16</sup> A biopsychosocial view is needed for both psychiatry and neurology in order to bring them closer together conceptually. More importantly, however, this view allows to develop the best possible treatments for our patients.

### Availability of Data and Material

Data sharing not applicable to this article as no datasets were generated or analyzed during the study.

### Conflicts of Interest

The author has no potential conflicts of interest to disclose.

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