Are the neurocognitive correlates of subtle subjective symptoms the way forward in psychiatry?

TILO KIRCHER

Department of Psychiatry, Marburg University,
Marburg, Germany

In their thoughtful overview, Sass and Byrom (1) correlate three levels of description: the subjective experience of the patient as understood by a third person (phenomenology), cognitive models and neuroscience. In particular, they link two diverging models with each other, i.e., selected phenomenological accounts of delusions and their potential underlying neurocognitive explications. They identify correlations between the two models and crystallize neglected areas in neurocognitive accounts, filling them with phenomenological notions. Basically, they argue to look for fine grained, subjective symptoms – beyond structured interviews and DSM/ICD criteria – and to relate them to neurocognitive data.

The authors' ambition is to find a neuroscientific explanation of subjective experiences. However, neuroscience should not be a validator for phenomenology, as they are two different realms of description, which should inform each other. There is of course a fundamental "explanatory gap". The subjective consciousness (first person perspective) has to be translated by the patient into a second person "output", usually verbal and non-verbal; this has to be understood by the interviewer and then related to brain states (third person perspective). The mind-body relation is a yet unresolved philosophical question; therefore we can only correlate such levels of descriptions, but we cannot explain them with one another.

Correlating symptoms with brain states has already been successfully achieved by a neuroimaging "symptom catching" approach, mainly for auditory hallucinations and formal thought disorder. In the beginning, cruder syn-

dromes have been related to brain activation (2,3), while recently a more fine grained and subjective psychopathology has been applied (4,5).

In the article, highly selected phenomenological ideas (models) are correlated with current neuroscience models. Since there is not much replicated data on the neurobiology of delusions, Sass and Byrom's account is rather a comparison of models. Ideally, one would have a phenomenologically derived symptom or experience and relate this to empirical data, rather than comparing models with each other. More empirically testable predictions from phenomenology would be welcome.

However, this begs the question of which among the many phenomenological accounts (Sass and Byrom only discuss a few) should be translated into experiments and tested. For current neuroscientific data acquisition, a fluctuating phenomenon/symptom is usually considered, and the patient is investigated experimentally at two or more points in time, with symptom severity being correlated with cognitive measures and/or brain states. This methodological constraint vastly reduces the number of phenomena possibly investigated.

A further current problem is that the conception of our patients' inner lives has been oversimplified by the operationalized diagnostic approaches. This oversimplification has been accompanied by a reliance on methodologies that are unable to capture the delicate forms of human experience and expression (6). If the cognitive scientist wants to base experiments on phenomenological notions, he or she must be deeply familiar with the philosophical-phenomenological literature. This needs thorough reading and understanding of the ideas, which are currently marginalized. Many French and German texts have not been translated into English and are

therefore inaccessible to a wider international audience. For example, Sass and Byrom discuss concepts by K. Conrad, but another important phenomenological contribution on chronic, negative symptoms stems from W. Blankenburg (7), both from Marburg. Their two main books have equally not been translated into English.

The clinician should be familiar with the rich descriptive and hermeneutic tradition of psychiatry. We only see in our patients what we know. There are other levels for the explanation of experiences than cognitive psychology and neuroscience, and a deep understanding of the patient's world is interesting and therapeutic in itself. A phenomenologically informed systems neuroscience, integrating environmental, genetic, molecular and brain imaging data in the same patient across his life, will eventually explain the etiology and pathophysiology of mental disorders. It can, however, not replace an interpersonally shared understanding of the patient's inner life as a humane value and source of content in itself.

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Phenomenology is Bayesian in its application to delusions

AARON L. MISHARA¹,
PHILIPP STERZER²

¹Department of Clinical Psychology, Chicago School of Professional Psychology, Los Angeles, CA 90017, USA; ²Department of Psychiatry and Psychotherapy, Charité-Universitätsmedizin, D-10117 Berlin, Germany

Sass and Byrom (1) argue that phenomenology “expands the range of testable hypotheses”. This resonates with our view that phenomenology “leads to neurobiological hypotheses, which can be tested experimentally” (2,3). It is also a welcome modification of Sass’ proposal (4) that phenomenology “serves an explanatory function”. If phenomenology “explains” schizophrenia by proposing its core essence as a disturbance of “hyper-reflexivity/ipseity” (4), it claims knowledge about causal relationships without recourse to testing hypotheses about mechanism.

The authors see a conflict between “enactive” or “embodied” approaches to cognition and “more intellectualistic sounding” prediction-error formulation. We suggest that this apparent conflict is related to a misunderstanding of the term *beliefs* in predictive coding accounts. In current accounts of Bayesian hierarchical predictive coding, a belief is considered merely a probability distribution over some unknown state and may or may not be consciously accessible (5). A central claim of hierarchical predictive coding models is that such beliefs are fundamentally embodied even at the lowest levels of sensory processing, clearly not implying intellectual conjecture and refutation. Accordingly, studies of patients with schizophrenia

point to an alteration of predictive mechanisms at low levels of sensory processing. Behavioral and functional neuroimaging studies of illusory visual perception in schizophrenia patients have suggested impaired predictive mechanisms in early visual cortex (e.g., 6,7). Similarly, mismatch-negativity (MMN), an electrophysiological signal that is thought to reflect the automatic registration of irregularities in sensory input, is reduced in patients with schizophrenia (8). The empirical evidence for altered predictive coding seems to contradict the authors’ assumption that the predictive mechanisms involved in delusion formation/maintenance necessarily implicate, or are limited to, cognitive or “intellectualistic” processes.

Furthermore, the authors suggest that the exaggerated prediction-error signaling giving rise to *hypersalience* does not account for *hyposalience* and an associated “anything-goes” attitude, which they propose may be due to a dysfunction in the default-mode network. Apart from possible problems with “reverse inference”, we question the assertion that *hyposalience* as described by the authors is incompatible with the notion of prediction-error dysfunction. To the contrary, predictive coding accounts actually predict that the proposed exaggerated prediction-error signaling (or imbalance in the precision of prediction errors and prior beliefs) (5) results in an impaired distinction between normally expected and unexpected events. This is exemplified by reduced MMN amplitude in schizophrenia conceptualized as a consequence of altered prediction-error signaling. In this context, attenuat-

ed mismatch responses in schizophrenia patients may actually not reflect the failure to register surprising events, but rather the fact that each event is surprising (5,7). Hyper- and hyposalience are two sides of the same coin, accounted for by a single factor, prediction-error dysfunction (9).

This is supported by Heidelberg psychiatrist Mayer-Gross’ (1932) observation of reduced anticipatory expectation in the “self disturbances”, due to the ongoing “interruption” of current goal-processing by the “made” or influenced perceptions, movements, thoughts, etc., which characterize those disturbances (10). There is only the compelling sensory evidence of *now*: “no temporal order prevails, each sensory impression is equally valued, replacing its predecessor”. This reduction in top-down, embodied perceptual expectation in the “self disturbances” observed by Mayer-Gross anticipates the predictive coding account of attenuation of visual illusions (e.g., the hollow-mask illusion) in schizophrenia and how this relates to delusions and related symptoms (as discussed by Corlett, Fletcher and Frith, and others).

The phenomenological psychiatrist Binswanger also described the self in schizophrenia as captive in the present moment in a “temporal shrinking” of past and future which resembles dreaming (11). In his fiction, Kafka depicts the reduced expectation in dreamlike-hypnagogic experiences, where protagonists report “expecting” the very events that “surprise” them (12). This is not “bizarre-as-banal”, but *the absence of banal altogether*. It is also not “anything-goes”, but can be formalized in the Bayesian hierarchy as outlined above. Similarly, Binswanger