

## Artigo Convidado/Invited Article

### The hermeneutics of mental symptoms in the Cambridge School

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*Current Psychiatry is in crisis. Decades of neuroscientific research have not yet delivered adequate explanations or treatments. One reason for this failure may be the wrongness of its central assumption, namely that mental symptoms and disorders are natural kinds. The Cambridge School has proposed that a new Epistemology must be constructed for Psychiatry, and that this should start with the development of a new model of mental symptom-formation. 'Mental symptoms' should be considered as hermeneutic co-constructions occurring in a intersubjective space created by the dialogue between sufferer and healer. Subjective experiences (caused either by neurobiological or psychosocial upheaval) penetrate the awareness of sufferers causing perplexity and/or distress. To understand, handle and communicate these experiences, sufferers proceed to configure them by means of templates borrowed from their own culture. Importantly, however, the same neurobiological information can be configured into different symptoms; and different neurobiological information into the same symptom. Therefore, 'mental symptoms' are dissimilar hybrid combinations of neurobiological and cultural information. To be ethical, therapeutic interventions must take into account such dissimilarities. Blind manipulation of the brain in all cases should be considered as counterproductive.*

**Key words:** Hermeneutics, phenomenology, psychopathology, epistemology

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## Introduction

Current psychiatric nosology entered in a state of scientific crisis due to several internal anomalies, i.e. seeming empirical shortcomings directly deriving from the way the system is structured and research is consequently designed (Aragona, 2009, 2014a). Accordingly, it was claimed that

the neo-Kraepelinian paradigm established by Robins and Guze and institutionalized in the DSM has resulted in so many problems and inconsistencies that a crisis of confidence has become widespread. [This drives] a transition from a period of normal science (where the paradigm serves as an integrating framework in which questions are asked and answered) to a period of *extraordinary science*. The defining features of the fragmented periods called extraordinary science include a) a lack of agreement on what are the most appropriate methodologies, b) magnification of the problems that define the crisis into the most important problems of the discipline, c) the generation of speculative new theories, and d) a dramatic increase of interest in exploring the philosophical assumptions of the discipline. (Zachar & Jablensky, 2014, p. 9-10)

Research in the epistemological history of psychiatry suggested that one key feature responsible for the current crisis was the implicit theoretical assumption of the 'atheoretical' DSMs that mental symptoms were facts to be merely observed at a purely descriptive level (Aragona, 2013a). It was argued that in clinical practice the act of 'diagnosing' is not based on such a bottom-up neopositivist abstraction, from the descriptive level of mental symptoms to the inferential level of diagnosis through the application of impersonal operational diagnostic criteria. On the contrary, it was suggested that 'diagnosing' relies on a hermeneutic circle where the parts (mental symptoms) and the whole (the psychiatric diagnosis) are in mutual relationship (Aragona, 2013b).

This hermeneutic standpoint is based on the idea that mental symptoms are not mere facts, i.e. objects that are simply ‘given’ and that can be directly described as such. Rather, they are co-constructed in the therapeutic relationship. This is how the Cambridge School (a collective name for several researchers interested in the history and epistemology of psychiatry) conceived the formation and meaning of mental symptoms (Berrios, 2013, 2014).

In this paper, we will present the main ideas of the Cambridge School about mental symptoms. In particular, we will discuss their four hermeneutic models of mental formation. Finally, some consequences for clinical practice and research will be considered.

### **The construction of mental symptoms**

Psychiatry partakes in the human and natural sciences. In different times their relative influence varies although both are always operative. With the exception of the fundamentalist extremes (physicalist eliminativism at one side, idealism and radical social constructivism at the other side), the vast majority of psychiatrists argue for models considering the possibility of an interplay between these two levels. However, one thing is to assert in general that both human and natural sciences shall be involved, and a totally different thing is to elaborate concrete models of how such interaction may take place. Following Jaspers (1946/1963), generations of psychopathologists continued to hold a sharp separation of methods (law-like explanations in the natural sciences, empathic understanding in the human sciences), leading to methodological pluralism, or multiperspectivism (Rosini et al., 2013). A different view appeared in the 1980’s, when American psychiatry definitively abandoned previous models of interplay between the physical level and the plane of meanings, and substituted them with the new view that an interpretation mainly occurs when objective mental symptoms are clustered in diagnostic categories (according to Spitzer, 2001, it is the act of diagnosing which is highly inferential). On this view such subjective interpretation is regarded with suspicion because it is considered as the main source of diagnostic unreliability. Three consequences were derived from this: first, mental symptoms were conceived as observable objects that are given in nature as we see and describe them; second, subjective interpretation was seen as a pejorative act because it undermines scientific rigor; and third, it was suggested that this ‘problem’ could be avoided by using a-priori defined operational diagnostic criteria (Aragona, 2014b). This model led to forty years of biologically oriented empirical research based on the view that symptoms were objective facts and that subjective interpretation was a danger that psychiatric science had to avoid at any cost. In this context, researchers

appeared satisfied to use their statistical analyses to correlate biological variables (genetic profiles, neuroimaging, psychopharmacological effects etc.) to numbers derived from the assessment of mental symptoms through common rating scales. The current crisis within psychiatry shows that this model is not working well.

The Cambridge group puts forward the view that the failure of the model is directly related to the assumption that mental symptoms are natural kinds. On this assumption, research approaches borrowed from the natural and biological sciences have been adopted to try to capture, describe and even define mental symptoms (e.g. Cuthbert & Insel, 2013). This has encouraged and perpetuated the narrow conception of mental symptoms as brain events or entities and any contribution of the human sciences, and hence interpretation, to the understanding of mental symptoms has been dismissed or included as a non-participating addendum. However, if psychiatry is conceived as truly partaking of both natural and human sciences then the very structure of psychiatry and its objects, namely, mental symptoms and mental disorders will be constituted in a deep sense from elements of both. This means that in order to make sense of psychiatry, we need to understand why and how the current language and objects of psychiatry were constructed (Berrios, 2014; Marková & Berrios, 2012). In turn, this will allow us to determine the type and extent of interpretation involved in defining and describing the objects of psychiatry. Challenging the assumption that mental symptoms are natural objects which can be reliably grasped at purely descriptive levels, without the need of interpretative skills, this paper focuses on two main questions: what kind of objects are mental symptoms, and are they subjected to interpretative elaboration (hermeneutics) before being fixed in their final form?

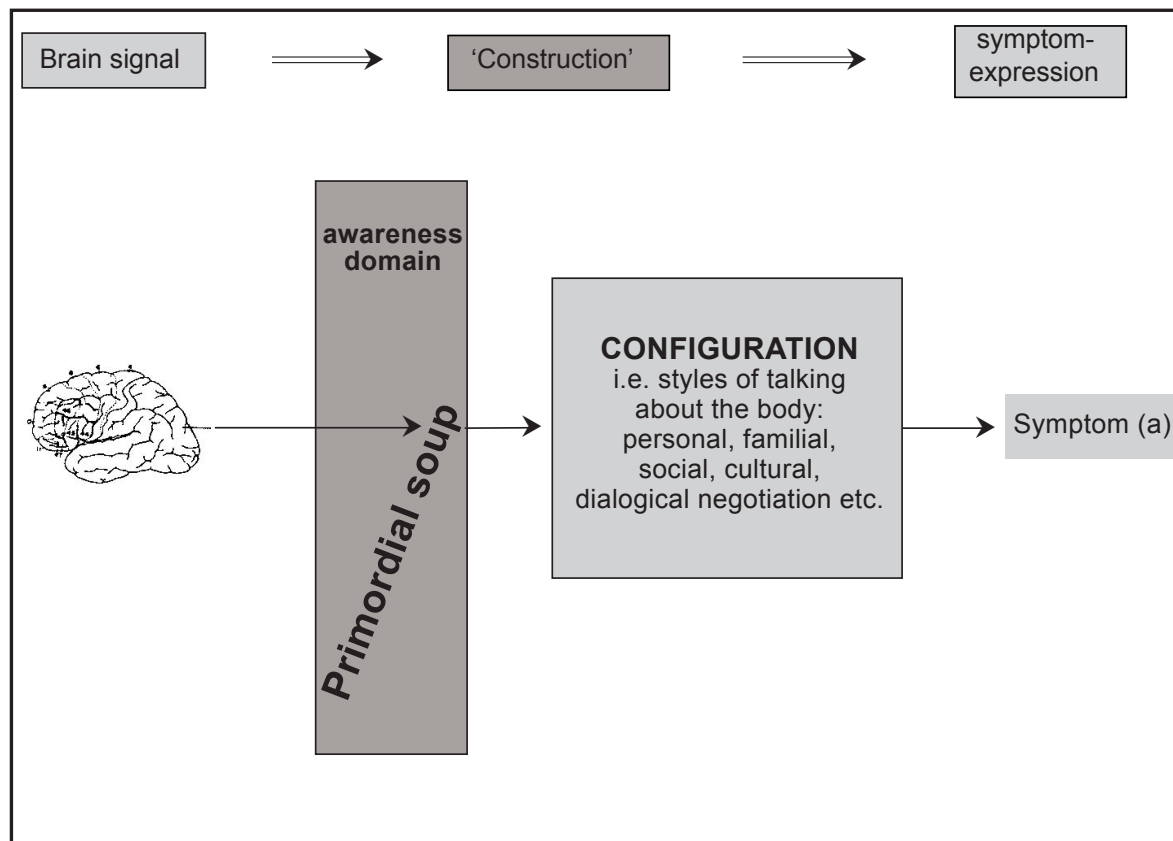
As the 'units of analysis' of psychopathology, 'mental symptoms' include (i) subjective complaints (e.g. feeling anxious or depressed, hearing voices, etc.) and (ii) signs and behaviors determined through observation or instruments (e.g. psychomotor retardation, cognitive deficits, disinhibition, etc.). The Cambridge school maintains that mental symptoms are heterogeneous in terms of their origin and structure, and that this heterogeneity tends to be ignored both clinically and in research (Marková and Berrios, 1995; 2009). This can be illustrated by looking at ways in which symptoms might arise. In this regard, four pathways of symptom formation have been postulated where nature (neurobiological activity), personal capacities and narratives, familial and social idioms of distress, and interpersonal negotiation of meaning, are all operative and intertwined at different levels. Depending on the way symptoms arise and are configured, their structures will vary in terms of the extents to which the biological and the semantic factors mentioned above will contribute to their formation (Berrios, 2013, 2014; Marková & Berrios, 1995; 2012).

The first group of mental symptoms, namely, subjective complaints (e.g. anxiety, depression, anger, suspicion, thoughts of being followed and/or persecuted,

experiences of loss of control, hearing voices etc.) can be spontaneously offered or are elicited by the clinician. In both cases, they are reported by the subject as something happening in her internal sphere of consciousness. Being private experiences, they cannot be verified by means of external objective verifiers (in the same way that, for example, a temperature change can be verified). Moreover, it is impossible to verify the *fit* between the name used to refer to the experience, and the experience itself. Hence, different patients may give different names to their experience, depending on different personal and cultural variables.

Introspective reports carry several difficulties, theoretical/epistemological as well as practical. Here we focus on the latter. In order to complain about an experienced mental symptom, subjects have firstly to identify the particular experience. This requires an ability to identify, differentiate and denominate a given experience. Cultural ways to perform this ‘configuring’ activity are apprehended during personal development, with some degrees of variability even within the same cultural or familial context. Thus, personal, familial, and socio-cultural factors cooperate in shaping the so-called ‘idioms of distress’. Some patients report their distress in the symptomatic form which is more usual and ‘expected’ in their socio-cultural context. Other patients may configure similar experiences in a more idiosyncratic modality. In both cases, the same original experience is shaped according to personal and cultural factors. The most striking case is that of completely new experiences, as it is the case for several mental symptoms (e.g., those considered ununderstandable and processual by Jaspers). A good example is the so-called *Wahnstimmung*, i.e. the pre-delusional state when things have lost their usual, commonsensical and obvious sense, their meaning is suspended, and the subject lives in the sinister feeling of something yet unknown, but possibly dreadful, taking place. In such a situation, as in any other case when the abnormal experiences are completely new, the patient may find these difficult to handle. An initial bewilderment will be followed by efforts to make sense of the new experience in terms of available categories, and it is in this configuring process that the final complaint is shaped. As stated, personality factors, education, imagination, adaptive capacities, socio-cultural factors etc. will guide this activity, which is considered as an hermeneutic process: i.e., a sense arising from the interpretative process starting from initial unstructured experiences. In other words, whether a particular subjective experience is articulated as a depressed mood, a feeling of fatigue, a particular pain or even a sense of dread, etc. may depend not only on some basic neurobiological activation that triggers the process, but also on non-biological, personal and cultural factors that configure the signal away from its original biological hallmarks. The Cambridge school called this modality of symptom formation ‘pathway (a)’, which is illustrated in figure 1 and is composed of the following steps.

**Figure 1**  
**Cambridge Model for Symptom-Formation: Pathway (a)**



***Pathway (a):***

By definition, subjective complaints are those mental states about which the individual is aware. Thus, in order to complain of low mood or apprehension or hearing voices etc., he/she has to do this on the basis of some interpretation of an internal experience. The cause of such an experiential change matters little at this point. It could be a spontaneous brain activity or it might be secondary to some acute stress, trauma, brain disease or ongoing pressures in one's life or combinations of many such things. Irrespective of cause, there must be some change experienced in awareness and this early conscious experience is called by the Cambridge group a 'primordial soup'. It is conceived as a formless, pre-linguistic and pre-conceptual experience lived by the patient with raw immediacy. The subject is aware of something happening in her, but at this stage it is an inchoate proto-experience. What then are the factors that will contribute to the transformation of this inchoate experience into a 'subjective mental symptom'?



At the first stage, factors around the development of the primordial soup itself will be important in the preliminary configuration. Here, the *rate* at which the experiential change develops, the *context* in which it occurs and the *quality* of the change will all play a part in how this experience will be configured. For example, a change that builds up slowly may draw on more sources such as memory or knowledge than something that occurs rapidly. Or, a primordial soup that shares some familiarity with known experiences may be interpreted more easily than something that from the beginning is alien. The transience or persistence of the change as well as concomitant experiences are also likely to affect the way in which this initial experiential change will start to be configured.

At a second stage, factors relating to the individual and his/her sociocultural background will be important in configuring the changes he/she is experiencing in awareness. This stage may be considered as a first hermeneutic step, i.e. a self-interpretation that the patient performs by subjecting the experience to a set of configurators which include personal, familial, social and cultural styles of shaping and naming experiences. For example, relevant here will be factors such as past experiences, personality traits, general intelligence, education levels, peer pressures, media influences, language skills and many more. Thus, a tendency to introspection might generate more detailed and coloured descriptions of some experiences; the level of education might determine the range and type of vocabulary chosen to express the experience; a culture discouraging emotional referents might prompt a more 'cognitive' or 'somatic' description of the internal experience and so on.

The third stage (and second hermeneutic step) involves the interactional influences that will play a part in the configuration of the experiential change into a 'mental symptom'. Here the interlocutor may play a fundamental role, the pragmatics of the interaction with the clinician (or with someone else) influencing the formation of the articulated symptom. In other words, particularly where it is perhaps difficult for an individual to define or make sense of a particular experience, the interlocutor may strongly contribute to this shaping of experience both through direct suggestion as well as by a process of joint construction/negotiation. This is particularly relevant when clinical interviewing may actively help the subject to disambiguate complex subjective experiences. Accordingly, it must be stressed that working diagnostic hypotheses may introduce important biases in the way in which the clinical interviewer helps the subject to re-configure the final version of the mental symptom. Hence, in this context the diagnostic act is never a neutral description but an active part of the co-construction of mental pathology.

In this way, the structure of the subjective mental symptom can be envisaged as composed of a neurobiological element enveloped in a 'semantic' element. This latter is in turn constituted by the meaning as configured by (i) individual and socio-cultural factors and (ii) interactional forces (through interaction with

others and the environment) (see figure 3). So the crystallized and fully configured symptom is not a mere 'object' but the complex product of this interplay of multiple factors.

### ***Pathway (b):***

606 The second group of mental symptoms are the observable signs and behaviours, e.g. flight of ideas, disinhibition, psychomotor retardation, neologisms, tardive dyskinesia, and so on. In this case it is the clinician that observes, identifies and names them, the patient not being necessarily aware of them. In such cases, the Cambridge school postulates that the signal bypasses consciousness and directly results in the consequent behaviour/utterance. In other words, the signal suffers less semantic enveloping or processing than in the case of signals processed by pathway (a), symptoms therefore having a more direct relationship to their neurobiological signal. However, here it is the clinician who may influence to some extent the formation of these symptoms as symptoms. Thus, factors relating to the clinician will be important here in terms of whether and how the clinician identifies and names the 'symptom'. For example, whether a clinician identifies a sign such as affective blunting might depend not only on the explicitness with which it is presented but also on the clinician's past experience, her knowledge and biases, current mental state (e.g. level of concentration), etc. In sum, symptoms of pathway (b) are more directly related to the basic neurobiological signal and are subject to less semantic configuration than those of pathway (a). However, even in this case some semantic shaping occurs, the clinician having an important role in this respect.

### ***Pathway (c):***

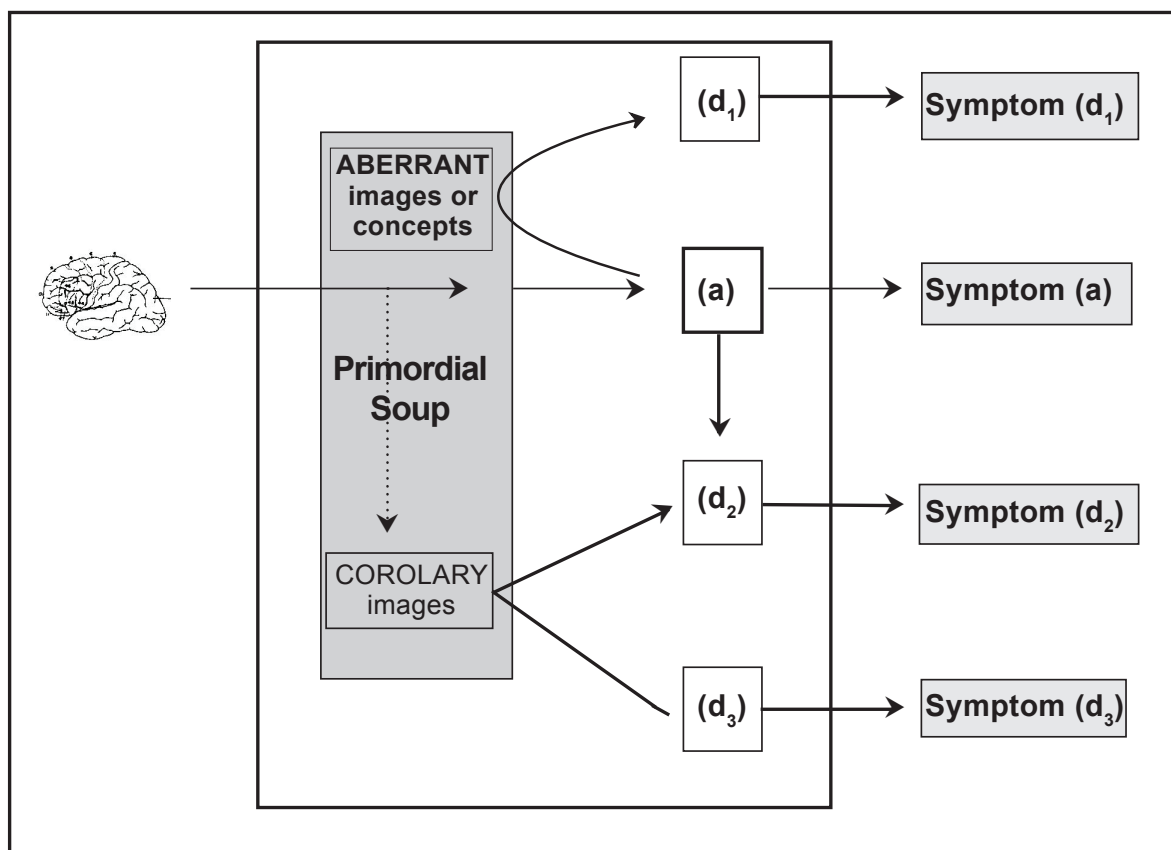
In some cases neurobiological signals (primary or secondary) may be associated with inchoate consciousness states (primordial soups) which are simply not configured by the individual, in the sense that they are not expressed as new symptoms. This could be because the primordial soup is so ephemeral that the subject lacks the time to become fully aware of it, or signals may eventually dissipate before they are configured. On the other hand, the primordial soup may not fit existing categories of description held by the individual and hence the experience remains unnamed. Other experiences might be fitted into already available stereotyped formats and expressed as already known symptoms, independently of their different pattern of formation.



**Pathway (d):**

Not every mental symptom needs to be configured starting from the original neurobiological-primordial soup complex. Some symptoms may be viewed as ‘secondary’ constructions originating from the experiential change induced by another ‘primary’ symptom. For example, in his original conceptualization of the group of schizophrenias, Eugen Bleuler (1911) distinguished between fundamental and accessorial symptoms concerning their diagnostic value, and between primary and secondary symptoms depending on their dynamics of formation. It is the latter distinction which is involved in pathway (d). For example, in Bleuler the disturbance of associations is primary, while the disorders of affectivity are a reaction secondary to this basic disturbance. Similarly, the Cambridge group sees as secondary the ‘anxiety’ that may develop as a reaction to a frightening hallucination.

**Figure 2**  
**Cambridge Model for Symptom-Formation: Pathway (d)**



**d1-d3 give rise to three types of ‘BEHAVIOURAL PHENOCOPIES’ of mental symptoms**

In pathway (d) symptoms are constructed on the basis of other symptoms which themselves might trigger changes in consciousness and hence second-order configuration. Again, there may be a number of mechanisms involved (Figure 2) and the factors involved in the formation of subjective complaints in pathway (a) (e.g. cultural values, past experiences, familiar idioms of distress, etc.) will likewise be important in the formation of these secondary constructs in pathway (d). It is noteworthy that while in Bleuler's model primary symptoms are usually conceived as directly arising from neurobiological activity, and only his secondary symptoms are formed through semantic/psychological mechanisms, in the Cambridge model the primary symptoms themselves are already semantically shaped according to the dynamics of pathway (a). Accordingly, in the Cambridge view both primary (pathway (a)) and secondary (pathway (d)) symptoms are semantically constructed. The difference is that the former are associated more directly with a primary neurobiological signal. Finally, it is also possible that some secondary constructs arise not on the basis of changes in consciousness triggered by primary constructs but as intellectual or cognitive (rather than experiential) responses to the primary experiences. What seems evident is that the relationship between symptoms formed along pathway (d) and any postulated brain signal is indirect and even more blurred than in pathway (a).

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To conclude this section, we shall stress that the pathways analysed above (a, b, c, and d) were described as distinct, individual processes. This is a necessary simplification for the sake of analysis but in real life it has to be understood that multiple interactions are likely to occur (i.e. one pathway will influence the other, etc.). Furthermore, symptoms do not arise in isolation and further interactions will naturally occur in the context of the interplay between concomitant symptoms.

### **Some clinical and research consequences**

The Cambridge model described above suggests the existence of various factors that are crucial to the formation of symptom and are itemized in the following list (Berrios, 2014).

- a) nature and intensity of the neurobiological signal,
- b) rate of duration of the experienced primordial soup,
- c) degree of novelty of the primordial soup (i.e. matching or mismatching with relevant memory or cognitive templates),
- d) the patient's state of consciousness, attributional attitudes, general knowledge (i.e. theories and attitudes about the world),
- e) cognitive and emotional frame in which the primordial soup occurs,
- f) capacity to name and classify subjective experiences,

- g) cultural and pragmatic context (i.e. what else does the patient want to do and say in addition to naming his symptom),
- h) individual capacity and inclination to make sense of an experience,
- i) biases introduced during the mental state assessment which may lead the subject to have his/her experiences disambiguated in particular ways.

If mental symptoms are often individual interpretations of personal (often blurred) experiences, then they are in effect *inter-personal constructs*. They are *constructs* in the sense that subjects construct a meaning out of rather inchoate pre-linguistic experiences. They are *personal* because the experience is lived as unique or personal to the individual, and is accessible to others only indirectly and hermeneutically. They are *interpersonal* in that they are both a) strongly influenced by social and cultural factors, which help to shape the specific way in which the subject makes sense and articulates the experience, and b) co-constructed together with the clinicians and/or other persons that talking with the patient assist and influence her in shaping and naming the experience. Mental symptoms can thus be viewed as elaborated by patients and co-elaborated with others, particularly with psychiatrists in the context of a clinical setting.

Thus, the first point to emphasise for clinical practice and research is that mental symptoms are the complex products of the interplay between neurobiological and semantic (personal, socio-cultural and dialogical) factors. Consequently, reductionist approaches ignoring the major hermeneutic components in their structures will clearly be unable to adequately capture mental symptoms in a valid sense. Clinicians need to be aware of this. The Cambridge approach is consonant with bio-psycho-social models, although it adds to previous contributions in this field a coherent model of how mental symptoms are formed. In other words, its claim is not limited to asserting the principle of the multilevel interaction, but provides a concrete model of the way such interactions may actually work.

The second consequence is that following the above pathways of formation, the often observed heterogeneity of mental symptoms is clarified (Berrios, 2013, 2014; Marková & Berrios, 2009, 2012). This point can be better elucidated by analysing the single pathways. In pathway (a), a neurobiological kernel is progressively ‘enveloped’ and ‘shaped’ by several levels of semantic configuration. It thus becomes possible that, firstly, the same basic neurobiological signal can result in different mental symptoms and, secondly, the ‘same’ symptom can be associated with different brain signals.

In the first case, on account of the differences between individuals in terms of past experiences, socio-cultural variables, capacities, dialogical influences and so on, a similar sort of ‘primordial soup’ could be configured differently thereby giving rise to different mental symptoms. For example, a particular unpleasant internal state might be interpreted as depressed mood in one individual while the ‘same’ or similar

primordial soup might be interpreted by another individual as anxiety or fatigue or pain. This means that, as mentioned above, it is possible that different constructions might issue from the same primordial soup if individuals are from different social/cultural backgrounds, if they have different views and ideas about the world, if they are in different mood states, or indeed if the context of the experience is different (e.g. in a conversation with a neighbour, with a clinician or with a stranger).

In the second case, different primordial soups (with different associated brain signalling), as experienced by different subjects, could be configured into the 'same' symptom. For example, similar feelings of low mood, fatigue, lack of motivation, etc., may be produced in response to a brain tumour, an initial dementia, or major depression. Hence, different sorts of lesions or dysfunctions can result in similar internal experiences and consequently configured as the 'same' symptom. Another possibility is that different brain signals produce different internal experiences, but the patient interprets such different primordial soups by using the same configuring categories, hence resulting in the 'same' symptoms. For example, someone with limited experience or vocabulary may interpret different states of 'sadness', 'emptiness', 'gloominess', 'tension', 'irritability', etc., as a generic feeling of 'depression', independently from their different origin and experiential nuances. This last possibility also characterizes those 'pathway (c)' symptoms that are expressed through preformed and already available formats. Finally, similarity can also be generated by the interviewer. For example, a patient may report slightly different feelings but the clinician does not know the corresponding differentiation in general psychopathology; i.e. many young clinicians trained with the DSM are familiar with ample and commonsensical definitions of symptoms and often ignore the qualitatively nuanced differences established by classic psychopathologists. Alternatively, the clinician may sense there is a difference but the structured interview he is using forces him to place the phenomenon in a preformed and rigid category, hence neglecting the nuance (the so-called Procrustean effect).

In the case of pathway (b), the relationship between neurobiological signals and the resulting behaviour is more direct, less influenced by hermeneutic envelopes. In this case heterogeneity is expected to be reduced, but only if the neurobiological signal carries a specificity. The above discussed level of heterogeneity introduced by the interviewer is operative also in this case: i.e. the same behaviour or facial expression may be interpreted differently by the clinician depending on her own knowledge and interpretative skills.

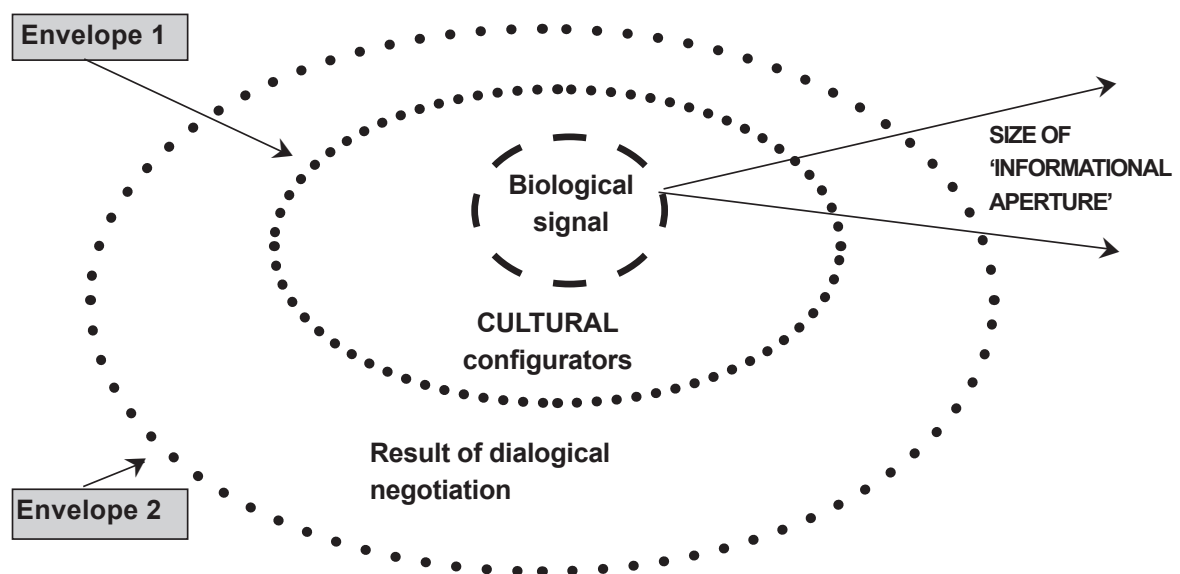
Finally, in pathway (d) symptoms are entirely produced at the semantic level, leaving space to huge interpretative differences depending on all the factors reported above.

To sum up, mental symptoms are structurally different in relation to their different process of construction, and this heterogeneity needs to be acknowledged in clinical practice and research.

Finally, the consequences of this model for current neuroscientific research must be considered. There is little doubt that the recent impressive development of neuroimaging and neurophysiological technologies has resulted in a better knowledge of brain structure and function. However, this has raised new epistemological challenges. In particular, the capture of brain function, especially the higher neurocognitive functions, such as memory or problem solving, is conceptually problematic (Uttal, 2004). And, when it comes to capturing mental symptoms, such conceptual difficulties simply multiply. The common claim that it is possible to 'localize' mental symptoms raises several questions. For example, to what extent does it make sense to try to capture mental symptoms using techniques designed to capture physical structures and physiological processes? Or, in other words, are mental symptoms reducible to neurocognitive function 'without residual'? And if a relationship between brain addresses and mental symptoms exists, is it a one-to-one correspondence so that we can hope to discover a specific neurocognitive alteration for every different mental symptom? The Cambridge model of symptom formation discussed above suggests that the relationship between neurophysiological variables and mental symptoms is much less direct and linear than commonly claimed. As described earlier, in this model mental symptoms are conceived as complexes of neurobiological and 'semantic' (individual, socio-cultural and dialogical) elements. These can be represented by a structure consisting of a neurobiological kernel surrounded by configuring envelopes (Figure 3). Here a key concept is that of 'size of informational aperture' (Berrios, 2013).

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**Figure 3**  
**Semantic wrappers and informational aperture**



According to this concept, even when a neurobiological signal is the starting point of the constructive process, the degree of its correspondence to the final mental symptom is variable. In general, the model suggests that many different kinds of factors are likely to influence the interpretation of a particular internal state. And, the construction of such semantic envelopes precludes a specific and direct relationship between a 'final mental symptom' and a particular neurobiological signal. We can take a closer look at this relationship by examining the different pathways of symptom formation. It would appear that the most favorable condition for a relatively direct relationship between mental symptoms and neurobiological signaling would be along pathway (b). Here, the relative lack of configuration on the part of the patient means that mental symptoms are generated as more or less direct expressions of brain signals. In such cases we have the highest size of informational aperture, and neurocognitive inquiry is likely to reach its best results. Of course even in this case there will be configuring influences on the formation of the symptom but these will come mainly from the clinician exploring the mental state of the patient (and hence depend on descriptive abilities, negotiations of meaning with the patient etc.). However, the effect of this possible 'distortion' may be less relevant than in other pathways. The opposite extreme is pathway (d). In this case, more or less configured symptoms are reprocessed in a second-order configuration process leading to yet another symptom. This double processing and the absence of a direct or relevant brain signal makes symptoms generated via pathway (d) totally dependent upon the cultural contingencies. Accordingly, in this case the role of neurobiological activity is at best epiphenomenal, and employing neuroimaging techniques to find out the brain localization of this symptoms looks unpromising. Finally, in pathway (a) (and partly (c)) semantic factors play a significant role in symptom formation. Here the brain signal is only one component of the final symptom, whose formation is culturally, socially, and personally shaped by both the patient and the interviewer. Accordingly, the original brain signal being subject to several levels of semantic reconfiguration, the size of informational aperture (i.e. the level of correspondence between brain signal and mental symptom) is significantly reduced. This is why the Cambridge school believes that the overt features of a crystallized and fully configured symptom of this kind tell very little about the original signal and its brain address. Hence, in such cases the existence of a direct relationship between neurobiological addresses and mental symptoms is unlikely because the factors influencing the interpretation and articulation of the original primordial soup associated to such addresses would at best render the relationship tenuous. In conclusion, due to their different pathways of construction and the different role of semantic modulators in their formation, not all mental symptoms lend themselves to brain localization, and hence to neuroimaging research (Berrios, 2014; Marková & Berrios, 2014).



## Conclusion

Research in psychiatry, psychology and psychopathology cannot be limited to neurobiological approaches. Human subjectivity is a fundamental part of their scientific field of inquiry. This means that scientific formalization and human understanding are intrinsic and inextricable components of their work. The Cambridge views presented in this paper suggest possible models of interaction between the naturalistic (brain signals) and hermeneutic (semantic configurators) factors involved in the formation of mental symptoms. Four possible pathways of symptom formation were presented, highlighting the differences involved and their consequences for clinical and research practice. The heterogeneity of mental symptoms challenges the dominant neopositivist view of mental symptoms as 'real' objects that can be directly observed and described. In contrast, based on a hermeneutic construction of mental symptoms by means of 'semantic' configurators, the Cambridge model attempts to explain why and how the heterogeneity of mental symptoms arises. Moreover, the Cambridge model suggests that for many mental symptoms the search for direct and univocal neurobiological correlates is unlikely to add to the understanding of these symptoms and can only have limited validity in terms of mapping the structures involved. The reason is that semantic modulatory factors will intervene both at the stage of the generation of the brain signal, the sensing of the primordial soup, the cultural configuration of this pre-linguistic experience, and its articulation into a speech act. The fewer the modulatory factors involved, the closer the final symptom will be to the original brain signal. The more modulatory factors, the less representative it will be to the point that nothing in the final symptom will be redolent of its original brain address. Hence, research directed at understanding such symptoms should be better aimed at developing new hermeneutical approaches which could seek to disentangle such constructive forces.

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**Abstract**

(A hermenêutica de sintomas mentais na Escola de Cambridge)

*A Psiquiatria atual está em crise. Décadas de pesquisa neurocientífica ainda não resultaram em explicações ou tratamentos adequados. Uma das razões para esse fracasso pode ser sua hipótese central equivocada, ou seja, de que os sintomas e desordens mentais são tipos naturais. A Escola de Cambridge propôs a construção de uma nova epistemologia para a Psiquiatria, que deve começar com o desenvolvimento de um novo modelo de formação de sintomas mentais. “Sintomas mentais” devem ser considerados uma coconstrução hermenêutica que ocorre em um espaço intersubjetivo criado pelo diálogo entre paciente e médico. Experiências subjetivas (causadas por convulsão neurobiológica ou psicossocial) penetram na consciência dos pacientes, causando perplexidade e/ou sofrimento. Para entender, manipular e comunicar essas experiências, os pacientes seguem para configurá-las por meio de modelos emprestados de sua própria cultura. É importante notar, no entanto, que a mesma informação neurobiológica pode ser configurada em diferentes sintomas e diferentes informações neurobiológicas, no mesmo sintoma. Portanto, “sintomas mentais” são combinações híbridas diferentes de informações neurobiológicas e culturais. Para serem éticas, intervenções terapêuticas devem levar em conta essas diferenças. A manipulação cega do cérebro deve, em todos os casos, ser considerada contraproducente.*

**Key words:** Hermenêutica, fenomenologia, psicopatologia, epistemologia

(L’herméneutique de symptômes mentaux selon l’École de Cambridge)

*La psychiatrie contemporaine est en crise. Des décennies de recherche neuroscientifique n’ont pas été capables de produire ni des explications, ni des traitements appropriés. Cet échec est peut-être partiellement dû à l’ambiguïté de son hypothèse centrale, selon laquelle les symptômes et les troubles mentaux sont des types naturels. L’École de Cambridge propose la construction d’une nouvelle épistémologie pour la psychiatrie, à commencer par l’élaboration d’un nouveau modèle de formation de symptômes mentaux. Ceux-ci doivent être considérés comme des co-constructions herméneutiques qui se produisent dans un espace intersubjectif créé par le dialogue entre le patient et le médecin. Des expériences subjectives (qui résultent de bouleversements neurobiologiques ou psychosociaux) pénètrent la conscience des patients et causent la perplexité et/ou la souffrance. Pour comprendre, gérer et communiquer ces expériences, les patients les configurent à l’aide de modèles empruntés à leur propre culture. Cependant, il est important d’observer qu’une unique information neurobiologique peut être configurée par de différents symptômes et que des informations neurobiologiques différentes peuvent à leur tour être configurées par un seul symptôme. Par conséquent, « les symptômes mentaux » sont des combinaisons hybrides différentes, composées d’informations neurobiologiques et culturelles. Pour pouvoir respecter l’éthique, les*

*interventions thérapeutiques doivent prendre en compte ces différences. La manipulation aveugle du cerveau doit, dans tous les cas, être considérée comme solution contre-productive.*

**Mots clés:** Herméneutique, phénoménologie, psychopathologie, épistémologie

(La hermenéutica de los síntomas mentales en la Escuela de Cambridge)

*La Psiquiatría actual está en crisis. Décadas de investigación neurocientífica aún no han entregado explicaciones o tratamientos adecuados. Una de las razones a las que se le atribuye este fracaso puede ser la injusticia de su supuesto central, es decir, que los síntomas y desórdenes mentales son tipologías naturales. La Escuela de Cambridge ha propuesto que una nueva epistemología debe ser construida para la Psiquiatría y que esto debería empezar con el desarrollo de un nuevo modelo de formación del síntoma mental. Los “síntomas mentales” deberían ser considerados una co-construcción hermenéutica que se da en un espacio intersubjetivo creado por el diálogo entre el paciente y quien cura. Las experiencias subjetivas (ya sean causadas por trastornos neurobiológicos o psicosociales) penetran la consciencia de los pacientes causando perplejidad y/o angustia. Para entender, controlar y comunicar estas experiencias, los pacientes proceden a configurarlas a través de plantillas tomadas de su propia cultura. Cabe destacar, sin embargo, que la misma información neurobiológica puede ser configurada en diferentes síntomas y diferentes informaciones neurobiológicas en el mismo síntoma. Por lo tanto, los “síntomas mentales” son combinaciones híbridas disímiles de informaciones neurobiológicas y culturales. Para ser éticas, las intervenciones terapéuticas deben tener en cuenta tales disimilitudes. La manipulación del cerebro debería ser considerada, en todos los casos, contraproducente.*

**Palabras clave:** Hermenéutica, fenomenología, psicopatología

(Die Hermeneutik der psychischen Symptome gemäß der Schule von Cambridge)

*Die zeitgenössische Psychiatrie befindet sich in Krise. Jahrzehnte neuro-wissenschaftlicher Forschung haben weder angemessene Erläuterungen, noch Behandlungen geliefert. Einer der Gründe für dieses Versagen könnte die Ambiguität ihrer zentralen Hypothese sein, nämlich dass psychische Symptome und Störungen natürliche Typen sind. Die Schule von Cambridge schlägt nun vor, eine neue Erkenntnistheorie für Psychiatrie zu erstellen, die mit der Entwicklung eines neuen Modells der psychischen Symptombildung beginnen müsste. „Psychische Symptome“ sind demnach hermeneutische Co-Konstruktionen, die sich in einem intersubjektiven Bereich manifestieren, der durch den Dialog zwischen Arzt und Patient geschaffen wird. Subjektiven Erfahrungen (die entweder durch neurobiologische oder psychosoziale Änderungen verursacht werden) dringen in das Bewusstsein des Patienten und verursachen Verwirrung und/oder Leiden. Um diese Erfahrungen zu verstehen, zu verarbeiten und mitzuteilen, konfiguriert sie der Patient mit Hilfe von Modellen, die aus seiner eigenen Kultur entlehnt werden. Es*

*muss hier jedoch beachtet werden, dass die gleiche neurobiologische Information in unterschiedliche Symptome konfiguriert werden kann und dass andererseits verschiedene neurobiologische Informationen mittels eines einzigen Symptoms konfiguriert werden können. Daher sind „psychische Symptome“ abweichende hybride Kombinationen, die aus neurobiologischen und kulturellen Informationen bestehen. Um der Ethik gerecht zu werden, müssen therapeutische Interventionen diese Unterschiede berücksichtigen. Blinde Gehirnmanipulation muss in allen Fällen als kontraproduktiv angesehen werden.*

**Schlüsselwörter:** Hermeneutik, Phänomenologie, Psychopathologie, Erkenntnistheorie

(精神症状在剑桥学派的解释学)

当前精神病学正处于危机之中。几十年神经科学的研究还没有提供足够的解释或处理。其中一个可能导致此失败的原因是其核心假设的不正确性：精神症状和疾病是自然种类。剑桥学派提出了需要构造精神病学的一个新认识论，而这应该开始于发展精神症状形成的一个新模式。对于“心理症状”的考虑应由患者和医者在对话时共同建设的一个主体间的空间。主观经验（由任何神经生物学或心理状态所产生的）穿透患者的意识而导致困惑和/或痛苦。为了理解，处理和交流这些经验，患者会借鉴自己文化的内容设置此经验的意义。可是重要的是，同样的神经生物学信息可产生不同的症状；和不同的神经生物学信息产生相同的症状。因此，“精神症状”是神经生物学和文化信息的杂交混合体。以道德方面，治疗者在执行治疗时必须考虑到这种相异性。盲着操纵他人的脑会在所有情况下作为反作用。

**关键词：**诠释学，现象学，精神病理学，认识论

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