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Logic Structure of Clinical Judgment and Its Relation to Medical and Psychiatric Semiology

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Key Words

Clinical judgment · Clinical semiology · Psychopathology · Clinical psychiatry · Clinical medicine

Abstract

Background: The logical nature of clinical judgment has been conceptualized in different ways, but a clear connection between the features of clinical judgment and those of semiology is still lacking. **Methods:** The characteristics of clinical judgment, medical semiology, and psychiatric semiology are described. Connections between them are drawn. **Results:** Clinical judgment is described as an abductive inference. Abductive inferences are especially useful to balance universal and singular information. In psychiatric semiology, due to some specific features, a careful balance between the information present in descriptive definitions and the information absent from the definition but present in singular symptoms is needed. The main types of out-of-definition information are reviewed. **Conclusions:** The implications of the results for diagnosis and research are drawn.

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Introduction

Clinical judgment, as it was described by Feinstein in a series of classic papers [1–7], consists of a sequential process which leads from the input data of the patient's manifestation of disease to the output result of diagnostic entities [4]. Of all the multifarious components proposed by Feinstein, which encompass everything from the selection and validation of manifestations to the construction of algorithms, I shall focus mainly on symptom identification. Diagnosis begins with sign and symptom identification and finishes in a putative disease, of course. But naming some particular manifestation as a particular sign or symptom is actually a 'diagnosis' itself [5], and the logic procedure which leads a clinician through symptom diagnosis is the same type of logical inference found in a 'syndrome diagnosis', in spite of the obvious differences of input and output.

From now on, I shall consider clinical judgment as a logical inference which leads from some empirical, particular fact to some putatively suitable diagnosis. Early contemporaneous work on the subject considered clinical judgment an inductive, deductive or Bayesian inference [8–10] and even proposed for it a structure similar to that of laboratory experiments [1]. However, it was also

noted that some constraints which bound clinical judgment did not easily fit these models [11-14]: clinical judgment deals with single particulars for which some category must be found (induction infers a general rule out of several particulars and deduction needs the rule in advance). It must balance general knowledge about diseases and its presentations with specific features of the case. It faces 'ill-structured problems' where the presenting problem and the goal of reasoning process are not defined in advance. It follows a 'known-effect-to-putative-cause' reasoning direction, with different possibilities of interpretation of the empirical fact. Thus, a number of authors turned to another kind of logical inference called abduction [11, 13–14]. In the first part of this paper, I shall introduce a model of clinical judgment based on abductive inference and then proceed to show that this logical procedure is of particular significance for psychiatry, due to some characteristics of psychiatric semiology. In a subsequent paper, I will employ this theoretical stance to examine some contradictions consistently appearing in empirical work on the 'continuity model' of hallucinations and delusions.

In the sections dealing with clinical semiology, I shall draw upon some specific terminology which I must clarify beforehand. It is common practice in clinical semiology to set apart symptoms and signs. Symptoms are considered the subjective manifestation of disease and signs, either spontaneous (jugular throb) or produced (Kernig's sign), their objective counterpart. But both symptoms and signs share a common relation to injury or dysfunction. They both are *caused* by some anatomic or physiologic damage. Thus (the last and least in a long tradition which starts in Laënnec), I shall deal with symptoms and signs as close relatives and call them signs altogether. Wherever distinctions are needed, I shall make them.

But, either objective or subjective, manifestations of disease are not (yet) signs. For something to be a sign of something, some relation must be established between two elements in which element A stands for element B. This relation is called 'code', and this codification is culturally, historically forged. There are no such things as 'natural signs' in clinical semiology. 'Raw' features of disease should be considered 'pre-sign' or 'pre-symptomatic' material which becomes a sign only after the procedures which include them in medical discourse. Any medical sign, then, is the product of some putative biological damage and some culturally achieved codification and not a mere relation between 'natural features' of the world. As stated above, a name is a label, a label is a diagnose [2]. Thus, whenever I deal with medical signs, 'sign' should

be understood as a compound of manifestation plus name, raw material conceptualized within medical discourse.

Peircean Characterization of Clinical Judgment

Clinical Illustration 1

A 33-year-old woman entered the psychiatry ward room and exposed her complaint. She was suffering from insomnia, which she attributed to the maintenance work in her building and the heat of July in Madrid. She showed no psychomotor disturbance or gross behavioral abnormalities. Both the staff and the trainee psychiatrists considered the expression of the complaint a little 'over-emotional' and her prompt denial of further symptomatology 'a bit too prompt'. Her medical and psychiatric report offered no significant information. She received a prescription and left the ward somewhat relieved. A number of questions arise: Which are the most common causes of sleep disturbances in young women? Does this particular disturbance show any specific pattern? Which other symptoms are present or absent? Should any meaning be accorded to the verbal and non-verbal expressive qualities of her behavior? Which are the putative causes and consequences of the disorder? Is she reticent out of suspiciousness? Is she just trying to get a benzodiazepine prescription?

Schleifer and Vannatta [14] have proposed three elements as constituent to the epistemic core of clinical judgment: first, some 'knowledge base', which comprises the current body of empirical knowledge about symptoms and disorders. It also includes descriptive definitions of signs and symptoms, either operationalized or not, demographic data concerning syndromes and pertinent laboratory or neuroimaging results [5]. Following some science theorists, a frame theory or general theoretical assumptions should be included as part of the knowledge base, as they are needed for any empirical data to be meaningful [15] (in clinical illustration 1, it might consist of the statistically more likely causes of sleep disturbances in young women, plus the commonly co-present symptoms or the incidence of major psychiatric disorders in which insomnia is found). Second, clinical judgment includes a method for hypothesis formation (the logic of diagnosis itself, which will be developed later; it leads from this particular complaint to some general disorder). And finally, it includes a reflection on the particular case in relation to the diagnostic possibilities which arise from the conjunct work of the empirical/descriptive/theoretical database and the logic of hypothesis formation (Is this diagnostic possibility the single most likely, parsimonious and exhaustive one? Did I take into account all the information available?).

The abductive logic of hypothesis formation, introduced by the American logician, philosopher and bona fide genius Charles Sanders Peirce, features three properties. First, it is always rooted in a particular situation/ symptom/case. Inference begins with a (yet) unexplained/unexpected fact C (somebody complains about her mind being controlled by the government) and proceeds the following way: if hypothesis A (the patient suffers from psychosis) were true, C would be explained/ expected, so there is good reason to suspect that A is true [16, 5.186]¹. (Deductive inferences proceed just the other way round. Once a general hypothesis is proposed, we may anticipate some effect and confirm/discard it empirically. Inductive inferences are supposed to furnish some general rule concerning a number of putatively correlated particulars.)

Second, it is 'retroductive'. As Schleifer and Vannatta [14] as well as Fischer [13] and Feinstein [5] state, hypothesis in clinical judgment runs from the effect (speckles, chest pain, unexplained abnormal behavior or experience, insomnia) to putative causes (measles, angor pectoris, schizophrenia). In classical logic, this would be an example of fallacious reasoning called fallacia consequentis, but this is the very feature called 'retroduction' by Peirce [16, 5.276, 6.479]. General abductive inference and common clinical practice is retroductive, as we always come across some effect whose cause must be inferred (i.e., 'extracted') from the case. However, once one or several hypotheses have been abducted from a single case, they should be tested to prove their predictive power and their general pertinence [16, 6.470ff.] ('If this is a psychosis, she is likely to behave so and so, but if she has been exposed to heavy pre-electoral propaganda, maybe she is just talking in metaphors.' 'If this pain is angor, then we will find either ECG/CPK abnormalities or both.'). And this test should proceed deductively and allow some prediction going from cause to effect.

Abduction, then, is common to both 'diagnostic hypothesis' and 'experimental hypothesis'. Feinstein [1] even conceptualized clinical judgment as a 'bedside' experiment, formally analogous to laboratory experiments in spite of all the obvious differences. If there is

some true kinship of 'bedside experiments' and laboratory experiments, it may lie within the abductive inference itself.

Third, abductive inferences are kin to perceptual judgments (those involved in 'interpreting' sense data) [16, 5.173]. Perception may be defined as psychic act, with a subject pole and an object pole, which picks up an object or quality among several within a spatiotemporal frame; which is always perspectivistic and body centered; and which allows identification (one among several other objects) and re-identification (in different times) [17]. Perceptual judgments are actually considered by Peirce extreme cases of abductive inferences [16, 5.181], in which some balance is achieved between the singular features of the object perceived and the features it must share with the rest of the members of its class. I consider this connection to perceptual judgments of utmost importance for clinical judgment, because it allows logical inference to balance the descriptive information included in symptom definitions with a wealth of information (contextual or expressive, for instance) which cannot be included in symptom definitions but which play a crucial role in symptom eliciting. In clinical illustration 1, clinicians must decide whether 'promptness' and 'disproportion' deserve special attention or may be dismissed. And, further, if they are better explained by the interview context, the insomnia itself, the reticence and suspiciousness of a deluded patient or just bad temper. Another non-clinical example on the abductive nature of perceptual judgments could consist of a 'tree naming' exercise. In front of the same object, the lay observer would call it 'tree', a second, more seasoned observer 'pine tree', and an expert 'Mediterranean pine tree, roughly 20 years old'. The object is one and the same for the three observers, but the balance between relevant and non-relevant information varies. The more experienced and skilled the observer is, the more reliable, valid, and nuanced the judgments are.

Fourth and last, abduction proceeds by colligating features of facts present but as yet unattended in a new, creative fashion [16, 5.171, 5.581]. This synthetic-creative side of logical inference explains why one and the same series of behaviors or experiences may be considered 'cyclothymia' or 'borderline personality disorder' or 'normal quick-temperedness'. The diagnostic (and semantic) weight accorded to a number of descriptive and non-descriptive features colligated may lead to different results. Hitherto, the similarities between the lay characterization of clinical judgment and its reconstruction as an abductive inference may seem instructing but of painfully

Peirce's texts are quoted following academic usage with the number of the volume and the paragraph.

scholastic interest. Further deepening into the logic of abductive judgment will shed some light on the special difficulties of symptom eliciting in psychiatry.

On the Mutual Aid between Clinical Judgment and Clinical Semiology

As far as mere abductive reasoning is concerned, I have not yet presented any differences between medical and psychiatric clinical judgment. But medical and psychiatric diagnostics are different, and the signs, symptoms, and syndromes abducted are different. I shall present some differences concerning medical and psychiatric semiology and then proceed to show how they are echoed in different kinds of abduction.

A good introduction may be found in Feinstein [5]. Roughly put, clinical reasoning follows this path: first, signs are identified; second, signs are referred to a domain [part of the body which is the source of the manifestation (organ or system)] and to a disorder (abnormality in the structure or function of a domain); third, disorders are referred to an underlying pathological process, and fourth, diagnostic reasoning proceeds to pathoanatomic entities and causal entities.

Feinstein assures that psychiatry remains in the domain-disorder stage, 'psyche' being the domain and major depression or schizophrenia the disorder. Feinstein's view is of course debatable, and many psychiatrists would choose 'brain' as a domain and display their knowledge of a wide array of anatomical or functional findings. But the point I want to stress here is that there is no pathway from signs to anatomic/functional/etiologic entities in psychiatry which could be considered analogous to those of clinical medicine. In this section, I shall substantiate this peculiarity of psychiatry on the nature of its signs.

Clinical Illustration 2

'I need help or I will kill myself.' A 44-year-old Caucasian male entered the psychiatric ward room. He had come accompanied by his 70-year-old mother. He claimed he was afraid of his mother plotting against him with her criminal colleagues. Reportedly, she was fresh out of prison and had resumed her drug dealing activities. He insisted his telephone was wired and that he was being followed by the police as a suspected accomplice. He was highly anxious, restless, barely slept, and 'worst of all, nobody believes me'. His mother belied her son's story. He was diagnosed with acute delusional disorder and re-

ceived antipsychotic medication. Eventually, the whole story proved true.

Medical semiology is mainly characterized by the connections drawn between two series of elements: the observed/provoked bodily manifestations of disease (unilateral mydriasis/light arreactivity with spared accommodation) and those of the subjacent injuries or dysfunctions. The link between them is considered causal [18, 19], either known or (yet) unknown. I will call relations between sign (remember: 'manifestation plus name') and injury 'vertical'. Medical signs also keep relations of mutual determination (I will call them 'horizontal'). The meaning of dullness in thoracic percussion may be 'edema' or 'fibrosis' depending on the other symptoms present or absent: fever, dyspnea, cough. Therefore, the meaning of any medical sign is determined by the subjacent injury/dysfunction which causes manifestation and by the syndrome amidst which the sign is found and which reduces the polysemy of isolated signs [18].

Psychiatric signs or symptoms have not established these vertical, causal relations, although they do entertain horizontal syndromic relations. As I have shown elsewhere [20], there is a good number of reasons for this lack of causal remission, but maybe the most straightforward of them is the very biology of the brain. Several reviews of the neuronal networks putatively involved in the physiopathology of the depressive disorder [21] or cognitive deficit in schizophrenia [22] point to the same conclusion: they are scattered over so many brain locations and they interact in so many ways that the very concept of correlation or remission should suffer a complete refurnishing to remain operative.

A second key feature of psychiatric semiology is the relation between sign-type and sign-tokens. I will employ the term 'sign-type' as a 'universal' or 'category name' synonymous to the descriptive definition of a sign: 'hallucination = perception without object'. Sign-tokens are any particular item which belongs to any category, i.e., the particular hallucination, delusion, obsession found in concrete, individual, empirical patients.

According to Eco [23], type-token relationships belong to two kinds: ratio facile (RF) and ratio difficile (RD). In RF, every feature needed to characterize any token is included in the descriptive definition of the type. In RD, some extra information is needed.

In clinical medicine, relation between type and token is RF. Descriptive definitions of the sign include every relevant feature of the token. In turn, these features may be referred to their immediate causal processes.

RD is well known by clinical psychiatrics, trainers and trainees. The difference between schizophrenic and melancholic delusion or depersonalization may be observed and pointed out (and thus receive what is called 'ostensive definition'), but it cannot be found in the descriptive definition of delusion. It is also the case with many other symptoms, such as true obsessions versus schizophrenic and even autistic pseudo-obsessions. In this RD relation between types and tokens rests part of the special importance of abductive reasoning in psychiatry. As causal vertical correlations are lacking, the bulk of the work in clinical psychiatry does not consist of finding signs ('manifestation plus name') which lead to causal entities but in a meticulous, probing work with pre-symptomatic material in order to achieve consistent, reliable, valid symptom labeling. In this task, the balance between features included in a descriptive definition and features excluded from it becomes crucial. In short, while medical semiology consists of a thesaurus of signs whose descriptive definitions correlate with anatomical/functional damage, psychiatric semiology consists of a thesaurus of signs and symptoms whose descriptive definitions need to be completed in order to reach good 'symptom diagnosis'.

Different Signs, Different Judgments

How are these different signs managed in clinical judgment? Every clinical judgment is abductive, sure, but some species within the genre have been described. Umberto Eco classified abductive judgments into three main categories: hypercodified, hypocodified, and creative (and added a subsidiary one, the meta-abductions, which we will not analyze here) [24]. In hypercodified abductions, there exists an inferential rule followed every time any given sign appears. The rule, then, becomes a code. Eco himself chooses medical signs as cogent examples: Kernig's sign implies meningeal irritation. The rule is well chosen if it leads from sign to damage, in other words, if the rule is mapped onto a causal relation. As Feinstein [5] puts it, a sign 'implies' a damage, but a damage 'causes' a sign. The rule is valid as long as 'causal' and 'implication' relations follow the same pathway in opposite directions.

In hypocodified abductions, the inferential rule must be selected from a number of equally pertinent options already known to the clinician. A common example could be the need to ascribe a hallucination or a persecutory delusion, present together with a limited number of other common symptoms, to either schizophrenia or mania; or thoracic dullness to either edema or fibrosis. The absence of univocal relationship between symptom and injury allows different ways to gather disturbances in a coherent whole.

In creative abductions, the rule must be found ex novo. A quite unproblematic example could be the causal weight of a random biographical event in a given clinical situation. It is very likely that similar circumstances will be quite unique; therefore, no explanation provided will make it into a law of general validity.

Maybe less intuitive, notwithstanding its maximum clinical importance, the process of symptom eliciting also consists of either hypocodified or creative abduction. As mentioned before, it is forceful for psychiatric signs to balance the information included in the descriptions of the thesaurus and the information present in the case but absent from the descriptive definition. But these 'not-indefinition' characteristics of individual symptoms vary from case to case. Ergo, the clinical judgment which identifies some particular delusion cannot fix and codify the rule abducted from this particular case. When some abnormal behavior is again found, a new balance between the general features of the 'sign type' candidates and the individual features of the empirical experience must be achieved.

In clinical illustration 2, the patient's speech acts and behavior may be 'abducted' following different rules: Is he delusional? And if so: mixed manic or anxious paranoid? Is he just afraid of his mother's past deeds and thus overvaluing the chances of new criminal behavior? Is everything just plain truth? As long as there is no 'gold standard test' against which a hypothesis may be checked, careful consideration of all kinds of semiotic material available is crucial. This is, of course, a general principle which affects differently the various items of the semiological psychiatric thesaurus. If we compare anterograde memory impairment (a more or less discrete psychic function, with well-established anatomical correlations) and hallucinations or delusions in Alzheimer's disease, we will get a nice, concise grasp of the differences.

In the next paragraph, I shall advance a provisional outline of the 'not-in-definition' information at work in symptom eliciting. Of course, operationalized descriptive features play a definitive role here, too. But, for the sake of space, I shall focus on non-codified material.

'Not-in-Definition' Material at Work: An Outline

Psychiatric sign-token contribution to the symptom identification process consists of traits and features which may be described but which vary between tokens. They 'individuate' the symptom, then. These individuation conditions include:

- Extrinsic individuation conditions (EIC), such as time, space, and putative causal relations [25, 26]. I will pay no more attention to EIC, as they are quite straightforward ('this patient's' delusion, 'which problem are we talking about? The exhilaration following last cocaine binge, which provoked mild paranoid ideation').
- Intensive individuation conditions (IIC), such as the intensity, persistency, and frequency of the features included in the sign-type definition and the balance between them (as in differential diagnosis between somatoform disorders and their delusional counterparts, based on the 'intensity of conviction').
- Contextual individuation conditions (CIC), such as:
- (1) Relations between the content of the utterance and the expressive qualities of the utterance, where expression works as a microcontext to content [27]. The utterance 'I have never felt so sad in my life' may change its meaning depending on the quality of the expression: Irony ('I'm really happy'); sarcasm ('I always feel the same, if you were a good therapist you would know'); need of care (as in histrionic personality disorders) or plain melancholic but not anesthetic depression. Of course, this does no belie the suffering of non-melancholic patients. But the relationship between content and expression may well isolate different phenomena which, if the content of the utterance was the only material taken into account, would be grouped together, as it has been the unfortunate case with the final common pathway of depression approach to mood disorders.
- (2) Other symptoms present or absent, as it was advanced above. Some behaviors and experiences may be considered as 'apathy-avolition' or 'blunted affect' depending partially on the co-present symptoms. Thus, poorly known conditions may be bypassed or altogether ignored, as other, more easily recognizable symptoms are preferred for diagnostic purposes. The risk of backlash is double: we may use hallucinations and delusions (less specific) to diagnose schizophrenia in spite of more specific but less readily identifiable symptoms (schizophrenic autism or hyperreflexivity or disturbances of ipseity). Thus, we face overinclusive categories. Then, these symptoms of schizophrenia

- which have been bypassed, which may very well be the core of the syndrome and whose characteristics are worse understood, are modeled following other diseases, as is the case with analogies between poor insight and anosognosia.
- (3) Patient's biography and premorbid personality, which are classic contextual dimensions underlying conceptual dichotomies, such as 'development versus process', 'delusion versus delusion-like'.
- (4) The background network of meaning and knowledge which supports any singular experience and without which there can be nothing such as experience. It has been analyzed by Husserl, Merleau-Ponty, Wittgenstein, Waissmann or Searle (I will not enter into their differences here). Stanghellini [28] has fruitfully employed these concepts in his psychopathological analysis of common sense. Another good example could be the differences between the obsessive or pseudoobsessive symptoms, seemingly all-pervading in psychiatric diseases. Are obsessions in obsessive-compulsive disorder, schizophrenia, schizotypal personality, Asperger's syndrome, and major depressive disorders one and the same phenomenon? Patients with borderline personality disorders often describe themselves as 'perfectionist' or 'obsessive' just the same as obsessive personalities do. But what about the differences? Obsessive symptoms and obsessive traits in these conditions may be equaled if only a sort of 'common minimal verbal content' is heeded. But the experience from which this content arises may be quite different: cognitive difficulties to manage changes in routines and familiar entourages as in Asperger's syndrome, hyperreflexivity and loss of natural evidence as in schizophrenia, misconstrued narrative identity and interiorized lack of external validation as in borderline personality disorders.

'Not-in-Definition' Material at Work – An Example (I): Evaluative Judgments and Clinical Criteria

The need of balance between definitional and 'not-indefinition' material in the process of symptom identification is not only related to current operationalized glossaries. It is deeply entrenched both in the basic features of human experience, behavior, and speech and in the way psychopathology makes sense out of their derailment. I have reviewed elsewhere the relations between human experience and language and symptom individuation [20]. But in the next two sections I shall stick to clinical judgment and show how its abductive structure is needed in order to handle the 'not-in-definition' material described by Sadler and Fulford [29] and Stanghellini [30].

Sadler and Fulford [29] have discovered as many as seven possible evaluative judgments necessarily made when applying DSM-IV-TR diagnostic criteria for personality disorder. These judgments 'nested' in diagnostic acts are involved in determining whether a criterion is met, in other words, if token fits type. The seven judgments proposed are: (1) 'semantic-phenomenal matching' (Does the patient's phenomenal clinical presentation match the criterion semantic content?); (2) 'solicitation choice' (Which is the appropriate approach to the soliciting of data from the patient?); (3) 'sociocultural context' (Which cultural norms are relevant to a particular criterion?); (4) 'performance-context matching' (Appraisal of the patient's performance relevant to the sociocultural norms in 3); (5) 'deviance threshold' (Is the patient's deviance from specific cultural norms substantive enough to warrant meeting the criterion?); (6) threshold characterization (Is the deviance threshold qualitative or quantitative?), and (7) 'disvalue characterization' (Is the deviance related to the criterion 'for the worse'?).

These judgments are considered normative warrant, for they involve one or more justifications for the normative elements involved in applying diagnostic criteria to concrete patients. The series of judgments may be objected to, and the 'judgmental' nature of some of them put into question. However, this paper provides a nice example of the wealth of 'not-in-definition' information needed to determine whether any criterion is met.

'Not-in-Definition' Material at Work – An Example (II): Clinical Judgment and the Threefold Division of Psychopathology

Stanghellini [30] has argued for a threefold division of psychopathology, comprising descriptive, clinical, and structural psychopathology. Descriptive psychopathology (akin to the term 'psychiatric semiology' employed here) should provide accurate descriptions of signs and symptoms. It is considered the common language of psychiatry, a tool which ensures communication in spite of theoretical differences. Clinical psychopathology chooses signs and symptoms with (ideally) good reliability and validity, for diagnostic purposes. And structural psychopathology disentangles the many threads interwoven in human subjectivity. Each psychopathology fulfills different roles, obeys different rules and employs different rea-

soning styles. There is, then, good reason to set them apart. But, in the outline of 'out-of-definition' material exposed above they were brought together again. Some CIC belong to clinical psychopathology, some to structural psychopathology. EIC involving causal relations belong to clinical psychopathology, and IIC are essential to grasp the descriptive features of descriptive psychopathology: if conviction is not 'intense' enough, then it is not a convincing conviction.

These claims are not contradictory. Stanghellini analyzes the dense, complex phenomena of abnormal human experience, language, and behavior. But, given the intrinsic semiological characteristics of descriptive psychopathology, some synthesis must be performed in order to reach full semanticity of signs and symptoms. As I have shown elsewhere [20], the different species of psychopathology set apart by Stanghellini are forcefully needed to reach the minimal intelligibility of human experience, language, and behavior encoded in the words 'melancholy' or delusion. As far as clinical judgment is concerned, I do believe abductive inference constitutes a good model of the formal mechanics needed to balance the different approaches and scopes of clinical, descriptive or structural psychopathology and their different involvement in symptom eliciting.

Conclusions

I have argued for two related theoretical issues. First, that psychiatric clinical judgment is an abductive inference. Second, that abductive inference bears special significance for psychiatric diagnosis, as it is the best way to (1) balance definitional and 'not-in-definition' information and (2) deal with the cognitive and epistemic nature of psychiatric semiology. Besides, I have outlined three types of non-codified information, EIC, IIC, and CIC, which vary between individual signs but which are nonetheless essential for symptom eliciting. Which consequences are to be drawn?

First and most important, descriptive definitions of mental signs and symptoms must be 'completed' some way or another during symptom eliciting. Clinicians are trained to do so through repeated exposure to paradigmatic examples, although maybe in some less-explicit-than-desirable fashion. Non-clinicians psychometric interviewers receive training too, but it is now commonly admitted that supervision by experienced clinicians is a quality index of empirical research. But if no such control is provided, as in online interviews or self-adscription

formularies, which information is fulfilling this role? And how?

Second, the pragmatic constraints of research on hallucinations and delusions in non-clinical studies involve some loss of this kind of 'out-of-definition' descriptive information. May this lead to an over-inclusion of phenomena? Are we grouping together phenomena with only loose 'family resemblance', such as common suspiciousness, paranoia, and acute delusions? Recent empirical work seems to point in this direction [31]. Which is then the cognitive structure of symptom categories? These questions will be answered in a sequel paper that will draw on the theoretical model introduced here and will apply it onto the extensive body of work dealing with continuity models for hallucinations and delusions.

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