

## Klaus Conrad (1905–1961): Delusional Mood, Psychosis, and Beginning Schizophrenia

Aaron L. Mishara<sup>1</sup>

Department of Psychiatry, Clinical Neuroscience Research Unit, Yale University School of Medicine, CMHC 339-A, 34 Park Street, New Haven, CT 06519; Present address: Department of Psychiatry, Brain Mapping Unit and Behavioural and Clinical Neurosciences Institute, Cambridge CB2 2QQ, UK

**Klaus Conrad's major contribution to the phenomenology of psychosis focused on the patient's experiences during the prodromal and early psychotic phases of schizophrenia. The literature in English concerning his work is sparse, in part because Conrad's work contains complex concepts that lose much in translation. This communication attempts to clarify Conrad's thought, especially as it pertains to the role of mood and delusions in beginning psychosis and its underlying neurobiology.**

**Key words:** delusional misidentification/delusions/Gestalt psychology/microgenesis/neo-phenomenology/perception/phenomenology/prodromal schizophrenia/conscious states

### Introduction

Acclaimed as a “great German psychiatrist,”<sup>1</sup> Klaus Conrad advanced the concept of prodromal “delusional mood” or “atmosphere.” Thought to have provided “one of the most impressive descriptions ever written concerning early schizophrenia,”<sup>2</sup> his views “on the origin and development of psychotic experiences” are considered “... something completely new, and ... a source of new ideas for research on psychopathology.”<sup>3</sup> Furthermore, the concept of basic symptoms as self-experienced subclinical prodromal disturbances is rooted in Conrad's work and is a central element of the Bonn Scale for the Assessment of Basic Symptoms.<sup>4,5</sup> Nevertheless, the literature in English concerning Conrad is sparse. Some of the barriers are linguistic. Others are conceptual or historical, and some of the barriers are simply because his

work has been misunderstood. In this communication, I elaborate on a few of his core concepts regarding beginning schizophrenia.

### Conrad's Stage Model of Beginning Schizophrenia

As director of a neurologic military hospital, Conrad interviewed and carefully documented the reports of a large sample of soldiers ( $n = 107$ ) experiencing beginning schizophrenia. From these reports, Conrad developed a “stage model” for the formation and maintenance of delusions in beginning schizophrenia.<sup>6</sup> The following case exemplifies this model.

#### *Case History*

Due to possible psychosis, a 32-year-old first-class private, Karl B., was brought to Dr Conrad's hospital. In his interview with Conrad, the patient reports that “everything begins” one morning as his unit breaks to leave camp. When the sergeant asks him for the key to his quarters, it is suddenly clear to him that it is a ploy to “test” him. While departing in the bus, he notices that his comrades are behaving strangely: They know something that he is not supposed to know. One of his comrades asks “conspicuously” if he has any bread. At midday, they arrive in a town to relieve units positioned there. A few in his company are charged with finding quarters for the rest of them. This is only a ruse for the few to receive instructions in how to deal with him while he waits with the others in the motor coach. One after another, groups of men leave the coach only for the others to return. “It is clear that they are all receiving their instructions” about him. The patient is unable to explain how he sees this. He simply “sees it.” He straightens the quarters assigned to him and then goes below to buy cigarettes. He proceeds through a garden, where many noncommissioned officers, the staff sergeant, and some women are sitting. They are surprised to suddenly see him there and are planning that the women fabricate something with him that evening. One of the officers then drives off to inform the superiors about him. In the pub that evening, the music, the woman selling cigarettes, and the conversations have been prearranged to test whether

<sup>1</sup>To whom correspondence should be addressed; e-mail: aaron.mishara@yale.edu.

he notices. Everyone has been instructed and knows exactly what to do.

Due to his failure to follow orders, which is “another test,” the staff sergeant transports him by car to the psychiatric hospital. Everything along the road, eg, piles of stone, construction sites, sheep crossings, is arranged to test whether he notices. While looking out the window, the staff sergeant observes whether he correctly notes all of this. Later he thinks, “There must be some kind of peculiar effect emanating from me. Other people are under my influence as if under a spell.” That is, the persons who experience his gaze, ie, his looking at them, exhibit a distorted facial expression or bodily behavior, indicating the tension they receive from him (“omnipotence”).

When he reaches Conrad’s military hospital, the delusions have progressed from external space to the inner space of his body. The patient reports that a “wave apparatus” controls his movements from some distance through electric current. Adjusted by a dial, the current changes from having negligible control over his movements, preserving his “free will,” to having complete control, at which point, the machine “inputs” commands.<sup>7</sup>

In Conrad’s stage model, there is often a prodromal delusional mood prior to the onset of the delusions. This may last for days, months, or even years.<sup>7,8</sup> During this period, the patient experiences an increasingly oppressive tension, “a feeling of nonfinality” or expectation. The individual describes that something is “in the air” but is unable to say what has changed. Although the experience may be varied, the subject experiences a marked change of “emotional-motivational” state. At first, this is associated with the most salient experiences but eventually spreads to pervade the patient’s entire experiential field. The patient may feel not only excitement, “intoxicated” anticipation, but also suspiciousness, fear, depressive inhibition, guilt, a feeling of separation from others, and often a combination of these. She or he may perform abrupt, seemingly meaningless actions.

Conrad calls this initial expectational phase “Trema” (stage fright) as the patient has the feeling that something very important is about to happen. Attention is drawn toward irrelevant stimuli, thoughts, and associative connections, which are distressing and unpredictable: “The perceptual background acquires entirely new characteristics. Everything that lies in the periphery to one’s attention, what is behind, or not part of the current thematic focus” becomes a potential threat.<sup>7</sup> The perceptual background, which remained unnoticed, now takes on a character of its own. The sense of threat (or whatever the predominant quality of the delusion happens to be) spreads to the entire perceptual field. This means that what is not yet noticed in a potential perceptual gestalt takes on “saliency even before the patient orients to it, ie, before becoming the focus of attention.”<sup>9</sup> The patient’s changed internal motivational-emotional state, the delusional mood, imbues the entire field of experience

with a transformed “physiognomic” quality (ie, a sense of potential revelation/threat accompanied by affective tension due presumably to underlying neurobiological changes). Nevertheless, the subject (like the anosognosic neurological patient) does not attribute the changes to his/her own state but externalizes them to some, yet to be understood process in the world.

The delusions appear suddenly as an “aha experience” (“Aha-Erlebnis” or “revelation”) concerning what had been perplexing during delusional mood and often bring relief. Conrad<sup>7</sup> describes this as a reflexive turning back on the self in which the universe is experienced as “revolving” around the self as middle point (ie, “anastrophe,” see table 1). The delusions are not primarily a psychoanalytic defensive reaction meant to protect the self but involve a fundamental “reorganization” of the patient’s experience to maintain behavioral interaction with the environment despite the underlying disruption of perceptual binding processes.<sup>7,10</sup> At the aha-moment, the patient is to unable shift “frame of reference” to consider the experience from any other perspective than the current one. The transition from delusional mood to the Aha-Erlebnis of the delusional revelation occurs precisely at the moment of loss of the patient’s ability to distance from the experience, ie, “to achieve an exchange of reference frames or perspectives, ie, to consider the situation—even if only temporarily—with the eyes of the other(s).”<sup>7</sup> In becoming delusional, the patient, however, “is not conscious of this loss of this ability to transcend” the current experience.<sup>7</sup> Conrad explains: “Borrowing from ancient Greek, the artificial term ‘apophany’ describes this process of repetitively and monotonously experiencing abnormal meanings in the entire surrounding experiential field, eg, being observed, spoken about, the object of eavesdropping, followed by strangers.”<sup>11</sup> In our case example, wherever Karl B.’s glance falls, every “component of his experiential field” appears to stand in a special relation to him, eg, the “instructions,” the “preparations,” and the “being staged.” “His ‘world’ becomes transformed into a situation specifically meant to ‘test’ him.”<sup>7</sup> Once the apophany takes over the perceptual field, no aspect of the field remains untouched. Then, “everything becomes conspicuously salient. The patient often interprets the course of events as if a film were being made or a theater-piece performed”<sup>7</sup>.

Each stage of beginning schizophrenia involves the subjective “reorganization of meaning” with the view of preserving the subject’s “vital” relationship with the environment (mediated by what Conrad’s contemporary, von Weizsäcker described as a perception action cycle).<sup>6,7,10,12</sup>

Conrad finds that the “delusional misidentification” of persons, a symptom seen in some neurologic disorders, also occurs in beginning schizophrenia and is rooted in delusional perceptions. Of the 107 schizophrenia patients, Conrad<sup>7</sup> describes in his monograph 17 report misidentification experiences. Most are “positive” misidentifications,

**Table 1.** Conrad's Stage Model of Beginning Schizophrenia

Stage	Term
I	<p>Trema (derived from Greek, colloquial for stage fright)</p> <p><b>Meaning</b></p> <p>Delusional mood (or atmosphere)</p> <p><b>Characteristics</b></p> <p>Undefinable, but increasingly upsetting quality spreads from salient aspects to entire perceptual field. Patient feels anticipatory excitement, suspiciousness, alienation, fear, guilt, depression, or combination of these. Patient may perform abrupt, seemingly meaningless actions</p>
II	<p><b>Apophany</b> (Greek apo [away from] + phaenein to show → revelation)</p> <p><b>Meaning</b></p> <p><b>Delusion as revelation</b> (Aha-Erlebnis)</p> <p><b>Characteristics</b></p> <p>Perceptual Gestalt experienced incompletely in terms of its expressive rather than its objective material holistic qualities. Inability to transcend current perspective or to shift frame of references. Abnormal connectedness between seemingly unrelated meanings. Delusional perception, misidentification. Relentless (“monotonous, repetitive”) spreading of the delusion as both “elastic” and fixed to new gestalts.<sup>7,18,26</sup> Progression of delusions from external to inner “space”, delusional body sensations. Patient uncritically receptive and unable to detach, as if trapped between sleeping and waking. Thought insertion, thought broadcasting, hallucinations</p>
III	<p>Anastrophe (Greek, ana- (back) + strephein (to turn) → turning back)</p> <p><b>Meaning</b></p> <p>Patient feels self to be passive middle point (subject-directed complement to world-directed apophany)</p> <p><b>Characteristics</b></p> <p>Delusions of reference. Events and perceptions are related to self</p>

*Note:* Conrad's later stages apocalyptic-catatonic, consolidation (or partial remission), and residual defect state are not presented here.

meaning that a stranger, or unfamiliar person, is perceived as known (“misplaced familiarity”).<sup>13</sup> Conrad describes a patient with incipient schizophrenia who is placed temporarily in a guardhouse before transport. Being a former carpenter, the patient finds that the door, windows, floorboards, and bed frame in the cell have a “familiar” quality. He sees all at once that he himself is the carpenter of these objects. They look so familiar. They were removed from his old workshop. The windowsill has scratches on it, which he made as a child and has been removed from his childhood home. Everything revolves about the patient (anastrophe). The familiar expressive quality of his own workmanship emerges from each object he encounters in the cell and spreads (with monotonous repetition) to his entire perceptual field (misplaced familiarity of delusional misidentification).

### Background for Conrad's Concept of Delusions in Early Schizophrenia

In his drive toward integration, Conrad bases his phenomenologic study of beginning schizophrenia on the Gestalt psychological concept of holistic properties,<sup>14</sup> his phenomenological approach of treating patients as peers or collaborators in the clinical interview,<sup>15</sup> and his own observations of anomalous conscious experiences, including the introspective study of his own hypnagogic hallucinations when falling asleep.<sup>16,17</sup> (For review of the historical influences in Conrad's work, see references.<sup>18</sup>)

Conrad challenged the prevailing European “classical” view (K. Jaspers, H. Gruhle, and K. Schneider) that delusional interpretations of perception are two tiered, ie, an abnormal meaning attaches to an otherwise intact perception without understandable reason or cause.<sup>6</sup> He proposed that the delusion arises rather from an “already transformed Gestalt perception” wherein the affective and expressive “holistic” properties of the Gestalt become exaggerated. He noted, eg, that the attention of both healthy individuals and deluded subjects are captured more by a percept's expressive qualities when the objective or material properties of the percept are attenuated. For example, during a night walk, I see a tree trunk as a crouching robber.<sup>7</sup> Here, the attenuated perceptual structure contributes to the Gestalt's prevailing expressive-physiognomic quality. The physiognomic similarities (between the attenuated perception of the tree trunk and a robber) become more striking (or salient) than normally. In cases of delusional misidentification, the structural-material Gestalt properties recede and the patient relies on their physiognomic-expressive qualities. Thus, the delusional misidentification of persons or objects—as the case of the former carpenter's misidentification of the wooden objects in his cell—is embedded or “given” in the delusional perception. The patient perceives a detail in a stranger's face, eg, a scar or crooked tooth that represents an “expressive quality,” eg, “rugged.” However, it is not the actual scar or tooth in the encountered person but its “ruggedness” that allows

the patient to delusionally misidentify the new person with some prior person who was also experienced as rugged, as being the same person. In delusional mood, the physiognomic similarity between beings, and not their “objective” structural or material Gestalt properties, allows for their identification in the delusional perception.

Conrad recognized a similarity between delusional mood and what the dreamer normally experiences during sleep. The objective material Gestalt is suspended and both healthy and psychotic individuals rely on the physiognomic-expressive qualities of the truncated object perception. This process of impoverishing the perceptual field, which can occur naturally, is induced by an experimental procedure called microgenesis.<sup>7,16,17,19</sup> When healthy subjects are presented perceptual objects in impoverished or reduced conditions, they report similar experiences. Microgenesis<sup>7,16,17,19</sup> experimentally interrupts or diminishes the percept by means of taticopic presentation (often too briefly to be consciously experienced), by reduced illumination of the field, by diminished stimulus size, or by presentation of objects to peripheral vision, tasks in which schizophrenia patients have been shown (during Conrad's time) to be impaired.<sup>19</sup> According to Conrad, the impoverished conditions of stimulus presentation lead, in healthy subjects, to a loosening of the object's perceptual binding and the experience of attenuated perceptions, which mimic the patient's delusional, dream-like state.

Impoverishing the stimulus or interrupting perceptions truncates the perceptual complexity, which normally contributes to our everyday experience of complete objects. It accesses “the sequence of events ... assumed to occur in the temporal period between the presentation of a stimulus and the formation of a single relatively stabilized cognitive response (percept or thought) to this stimulus.”<sup>19</sup>

In the first phase of a microgenetic experiment, the stimulus is presented in the most attenuated manner, ie, as a diffuse, undifferentiated or vague background. A “nondeterminate something” is sensed but not directly seen. In the next microgenetic phase, the stimulus is presented with more illumination for somewhat longer taticopic durations, etc. Figures may be differentiated against the background but continuously flicker in non-stability. The physiognomic-expressive qualities continue to dominate over the figures' structural articulation. Their primitive differentiation emerges but disappears again. The subject has an anxious feeling of nonfinality, yet experiences a feeling of being transfixed, unable to look away. The subject cannot voluntarily distance, critically or reflectively examine the experience, but suffers it in a receptive noncritical attitude. For Conrad, this phase most accurately reproduces in the healthy awake subjects, the delusional patient's, or dreamer's experience.

In a final phase of the microgenetic experiment, the healthy subject recognizes suddenly, with relief and surprise, the actual Gestalt. The delusional patient, on the

other hand, remains attached to the earlier arrested phase of meaning. This marks a stable and sometimes relatively permanent loss of the capacity to shift frame of reference (ie, the ability to test reality) as “the subject is unable to shift back from the previously passive-receptive attitude to a critical attitude.”<sup>15</sup> In the progression to an ever more articulated, or finalized Gestalt, the healthy subject is ultimately able to “detach emotionally” and experiences a sense of completeness or closure. However, like the dreamer, the delusional patient is unable to detach from the incomplete perceptual meaning or pre-Gestalt (Vorgestalt).<sup>7,16,17</sup> Interestingly, and in conformity with Conrad's observations, patients with a history of persecutory delusions score higher than healthy controls on a need for closure scale.<sup>20</sup> Conrad characterizes the paranoid delusional patient as existing in “a world between waking and sleeping,” “a world of fluctuating Gestalten, concerning which, up to this point, the poet has more knowledgeable things to say than the psychologist.”<sup>21</sup>

The phenomenological details of delusional mood, as elaborated by Conrad, have been central in identifying its neurobiological underpinnings.<sup>10</sup> Kapur<sup>22</sup> advanced the idea that dopamine mediates the “salience” of environmental events and their mental representations. Specifically, a dysregulated, hyperdopaminergic state on the neural level leads to an aberrant assignment of salience (or importance) to aspects of one's experiences on the cognitive-mental level. One view of delusions is that they result from the cognitive effort made by the patient to make sense of these aberrantly salient experiences. Experimental pharmacologically induced psychoses (using the drug ketamine) model the link between inappropriately heightened perception, aberrant causal inference, and delusional ideation,<sup>23</sup> and a similar association has been reported in actively psychotic schizophrenia patients.<sup>24,25</sup> Antipsychotic medications dampen the hyperdopaminergic underpinning of aberrant salience and “provide a platform for the resolution of psychotic symptoms.”<sup>22</sup> However, if treatment is interrupted for a long enough time, the aberrant neurochemistry rekindles and delusional mood and cognition returns in the form of a psychotic “relapse.”<sup>22,26</sup>

## Conclusions

Due to its complexity and originality, Conrad's work presents us with conceptual and linguistic challenges. Conrad felt that classical psychology and psychopathology did not possess the “vocabulary” to describe the phenomenology of patient's experience of beginning schizophrenia.<sup>7,11</sup> Although his concepts are difficult, he tried not to hide in the obscurity of esoteric language. He did introduce new terms, albeit apologetically, because he felt that earlier work had not been adequate in conveying the patient's experience. Because his work is easily misunderstood, it is sometimes cited to support

concepts and viewpoints that are, at best, only remotely related to the original work (for reviews of the debate concerning the interpretation of Conrad, see references<sup>18,27–29</sup>). A large ( $n = 267$ ) retrospective study<sup>2</sup> of Conrad's stage model, including both men and women with a greater age span than Conrad's more homogenous male military sample, found support especially for his first stage, delusional mood (Trema), and to some extent, for the next stage, apophasic psychosis. Conrad's work remains timely for current researchers and clinicians due to his thoroughgoing commitment to respectful, collaborative study with patients concerning their subjective experiences of prodromal and beginning schizophrenia and to adhering as closely as possible to what patients endeavored to convey about their own experiences.

## Funding

NARSAD young investigator's award to A.L.M.

## References

1. Berrios GE. *The History of Mental Symptoms*. Cambridge, UK: Cambridge University Press; 1996.
2. Hambrecht M, Häfner H. "Trema, Apophänie, Apokalypse"—Ist Conrads Phasenmodell empirisch begründbar? *Fortschr Neurol Psychiatr*. 1993;61:418–423.
3. Ploog DW. Klaus Conrad, 1905–1961. In: Hirsch SR, Shepherd M, eds. *Themes and Variations in European Psychiatry*. Charlottesville, VA: University of Virginia Press; 1974:239–240.
4. Schultze-Lutter F. Subjective symptoms of schizophrenia in research and the clinic: the basic symptom concept. *Schizophr Bull*. 2009;35:5–8.
5. Huber G. *Psychiatrie*. 6th ed. Stuttgart, Germany: Schattauer; 1999.
6. Uhlhaas PJ, Mishara AL. Perceptual anomalies in schizophrenia: integrating phenomenology and cognitive neuroscience. *Schizophr Bull*. 2007;33:142–156.
7. Conrad K. *Die beginnende Schizophrenie*. Stuttgart, Germany: Thieme Verlag; 1958.
8. Jaspers K. General psychopathology. 7th ed. In: Hoenig J, Hamilton MW, trans. Manchester, UK: Manchester University Press; 1963.
9. Mishara AL. Is minimal self preserved in schizophrenia? A subcomponents view. *Conscious Cogn*. 2007;16:715–721.
10. Mishara AL, Corlett PR. Are delusions biologically adaptive? Salvaging the doxastic shear pin. *Behav Brain Sci*. In press.
11. Conrad K. Gestaltanalyse und Daseinsanalytik. *Nervenarzt*. 1959;30:405–410.
12. Weizsäcker, V. von. *Der Gestaltkreis. Theorie der Einheit von Wahrnehmen und Bewegen*. 4. Aufl. Stuttgart, Germany: Georg Thieme Verlag; 1950.
13. Motjabai R. Misidentification phenomena in German psychiatry: A historical review and comparison with the French/English approach. *Hist Psychiatry*. 1997;7:137–158.
14. Metzger W. *Psychologie*. Dresden, Germany: Steinkopff; 1975.
15. Ploog DW. Autobiographical sketch. *Hist Psychiatry*. 2002;13:358–360.
16. Conrad K. Das Unbewusste als phänomenologisches Problem. *Fortschr Neurol Psychiatr*. 1957;25:56–73.
17. Conrad K. Das Problem der Vorgestaltung. In: Schmoll gen. Eisenwerth JA, ed. *Das Unvollendete als Künstlerische Form*. Bern: Eisenwerth; 1959:35–45.
18. Mishara AL. The 'unconscious' in paranoid delusional psychosis? Phenomenology, neuroscience, psychoanalysis. In: Lohmar D, Brudzinska J, eds. *Founding Psychoanalysis*. New York, NY: Springer; In press.
19. Flavell JH, Draguns JA. A microgenetic approach to perception and thought. *Psychol Bull*. 1957;54:197–217.
20. McKay R, Langdon R, Coltheart M. Jumping to delusions? Paranoia, probabilistic reasoning, and need for closure. *Cognit Neuropsychiatry*. 2007;12:362–376.
21. Conrad K. Die symptomatischen Psychosen. In: Gruhle HW, Jung R, Mayer-Gross W, Mueller W, eds. *Psychiatrie der Gegenwart*. Vol 2. Berlin, Germany: Springer; 1960:369–436.
22. Kapur S. Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. *Am J Psychiatry*. 2003;160:13–23.
23. Corlett PR, Honey GD, Aitken MRF, et al. Frontal responses during learning predict vulnerability to the psychotogenic effects of ketamine: linking cognition, brain activity and psychosis. *Arch Gen Psychiatry*. 2006;63:611–621.
24. Murray GK, Corlett PR, Clark L, et al. Substantia nigra/ventral tegmental reward prediction error disruption in psychosis. *Mol Psychiatry*. 2008;13:267–276.
25. Corlett PR, Murray GK, Honey GD, et al. Disrupted prediction-error signal in psychosis: evidence for an associative account of delusions. *Brain*. 2007;130:2387–2400.
26. Corlett PR, Krystal JK, Taylor JR, Fletcher PC. Why do delusions persist? *Front Hum Neurosci*. 2009; <http://www.frontiersin.org/humanneuroscience/paper/10.3389/neuro.09/012.2009/html/>. Accessed November 11, 2009.
27. Mishara AL. Missing links in phenomenological clinical neuroscience? Why we are still not there yet. *Curr Opin Psychiatry*. 2007;60:559–569.
28. Mishara AL. Kafka, paranoic doubles and the brain: hypnagogic vs. hyper-reflexive models of disruption of self in neuropsychiatric disorders and anomalous conscious states. *Philos Ethics Humanit Med*. 2009; <http://www.peh-med.com/>.
29. Mishara AL. The phenomenology wars: self, psychopathology, neuroscience. *Philos Psychiatr Psychol*. In press.