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SCHIZOPHRENIC BLUNTING: AN APPRAISAL OF CONTINUITY HYPOTHESIS

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ABSTRACT

Considerable scientific attention has been directed towards the psychiatric condition of Schizophrenia because of its global prevalence and the serious effects that it has on the patient's life. An important and grossly under-studied symptom of this disorder is the Emotional blunting of Schizophrenia which is present in a considerable number of such patients. The etiology remains far from understood. Since it is a problem of emotional response, it is logical to look at other emotional states for a clue, of which the dream state is a potential candidate which remains inadequately probed. Here we review of evidences for continuity hypothesis and we will propose a dream related hypothesis for the blunted emotional response in Schizophrenia. Our hypothesis can be divided into two sub-hypotheses: 1) The emotional state of dreams, especially the negative emotions like fear is carried on (is continuous) with the waking state of Schizophrenia more than in normal individuals. We name it the "*Emotional continuity hypothesis*" which derives its name from the original "Continuity Hypothesis" and 2) Due to this continuity, the conscious state continuously experiences an internally generated fear (arising from unconscious mind) which it then suppresses as a compensation response to this negative experience. As this emotional suppression crosses a limit, it results in emotional blunting. We name this aspect the "*Emotional overcompensation hypothesis*". For our hypothesis we derive evidences from phenomenological, cognitive and neurobiological studies and we will appreciate that in all of these approaches, our hypothesis holds good. Phenomenologically, the continuity hypothesis has been well appreciated in several dream-models of Schizophrenia. But we add the questions of directionality and emotions to it which has not been asked before in this context. Cognitively, the phenomena of emotional suppression in Schizophrenia have been studied recently. We try to explore the reasons why emotional suppression rather than other emotion regulation techniques is more used in Schizophrenia and we see that the source of origin of these emotions plays a key role which has not been appreciated before. Finally the neurobiological models show the neural correlates of the emotional suppression in Schizophrenia in later stages of fear-experience are associated with a decrease in Amygdala activity. We try to join these separate bits and pieces of neurobiological evidences and will show that all of them put together makes a picture of emotional-overcompensation as per our propositions. This hypothesis should be tested further to develop a treatment module for Schizophrenia patients with the symptom of emotional blunting.

KEYWORDS :

INTRODUCTION

Considerable scientific attention has been directed towards the global disorder of Schizophrenia because of the serious implications that this has on the patient's life. However, an important and grossly under-studied symptom of this disorder remains in the form of Emotional blunting which is present in around 66% of such patients. The etiological understanding of this symptom remains far from clear. Given the fact that Emotional blunting is a poor prognostic finding which negative affects the course of the disease, it becomes all the more important to have a proper comprehension of this symptom [1]. Till now, much of the research work has focused on neuropathology rather than phenomenology [2]. We find it very strange that this emotional blunting has never been correlated with dreams of Schizophrenia patients given the fact that dreams and Schizophrenic psychosis states have been compared for almost a century now [3]. Here we use an old construct of dreams for demystifying this emotional blunting response in the form of "Continuity hypothesis".

There are two parts of our hypothesis: 1) We propose that the emotional state of dreams, especially the negative emotions like fear is carried on (is continuous) with the waking state of Schizophrenia more than in normal individuals. We name it the "*Emotional continuity hypothesis*" which derives its name from the original "Continuity Hypothesis" and 2) Due to this continuity, the conscious state continuously experiences an internally generated fear (arising from unconscious mind) which it then suppresses as a compensation response to this negative experience. As this emotional suppression crosses a limit, it results in emotional blunting. We name this aspect the "*Emotional overcompensation hypothesis*".

We approach our hypotheses by first describing the state of emotional blunting in Schizophrenia. We then elaborate the continuity hypothesis as applicable to general population and we will see how this hypothesis remains poorly defined inspite of so many years of work focussed on it. We will then move towards application of this theory in Schizophrenia. As we proceed, we will evaluate the literature on the emotional state of dreams and how continuity of these dreams can occur in Schizophrenia patients. Finally, we will propose our hypothesis with the back ground of description of *"Emotional suppression"* as an emotion regulation mechanism seen in Schizophrenia and will see that there are several phenomenological and neurobiological evidences in support of our model.

EMOTIONAL BLUNTING IN SCHIZOPHRENIA: A POORLY UNDERSTOOD CONSTRUCT

Emotional blunting is a negative schizophrenic symptom which results from deficits in emotional processing, typified by the inability to process fear adequately. This symptom is present in about 66% of patients and has a longitudinal stability which predicts poor outcome [1]. This emotional deficit has been assessed in several studies by emotional task performance. In a simple conditioning task using aversive emotional stimuli, for example, patients failed to develop an increase in response frequency to aversively reinforced trials, whereas healthy volunteers acquired a differential response to reinforced versus non-reinforced trials [4,5]. In addition to general abnormalities in the processing and attribution of negative emotional states [6,7], many schizophrenic patients manifest deficits in the recognition of fearful faces [8,9].

Inspite of these important findings, the exact etiology of the affective blunting in Schizophrenia remains unknown. The focus has been mostly on the neurobiological exploration which have provided some important results. For example,reduced amygdala volumes which are the best known neural correlates of fear processing have been found in schizophrenic patients with emotional blunting [10, 11]. The popular two-hit theory of negative symptoms also indicates a neurobiological defect (Dealt with in details below). But how exactly do these neurological defects begin and evolve over the course of time remain unknown. Emotional blunting is an intriguing phenomena especially because of two contradicting features simultaneously: 1)The experience of

emotion is normal or may even be enhanced [12,13] but 2)The outward emotional expression is reduced. Thus we need a hypothesis which not only explains the outwardly impairments in emotional expression but also the reason why the emotional experience is normal to heightened in first place. Thus our hypothesis will incorporate both these emotional features: experience as well as expression. For this, we take into account a state where emotions are experienced by not expressed- the unique state of dreams. Our further discussion will move around this unique state.

THE CONTINUITY HYPOTHESIS: A NEUROBIOLOGICAL REVIEW

The continuity hypothesis is perhaps the most popular hypothesis of dreams given by Hall and his colleagues after his extensive studies of dream diaries of people in 1950s to 1970s [14,15]. The theory puts for the notion that the dream state and the awake states are actually continuous with each other with regard to experiences. In addition to the support from dream diaries earlier, now it has received a very strong support from neurological findings. Neurophysiologically, it has been reported that the anatomo-functional pattern of sensorial analyses is preserved during REM sleep [16] and according to some authors, REM sleep and wakefulness can be fundamentally considered as equivalent brain states, probably subserved by an intrinsic thalamocortical loop, with the main difference lying in the influence of sensory inputs on cognition [17]. Moreover, experimental data emerging from neurocognitive research seem to indicate that the dreaming brain generates phenomenological consciousness events (ie, subjective and first person experiences) that are overall similar to those experienced during wakefulness [18].

However, the hypothesis is very ill-defined in itself. Unlike a proper hypothesis, there is no null-hypothesis for it. Also, the lack of clarity over the definition of *"continuity"* remains a notorious problem, especially the question whether the continuity is for thoughts, emotions or experiences. These issues have been pointed out and adequately elaborated by in a series of discussions between Hobson and Schredl [19, 20]. Here we highlight another important issue of this continuity which has been rarely addressed. We raise the question- is the continuity unidirectional or bi-directional? Whereas the hypothesis theoretically incorporates the effects of waking state on dreams and the effects of dreams on subsequent waking state, empirical researches seem to be only focused on the effects of waking state on dreams [20]. Thus we question that if the continuity only exists from sleep to wake states (WAKE → SLEEP continuity). Or is the continuity also similarly applicable from sleep to wake (SLEEP → WAKE continuity).

The WAKE → SLEEP continuity has been perhaps the most studied dimension of this hypothesis. It has been shown over several studies that the elements of pre-sleep waking state affect the dream content like life events and stressors [21,22]. Also, psychological daytime well-being has been reported to be inversely correlated with the level of aggressiveness and hostility in dreams [23].

However, the WAKE → SLEEP continuity has only been rarely studied, which will be the focus of our further discussion in this article.

WHY CONTINUITY HYPOTHESIS SEEMS APPEALING IN SCHIZOPHRENIA?

The bizarreness of Schizophrenic states and the dream states seem strikingly similar in first instance. Perhaps this has been the reason that historically philosophers and researchers have tried relate these states with each other.

This comparison was perhaps first made by Carl Gustav Jung In 1907 as reflected in his famous quote *"If we could imagine a dreamer walking around and acting his own dream as if he were awake, we would see the clinical picture of dementia praecox."*[3].These similarities have been also noted by the famous psychoanalyst Sigmund Freud [24].and Eugene Bleuler who proposed that "The modalities of thinking of schizophrenic subjects are very similar to dreaming" and hypothesized that in dreaming state "most of the characteristics of schizophrenic thinking (particularly delusional thinking) are explained by the differences Between the dreaming and the wakefulness way of thinking." [25] In Bleuler's clinical perspective, one of the most striking psychopathological traits of schizophrenia is that people who are affected with the disorder are detached from reality. These notions have also been echoed by, Euge`ne Minkowski, a French psychopathologist fellow of Bleuler." [26].

Somewhere in the middle of last century, these hypothetical constructs started receiving scientific evidences and this led to a more evolved theory, now popularly known as the "The dream theory of Schizophrenia". Simply put, the theory proposes that Schizophrenic psychosis is actually a state which involves a phenomenological dream-like state. Although the "Continuity theory" has never been cited openly in this dream model for Schizophrenia, it has been assumed implicitly in several of the prevalent dream theories of this condition [27,28].

An important support of this theory derives from the apparent analogies between dreams and psychotic states. For instance, both in dreams and schizophrenic psychosis, internally generated stimuli are perceived as having an external cause, suggesting a common disturbance in source-monitoring and the ability to make self-other distinctions. Agency attribution is also disturbed, as seen in schizophrenia patients claiming that others are controlling their thoughts [29]. This bears a striking similarity to the involvement of non-self dream figures in a majority of dreams. One study found that the mean average of dream figures in dream reports was 3.9 [30], and they are often described as agents that are not under the control of the dreamer [31]. Additionally, certain delusions, such as persecutory delusions, are frequent in dreams (e.g.many studies have found dreams of being chased to be among the most frequent dream themes).

In addition to these similarities between clinical state of Schizophrenia and dreams, there are other neurobiological similarities between the two. For example, Dzirasa and colleagues [32] could not distinguish the spectral content of REMS from wake state in psychotic-like rats: both wakefulness and REMS exhibited equal levels of fast and slow oscillations' power. Therefore, REMS and wake states seem to be intermingled in animal models of psychosis. Dopamine D2 receptors in the mesolimbic pathway are activated during REMS [33], the same pathway involved in psychosis [34, 35]. This could explain why dreams are characterized by a plethora of mental experiences that resembles hallucinations [36], suggesting that dreaming would be a good model for psychosis [37, 38, 27]. In accordance with this hypothesis, Dzirasa and colleagues [32] showed that reducing dopamine transmission, a therapy used to treat psychosis, also suppresses REMS. REMS and schizophrenia have similar variations in the levels of noradrenaline, serotonin, acetylcholine and glutamate. [37, 38, 39, 40]. Hallucinogenic substances also suggest a link between dreams and psychosis [41]: LSD increases the frequency of ponto-geniculo-occipital waves, which are associated with the execution of brief ocular saccades during REMS, constituting therefore a possible correlate for the visual experiences induced by LSD and during dreaming [42].Neuroimaging studies of the frontal cortex further indicate a proximity between dreams and psychosis. Schizophrenic subjects have this region severely impaired – a condition called hypofrontality [43,44]. Frontal areas also deactivate during physiological REMS, especially the dorsolateral prefrontal cortex [45]. Low frontal activity may reduce self-awareness and induce delirious thoughts and lack of rational judgment, which are present in both REMS and psychosis [36].

The rapid eye movement dreaming sleep stage and schizophrenia are both characterized by common intracerebral disconnections, disturbed responsiveness and sensory deafferentation processes. Moreover, in both states, there is dorsolateral prefrontal deactivation as shown by the decrease of blood flow. Finally,

identical pharmacological and neurochemical variations are observed for acetylcholine, dopamine, noradrenaline, serotonin and glutamate concentrations. Consequently, rapid eye movement sleep could become a useful new neurobiological model of this mental disease since more functional than current rat models using stimulation, lesion or drugs. Gottesman puts things this way "the dorsolateral prefrontal deactivation observed both during REM sleep and in schizophrenia seems to suppress or decrease its own functions, including the loss or decrease of reflectiveness, and at the same time disinhibits older subcortical structures and corresponding functions, with the exaggeration of accumbens' and amygdala nuclei's own processes: in our case, the appearance of hallucinations, delusions, bizarre thought processes, and affective disturbances [37].

Finally, there are important phenomenological differences between the dreams of Schizophrenia patients as compared to normal individuals.

A few studies have analyzed differences in dream content of patients with schizophrenia versus those without mental illness [28,19,46,47]. As per the reports of Lusignan et al [48], Patients with schizophrenia spontaneously rated their dream reports as being less bizarre than did controls. Also, both groups had a comparable density of rapid eye movements during REM sleep but a significant positive correlation between eye-movement density and dream content variables was only found in controls. Taken together, the findings suggest that dream content characteristics in schizophrenia may reflect neurocognitive processes, including emotional processing, specific to this disorder.

Researchers have reported that dreams in patients with schizophrenia tend to be simpler and less elaborate [49,50], less emotionally sophisticated and self-involved [50,51], more bizarre [52, 53,54], and more negative, violent, and unfriendly [51, 53, 55] compared to dreams of healthy individuals. In many cases, patients with schizophrenia see themselves as victims of hostility from outside in their dreams [53,56].

In summary, the available research indicates that compared to controls dreams of schizophrenics are shorter, contain more hostility, more strangers, with the dreamer rarely being the main character. Results are inconsistent on the level of bizarreness, whereas nightmares seem more prevalent in schizophrenics than controls.

CONCLUSION

Simply put, our hypothesis states that the emotional blunting in Schizophrenia is due to over-compensation of the negative tone by suppressing it. This negative emotional tone is carried to the awake state by being continuous. For this hypothesis, we derive phenomenological and neurobiological evidences from various studies for our hypothesis and this should be tested further as it could have important implications in treatment of emotional blunting schizophrenia patients which carries several important prognostic implications for them.

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