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OPINION PAPER

On the Neurophysiology of Delusional Disorders: Top-Bottom vs. Bottom-Up Theories

***Gentian Vyshka*[1*] *and Jera Kruja*[2]**

[1]*Faculty of Medicine, Department of Biomedical and Experimental, University of Medicine in Tirana, Albania*

[2]*Faculty of Medicine, Department of Neuroscience, University of Medicine in Tirana, Albania*

***Corresponding author:** *Gentian Vyshka, Faculty of Medicine, Department of Biomedical and Experimental, University of Medicine in Tirana, Rr. Dibres 371, Tirana, Albania*

Abstract

The psychopathology of delusions has been indelibly denoted from top-bottom theories, with a very long psychiatric tradition explaining the issue as a cortical derangement. The reverse face of the entire issue has been poorly if ever, scrutinized. The role of the peripheral nervous system in the installing and the maintaining of delusive ideas and convictions, albeit elusive, need to be addressed. The hallmark of bottom-up theory relies upon distorted primary processing of peripheral stimuli, with the cortex ingenuously entering a vicious circle of false perceptions, and therefore of false beliefs. Some research suggests also that the peripheral nervous system has an intrinsic role in pain memory, thus contradicting previously cortically-oriented theories. Charles Bonnet syndrome illustrates how a distorted or a severely damaged sense of vision might produce florid psychiatric symptomatology. Apart from mere hermeneutical points of view, psychopharmacology itself will testify how difficult is to eradicate delusions with antipsychotics, classical or atypical ones. The fact that these drugs act on central synaptic pathways, and are almost neutral to the peripheral nervous transmission of signals, will be another evidence of how the periphery of the nervous system might be a starter of the delusions, instead of being a remote, inert and innocent part of the whole.

Keywords

Delusion, Top-Down Theory, Bottom-Up Approach, Experiences

Introduction

When classifying and approaching delusions, delusional disorders and their highly particular psychopathology, the bulk of studies focusing on cortical dysfunction is overwhelmingly important [1-4]. Balancing a top-down approach that would rely specifically on cortex dysfunction with a bottom-up paradigm that would impute faulty or erroneous sensorial primary processing seems, therefore, a hard undertaking.

According to DSM-5 delusions are fixed beliefs, not amenable to change in light of conflicting evidence [5]. Not far from this definition is the other one equivalently delineating the delusion as a false or unshakeable idea or belief [6]. Table 1 summarizes some of the principal types of delusions, according to DSM-5.

Of course, other subtypes that might be identified and included inside some of the major forms mentioned above: delusions of misidentification, of death and immortality (somehow a form of Cotard's) and litigious delusions might be some exotic notions, although still valid and discussed [7,8]. Another very interesting definition, although mainly of clinical value, relates delusions to a "mono" symptomatic, i.e. not embedded in the context of a general paranoia [9]. Cotard's and

Table 1: Principal types of delusions [5].

Persecutory delusions	
Referential delusions	
Grandiose delusions	
Erotomanic delusions	
Nihilistic delusions	
Somatic delusions Bizarre delusions	Thought withdrawal
	Thought insertion
	Delusions of control

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Othello's syndromes might be considered within this frame [7,9].

Instead of calling this phenomenon a belief or idea, some authors expand their view centrifugally by using the notion of experience (delusional experience) [10]. This terminological dilemma might seem insignificant; however, the way you call it underscores the way as well you conceive the disorder, and also the concept that mirrors the underlying psychopathology. What we are trying to discuss here below is not the delusion as a symptom of schizophrenia, albeit some deductions are still valid. Instead we are focusing over the delusional disorder under the connotation of "partial psychosis". Such a denomination has been given mainly due to of the otherwise intact cognitive organization, as well as of the sense of reality [11]. However, difficulties into conceiving and strictly defining the diagnostic notions might be as well related with the continuous changes into the classificatory systems: those have seen substantial adaptations year after year [12].

TOP-BOTTOM Model: Cortex and Below

Corlett, et al. offer an exhaustive overview of the biology of delusions in a paper of 2010 [13]. While discussing the delusions of misidentifications, authors speculate that laterality of damage is important: Here we are upholding the top-down paradigm, with the cortex (and subcortical adjacent structures) principally imputed. The right hemisphere generates error signals, and the left hemisphere starts constructing explanations that will at the end, result in delusions [13]. The cortical area responsible has somewhere been even more precisely delimited, such as in the case of dysfunctions in the orbitofrontal cortex that specify top-down emotional predictions [13]. Hereby particular importance is granted to the notion of salience and misinterpretation of neutral or innocuous stimuli. The role of dopamine in controlling signal to noise ratio inside subcortical neurons, whose disequilibrium will thereafter encode uncertainty, will turn us back to the dopaminergic model of psychosis as a whole [14].

In fact, hyperdopaminergic state leads to an aberrant assignment of salience to the experience elements, shifting the imbalance from the brain to the mind level [15]. The salience theory of delusions relies mainly on the existence of aberrant signals from bottom-up salience detection systems; however, there might still be a decreased top-down regulation to make delusions, and probably hallucinatory state, to flourish [16]. Thalamo-cortical activity, which serves as a major filter to sensory input ascending versus cortex, may be generally reduced in psychosis, and this will suffice to produce and maintain either delusions or hallucinations to an invalidating point of suffering [17].

Of course, inside the sophisticated and complex cerebral matter-be it subcortical or cortical-there is a continuous circling of information in the form that subcortical systems (within brain, but beyond sensory systems) serve as a so-called and maybe for some authors a "bottom" source to the cortex. Instead, we are considering the cerebral matter as a single hardware in our approach. The ours would be a simplified top-bottom view, but this dichotomy might be helpful if primary sensory systems (vision, audition, somesthesia) are approached from their peripheral end. This so-called bottom-up hypothesis would be relying on the erroneous processing in the senses, principally in the entrance gate where the body of the first sensory neuron lies: Be it the dorsal root ganglion (spinal cord), the ganglionic cell layer (retina) or the cochlear ganglion (ear).

Karl Jaspers has contributed substantially to the actual comprehension of the phenomenology and neurobiology of delusions [18]. His are some milestone concepts on genuine, primary delusions (echte Wahnidee), delusion-like ideals (wahnhafte Idee), subjective experiences (Wahnerlebnis). In the pioneering descriptions of his clinical casuistic Jaspers strongly suggest that the core of delusion relies upon disordered cognitive-rational processing [19]. The introduction of delusion in the form of an abnormal consciousness of meaning to the patient's perceptual experience has been considered an extension to the initial Jaspers' definition of delusion [20]. However, Mishara, et al. [18] meaningfully underline the fact that Jaspers did not consider the possibility that the perceptual processing might be diminished or disrupted. From this disruption we think might start the opposite paradigmatic explanation of the delusional disorder: the bottom-up approach.

Bottom-Up Paradigm: The Immediacy of Experiences

If you call it a belief or an idea, you already are granting a cortical status to the phenomenon. But if you call it an immediate experience, if not a primary perceptual product, then you have to include inside the explanatory scheme also the sensorial system, and maybe right from its very peripheral division. Our previous studies have suggested that phantom sensation might originate in the peripheral nervous system [20]. Phantom limb sensation (and pain) seem very far from being considered as a delusional product. Nevertheless, some authors like Ramachandran have gone close to putting these concepts into a single basket [21].

Hence the deafferentation model is at hand: how about adopting it into the cases of patients completely blind but suffering from visual hallucinations? [22] Charles Bonnet syndrome probably has the right clues to suggest how an isolated visual (thus, sensory) deprivation leads to a psychotic situation. Be it not enough, history of psychiatry has as well wide reflections over the dilemma of a probable role of the peripheral ner-

vous system on some mental disorders. Approximately one century before psychiatrists believed that middle ear disease might lead to schizophrenia [23]. The same is suspected and suggested for olfactory dysfunction [24]. A delusional infestation might as well start with a skin disorder, suggesting a clear peripheral primum movens in a situation that has been given the cortical/subcortical explanation for granted [25].

Psychopharmacology itself will testify how difficult is to eradicate delusions with antipsychotics, classical or atypical ones [26]. The fact that these drugs act on central synaptic pathways, and are almost neutral to the peripheral nervous transmission of signals, will be another evidence of how the periphery of the nervous system might be a starter of the delusions, instead of being a remote, inert and innocent part of the whole. It might seem warranted if not already implied, a thorough examination of sensory organs (sight, audition, somesthetic system) when treating every delusional situation, before restricting its treatment to only psychiatric intervention(s).

Conclusion

The top-bottom versus the bottom-up theory concerning delusional disorders (of whichever type they present), remains still an open debate and a conundrum for the neurophysiology. The role of the peripheral nervous system has been generally considered as trivial to the cortical or subcortical dysfunction, continuously considered as responsible for the delusional activity accordingly to the dopaminergic hypothesis. Several sources suggest that sensorial deprivation or sense diminishing might have an important role during the manifestation of delusions.

Conflict of Interest

The authors have no conflict of interest to declare.

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References

1. Devinsky O (2009) Delusional misidentifications and duplications: Right brain lesions, left brain delusions. Neurology 72: 80-87.
2. Blakemore SJ, Oakley DA, Frith CD (2003) Delusions of alien control in the normal brain. Neuropsychologia 41: 1058-1067.
3. Allen P, Moore H, Corcoran CM, Gilleen J, Kozhuharova P, et al. (2019) Emerging Temporal Lobe Dysfunction in People at Clinical High Risk for Psychosis. Front Psychiatry 10: 298.
4. Coltheart M (2010) The neuropsychology of delusions. Ann N Y Acad Sci 1191: 16-26.
5. (2013) Diagnostic and statistical manual of mental disorders. (5th edn), American Psychiatric Association, Arlington, VA, 87.
6. Harvey RJ (1996) Review: Delusions in dementia. Age Ageing 25: 405-408.
7. Vyshka G, Çomo A (2019) Delusions of Immortality in a Post-War Society: The Albanian Case. Front Psychiatry 23: 613.
8. Konakanchi R, Grace JJ, Yap D, Guttmacher L, Szarowicz R (1999) Pimozide in the treatment of litigious delusions. Psychiatr Serv 50: 837.
9. Richardson ED, Malloy PF, Grace J (1991) Othello syndrome secondary to right cerebrovascular infarction. J Geriatr Psychiatry Neurol 4: 160-165.
10. Gallagher S, Mundale J (2009) Delusional experience. In: J Bickle, The oxford handbook of philosophy and neuroscience. 513-521.
11. Opjordsmoen S (2014) Delusional disorder as a partial psychosis. Schizophr Bull 40: 244-247.
12. Winokur G (1986) Classification of chronic psychoses including delusional disorders and schizophrenias. Psychopathology 19: 30-34.
13. Corlett PR, Taylor JR, Wang XJ, Fletcher PC, Krystal JH (2010) Toward a neurobiology of delusions. Prog Neurobiol 92: 345-369.
14. Fiorillo CD, Tobler PN, Schultz W (2003) Discrete coding of reward probability and uncertainty by dopamine neurons. Science 299: 1898-1902.
15. Kapur S (2003) Psychosis as a state of aberrant salience: A framework linking biology, phenomenology, and pharmacology in schizophrenia. Am J Psychiatry 160: 13-23.
16. Kapur S, Agid O, Mizrahi R, Li M (2006) How antipsychotics work-from receptors to reality. NeuroRx 3: 10-21.
17. Behrendt RP (2003) Hallucinations: Synchronisation of thalamocortical gamma oscillations underconstrained by sensory input. Conscious Cogn 12: 413-451.
18. Mishara AL, Fusar-Poli P (2013) The phenomenology and neurobiology of delusion formation during psychosis onset: Jaspers, Truman symptoms, and aberrant salience. Schizophr Bull 39: 278-286.
19. Berner P (1975) Zum heutigen stand der wahnforschung. Psychopathology 8: 1-3.
20. Vaso A, Adahan HM, Gjika A, Zahaj S, Zhurda T, et al. (2014) Peripheral nervous system origin of phantom limb pain. Pain 155: 1384-1391.
21. Ramachandran VS (1998) Consciousness and body image: lessons from phantom limbs, Capgras syndrome and pain asymbolia. Philos Trans R Soc Lond B Biol Sci 353: 1851-1859.
22. Koek AY, Espinosa PS (2018) Ave Maria and Visions of Children: Atypical Charles Bonnet Syndrome or Two Coexisting Deafferentation Phenomena? Cureus 10: e3191.
23. Mason P, Rimmer M, Richman A, Garg G, Johnson J, et al. (2008) Middle-ear disease and schizophrenia: A case-control study. Br J Psychiatry 193: 192-196.
24. Turetsky BI, Hahn CG, Borgmann-Winter K, Moberg PJ (2009) Scents and nonsense: olfactory dysfunction in schizophrenia. Schizophr Bull 35: 1117-1131.
25. Kimsey LS (2016) Delusional Infestation and Chronic Pruritus: A Review. Acta Derm Venereol 96: 298-302.
26. Skelton M, Khokhar WA, Thacker SP (2015) Treatments for delusional disorder. Cochrane Database of Systematic Reviews.