

The Causes of Schizophrenic Voice Hallucinations: A Critical Review

Álvaro Machado Dias

University of São Paulo, Department of Neuroscience and Behavior

Corresponding author: Prof. Mello Moraes, 1721 Caixa Postal 66261 Cidade Universitária, São Paulo, Brazil;
E-mail: alvaromd@usp.br

Abstract

Background: There are many theoretical controversies in relation to the etiology of the schizophrenic voice hallucinations (VHs) related to the fact that different approaches are settled over different levels of causation.

Aims: To review two influent and opposed models of schizophrenic VHs and to discuss the emerging divergences.

Method: critical survey of the literature.

Results: The two most important hypotheses are 'corollary discharge' and 'externalization and reality testing'. The first assumes that these symptoms are the automatic outcome of a brain dysfunction, while the second conceives of VHs as the result of a psychological disorder that affects some of the individuals at risk (this risk been defined by neurobiological abnormalities). There are evidences supporting both hypotheses and the best option to cope with their opposition is to consider that, at least to some extent, it represents a typical 'nature vs. nurture' opposition, where the apparent contradiction between the two, does not necessary reflects the impossibility of raising a third hypothesis integrating some of their most important features (German J Psychiatry 2010; 13: 331-36).

Keywords: schizophrenia, voice hallucinations, corollary discharge, cognition

Received: 15.3.2009

Revised version: 5.3.2010

Published: 17.3.2010

Introduction

Voice hallucinations (VHs) are the most recurrent symptom in schizophrenia, believed to affect 70-75% of the affected individuals (WHO, 1973; Nayani and David, 1996; Ford and Mathalon, 2005). Several distinctions can be made in relation to VHs; phenomenological, the most prominent categorization regards the source of the auditory stimuli (voices), which can be assumed by the patient to be inside the head (pseudo form) or outside of it. This last form is much more severe than the other, as it departures from the sense of belonging that characterizes normal thinking. It is also three times more common (Copolov et al., 2004), leading us to conclude that, in general, delusional states in schizophrenia are conditions where self-generated sensorial stimuli (e.g., VHs or visual imagery) are assumed to be extraneous to the patient's body. VHs tend to be frightening and enhance social withdrawal, producing severe mental suffering and deviant conducts (Schneider, 1959). There is a difference between sporadic hallucinations that may occur to a non affected individual (e.g. associated

to high fever) and schizophrenic VHs. In this last case, it is not only the emergence of the voices that characterize it as a psychotic manifestation, but some of its odd features, like the fact that the voices remain active for long periods, and that they do not disappear, regardless of efforts to avoid them (Beck and Rector, 2003). Based upon the fact that odd ideas impair normal reasoning in schizophrenia, several symbolically inspired models of the syndrome's aetiology have been proposed along its history (Bleuler, 1911; Bleuler, 1962; Chaika, 1990; Crow, 1990; Crow, 1995; Crow et al., 1996; Crow, 1999; Crow, 2000; Gianotti et al., 2001; Zislin et al., 2002); and following this very tradition, it was proposed that VHs represent a specific type of symbolic dysfunction, related to the perception of sub-vocalizations (Frith and Done, 1989; Frith, 1992). However, not everyone agreed with that idea; another very influent hypothesis says that VHs have strong psychological determinants, since the hallucinatory content is frequently constituted of warnings, feelings of guiltiness, shame, and blame (Bentall, 2002; Costafreda et al., 2008). It is not hard to perceive that these two hypotheses cannot be perfectly associated, although it might not be the case to say that they are completely opposed. Based on that, the aim of this paper is to critically discuss

them, in order to spawn a better understanding of this complex field of studies and its most important trends.

Discussion

The basis of the current VHs models

To begin with the discussion on discordant models of schizophrenic VHs it is important to define basic points of agreement. The first point is that these symptoms are correlated with biological dysfunctions. It is currently believed that abnormal frontal and temporal brain activity, as well as impaired frontotemporal connectivity represents core-risk factors for schizophrenic VHs (Sullivan et al., 1998; Lawrie et al., 2002; Takahashi et al., 2006; Okugawa et al., 2007; Zhang et al., 2008). A second point of agreement is that VHs represent a failure on the capacity to distinguish internal and external symbolic inputs (Heilbrun, 1980; Frith, 1987; Frith and Done, 1989; Frith, 1992; Bentall et al., 1994; McGuire et al., 1995; Frith, 1996; Fu et al., 2000; Allen et al., 2004; Braham et al., 2004; Fernyhough, 2004; Frith, 2005; Frith, 2005; Shergill et al., 2005; Kumari et al., 2008). "Hallucinations occur when private or mental events are not attributed to the self" (Bentall et al., 1994, p.53). From that standpoint, the two basic lines of thinking regarding the causes of such failure can be defined as: 1. the hypothesis that schizophrenic VHs reflect abnormal neural activity, in pathways related to sensorial processing of sub-vocalizations; and 2. the hypothesis that biological abnormalities are only the first step of the process that generates VHs in schizophrenia, which also includes internal conflicts and biased cognitive schemas, especially related to reality testing. Let's call the former 'type 1' hypothesis and the latter, 'type 2' hypothesis.

The corollary discharge hypothesis

The most prominent type 1 hypothesis of the current days is the 'corollary discharge hypothesis' (Frith, 1987; Frith and Done, 1989; Frith, 1992; Friston and Frith, 1995; Frith, 1996; Frith, 1996; Johnstone and Frith, 1996; Frith, 2005; Frith, 2005). This hypothesis is based upon the premise that sub-vocalization (and reasoning in general) does not require much sensorial activation, since it is already under intentional control, while the perception of stimuli arriving from the outside world requires higher activation due to their intrinsic unpredictability. "Any predictable signal has less impact on the nervous system unless it has some special priori value" (Frith, 2005, p. 758). From that perspective it follows that, for normal's, sub-vocalizations produce attenuated subjective experience in relation to the externally generated ones; and thus, that VHs represent a failure in the mechanism that produces this selective attenuation, leading the affected individual to experience internal voices with the same intensity as the externally-generated ones (Frith, 1987; Frith and Done, 1989; Frith, 2005; Frith, 2005). The guidelines of the corollary discharge hypothesis were defined in

the 19th century (Bell, 1823-1974); it was first applied to voice hallucination in schizophrenia by Feinberg (Feinberg, 1978; Feinberg and Guazzelli, 1999), receiving later support, not only from Frith, but, among others, from Judith Ford and collaborators (Ford et al., 2001; Ford et al., 2002; Ford and Mathalon, 2004; Ford et al., 2005; Ford and Mathalon, 2005; Ford et al., 2007; Heinks-Maldonado et al., 2007; Ford et al., 2008; Mathalon and Ford, 2008). According to the updated version of the hypothesis that is endorsed by Frith and Ford, the capacity to differentiate internally and externally generated stimuli is produced as the self-generated ones automatically generate an efferent copy of their own, which travels from motor to sensorial regions of the prefrontal cortex, where they attenuate the experience of the sub-vocalizations, much as self-tickling and other self-generated stimulations are less vivid than their externally generated counterparts. . In that sense, the hypothesis that VHs are a consequence of a corollary discharge failure represents a typical model of abnormal sensorial inhibition of self-generated stimuli, and thus a special type of attentional dysfunction, which impairs the normal sense of agency that is supposedly produced by the experience of attenuation. Several findings endorse the corollary discharge hypothesis: data from general motor-sensory communication dysfunction (Friston and Frith, 1995); the existence of normal frontotemporal coherence in gamma band under the production of speech, but not while listening to an external source; and the existence of cognitive dysfunctions related to connective abnormalities (Ford et al., 2001; Ford et al., 2005). However, it is also a fact that the hypothesis remains under debate in relation to some important features: although many neural pathways have been associated to corollary discharge in many species (Poulet and Hedwig, 2007), discredit in relation to its importance within inner speech perception (Mackay et al., 1993), and the overall importance of efferent copies in the understanding of the structure of perception as whole (Bridgeman, 2007).

The externalization and reality testing hypothesis

The main line of opposition to the corollary discharge is the conception of VHs as a byproduct of externalization and lack of reality testing (Bentall and Slade, 1985; Barkus et al., 2007; Reed et al., 2008). This hypothesis considers that VHs is not an emergent effect of a biological abnormality, but rather a construct, which emerges after externalization and biased evaluation of the source of verbal stimuli. As one may note, the first stage of the hypothesis (externalization) converges with general ideas of the corollary discharge hypothesis in the sense that it assumes the existence of a natural tendency to conceive internally generated sensory stimuli as externally generated; however, the second stage is more psychologically-driven, as it points to the idea of a psychological failure. In that sense, it has received support from authors with very different conceptions about the schizophrenic deficits (Bentall et al., 1991; Blackwood et al., 2001; Allen et al., 2004; Atkinson, 2006; Allen et al., 2007; Wood-

ward et al., 2007). In a fMRI experiment, Allen, et al. (2007) found that, among normal subjects and non-hallucinatory schizophrenics, an external voice (2nd person) leads to increased activation of the superior temporal cortex in comparison to the activation produced by their own voice, while hallucinators produce equal activation whenever listening to their own voice or in the target condition. Lately, this tendency towards externalization was replicated by Brunelin and coworkers, who used as controls both healthy subjects and depressive patients (Brunelin, et al., 2008), at the same time that Costafreda, et al. (2008) bridged the gap between the biological basis of externalization and its psychological structure, as they showed that the phenomenon was influenced by the valence of the target stimulus: externalization was intensified according to the negative status of the experimental voices. The perspective that VHs are associated to externalization and that the aversive nature of the mental content lays in the heart of the phenomenon differentiates this hypothesis from corollary discharge. That summarizes the most important aspects of Richard Bentall and collaborators' 'reactive' hypothesis on the nature of schizophrenic VHs (Bentall and Slade, 1985; Bentall and Slade, 1985; Bentall et al., 1991; Bentall et al., 2001; Bentall, 2004; Bentall, 2004; Bentall, 2006; Bentall, 2007); "paranoid patients, in common with patients suffering from depression, have latent negative beliefs about the self that are vulnerable to activation by negative life events. However, in contrast to patients with depression, we have assumed that patients with persecutory delusions attempt to avoid the activation of negative beliefs about the self by attributing threatening events to the actions of another person" (Bentall et al., 2001, p. 1163). Extending these ideas to VHs, Bentall and collaborators believe that they cannot be reduced to neurobiological dysfunctions, since externalization is assumed to be intrinsically related to psychological conflicts. In that sense, the main differences between the 'corollary discharge' and the 'externalization and reality testing' hypotheses are not only related to the fact that they are settled in different levels of analysis, but that the former assumes an unintentional standpoint, while the latter assumes that VHs are highly dependent on the conditions that favor externalization, and thus represent an intentional reaction to mental content. As proposed in different occasions, by Bentall and collaborators, these conditions are represented by external attribution of negative outcomes, biased self-evaluation, and the tendency to pre-attend to negative and threatening stimuli. Finally, it is interesting to note that Richard Bentall is famous for challenging the instrumental use of the concept of schizophrenia and for stressing the existence of a nosological continuum between normality and psychosis. In line with these ideas, his model assumes that schizophrenic VHs are not necessarily different from VHs that may be manifested outwardly of the psychotic spectrum, since both share the same basic psychological mechanism, which can be defined by the German concept of *verlegung*, as first described by Freud (1911).

Reasoning through the conflict of interpretations

Two lines of evidence favor the corollary discharge hypothesis in relation to the reality-testing model: hearing impairments are important risk factors for schizophrenia (Malmberg et al., 1995) and VHs tend to cease (or at least to decrease) with procedures to preclude sub-vocalizations (Bick and Kinsbourne, 1987). In a study that challenges Bentall's perspectives, Ishigaki and Tanno (1999) concluded that hallucinators (represented as 'H+') are prone to perceptual biases when compared to normal controls, but not to non-hallucinators schizophrenics (H-), while they have the same level of evaluative bias as normal controls. This means that their tendency to produce biased reality testing is less severe than among other types of schizophrenic patients (H-). In contrast, one advantage of Bentall's and collaborators' model is the fact that it explains the well-known fact that stressful conditions favors the emergence of hallucinations, much as endorsed by Beck and Rector (2002; 2003; 2005) who not only emphasize the importance of stress on the formation of delusional constructs, but also associate these constructs to affectively induced naïve information-processing. These last authors also assign another aspect of VHs that can be considered problematic to Frith's hypothesis: VHs do not necessarily resemble the experience of normal sub-vocalization; actually, hallucinations may feel 'metallic', 'sublime', 'devilish', etc., much as if they were produced by someone else, in opposition to sub-vocalizations, which are made of the own person's voice. From that standpoint, the content that produce the VHs is conceived as isolated chunks of declarative information that are triggered in affective contexts (Beck and Rector, 2003), which establish close connections with ruminant states of depression, and the automatic thoughts that are present within the scope of the obsessive-compulsive disorder (as first note by Rachman, 1981). Additionally, it is also true that any pure neurologic-inspired model must face the risk of being over static, favoring a clear-cut distinction between normal and abnormal functioning, which may not always be so clear within the clinical setting. Although VHs can have a sudden onset, schizotypy does not, and as suggested and confirmed in relation to the dimensional divisions of the Launay-Slade Hallucination Scale, some non-psychotic manifestations predict increased proneness to VHs, with emphasis on vivid daydreaming (Launay and Slade, 1981; Bentall and Slade, 1985; Levitan et al., 1996; Aleman et al., 2001; Larøi et al., 2004). One thing that has never been discussed is whether corollary discharge may be prone to psychological effects, therefore representing the neurobiological basis of externalization. If that proves to be the case, it could follow that both sides end up being correct; however, that would call for certain amendments to the corollary discharge theory, which would need to revise the idea that automatic efferent copies of a self-generated stimuli are directed to perceptive areas, diminishing sensorial activation. One hypothesis that can be raised in relation to that regards the possibility that, instead of being an 'all-or-nothing' process, corollary discharge represents a progressive tendency, which tends to be gradually inhibited by the activation of brain areas that trigger fear (e.g., amygdala), disgust (e.g., insula) in affected individuals. It is obvious that this is

not a simple proposal, as it automatically raises the problem of knowing why these affective states trigger VHs in the psychiatric population but not in normals, which is a matter that we hope to see solved in the near future.

Conclusions

In line with what we have considered, it is not possible to say that one of these two perspectives represents the ultimate explanation of schizophrenic VHs, but rather that it is important to consider the interesting aspects of both. With that in sight, the maintenance of two-level conception, which takes for granted that neurobiological dysfunction is in the core of the phenomenon, seems to be particularly interesting in face of the fact that VHs are very common symptoms, and thus suggestively associated to endophenotypic patterns. Although the nature of the biological dysfunction that underlies schizophrenic VHs is not fully established, it is worth considering that corollary discharge may be prone to psychological influences and that this whole discussion proves to be one more of those Nature vs. Nurture debates, where the claims raised by both sides are not as contradictory as they appear at first. This is the main perspective that we should have in mind, as we grasp these divergent perspectives and try to untangle the picture.

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