



Contributions of emotional prosody comprehension deficits to the formation of auditory verbal hallucinations in schizophrenia

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ABSTRACT

Deficits in emotional processing have been widely described in schizophrenia. Associations of positive symptoms with poor emotional prosody comprehension (EPC) have been reported at the phenomenological, behavioral, and neural levels. This review focuses on the relation between emotional processing deficits and auditory verbal hallucinations (AVH). We explore the possibility that the relation between AVH and EPC in schizophrenia might be mediated by the disruption of a common mechanism intrinsic to auditory processing, and that, moreover, prosodic feature processing deficits play a pivotal role in the formation of AVH. The review concludes with proposing a mechanism by which AVH are constituted and showing how different aspects of our neuropsychological model can explain the constellation of subjective experiences which occur in relation to AVH.

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1. Introduction: phenomenology of AVH and EPC

Emotional impairment in schizophrenia was first observed by Kraepelin (1919). Disturbances range from flat affect to intense bursts of inappropriate emotions such as anger and fear (Kraepelin, 1971 [1919]). The combination of these emotional disturbances points to one of the paradoxes of schizophrenia: whilst there can be a flattening of affect, as in negative symptoms, there can be also an increase in emotional arousal and reactivity, as in the positive symptoms of psychosis (Aleman & Kahn, 2005). Traditionally, deficits in emotion

processing have been linked to negative symptoms such as inefficient social interaction, and apathy and avolition towards social stimuli (Hoekert, Kahn, Pijnenborg, & Aleman, 2007). However, a possible link between emotional processing difficulties, particularly for vocal stimuli, and the presence of hallucinations or delusions (Rossell & Boundy, 2005) has also been reported. In fact, dysfunctional emotion processing may also be associated with the positive symptoms of schizophrenia (Aleman & Kahn, 2005). In line with this idea, it has been reported that maladapted forms of emotion regulation, such as expressive suppression, have been associated with severity of hallucinatory experience (Badcock, Paulik, & Maybery, 2011).

Although emotional disturbances are of central importance in psychosis in general (van 't Wout, Aleman, Kessels, Laroi, & Kahn, 2004), this review will focus on the impact of such disturbance in relation to hallucinations. Deficits in the comprehension of vocal emotion are

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thought to be specific for schizophrenic patients suffering from hallucinations (Rossell & Boundy, 2005; Shea et al., 2007). Additionally hallucinations in schizophrenia tend to be more often auditory than visual (Mueser, Bellack, & Brady, 1990). The congruency in modality between affective processing deficits and abnormal perception might suggest an underlying common mechanism. Auditory modality emotion recognition abilities are likely to be of central importance in the formation of AVH, and the study of this connection promises to reveal a mechanism linking both phenomena.

In everyday interactions, humans are persistently exposed to verbal communication, meaning that large amounts of social information are carried by the voice (Belin, Fecteau, & Bedard, 2004). Speech encodes semantic information, but also carries non-linguistic information collectively known as prosody. These prosodic elements comprise acoustic features such as pitch, amplitude, and segment and pause duration. Prosody can be used to disambiguate the meaning of an utterance (i.e. statement vs. question, known as linguistic/stress prosody) as well as to encode affective information which is known as emotional prosody (Belin et al., 2004). Thus, prosody allows for the encoding and decoding of feelings in speech.

One of the central characteristics of AVH is that the voices patients hear are often spoken in emotional tones (Copolov, Mackinnon, & Trauer, 2004), most often with a negative emotional valence (Nayani & David, 1996), e.g., angry voices shouting abusive language. One interpretation of the link between AVH and EPC deficits lies in the coincidence in emotional valence between both phenomena. Patients with AVH seem to have difficulties correctly identifying happy prosody, possibly because of a tendency to attribute fear or sadness to any stimulus (Tsoi et al., 2008) and research in schizophrenia suggests that perception and labeling of emotions such as anger and fear may also be impaired (Allen et al., 2004). Regarding perceived intensity of prosodic emotions, it has also been discovered that patients with AVH generally tend to rate frightening stimuli as more intense than controls and patients without AVH (Rossell & Boundy, 2005). Given that patients who hear “malevolent” voices tend to rate them as “very powerful” more often than patients who hear benign or benevolent voices (Birchwood & Chadwick, 1997), these AVH patients might also rate fearful stimuli in EPC tasks as more intense. It should be noted that this review focuses on AVH in schizophrenia and not in non-clinical population, as the emotional valences of hallucinations between both populations seem to differ.

Delusions and hallucinations also seem to be linked. It is reasonable to assume that individuals experiencing hallucinations may need to explain this anomalous perceptual experience, and such attempts may rise to delusions. From this point of view, hallucinations are understood as a primary phenomenon, and delusion a consequence of the former (Maher, 2006).

The relation between EPC deficits and AVH might also be mediated by attentional mechanisms. In the early literature, it was proposed that a breakdown in selective attention may overload working memory with irrelevant sensory data. Such an overwhelming sensorial influx putatively makes it difficult to integrate current perceptions with past experience, giving rise to abnormal perception and resulting in hallucinations (Chapman & McGhie, 1964). This proposal, however, does not explain why the perceptions reaching consciousness very often have a negative affective tone. Instead, this review proposes that an attentional bias towards negative affects in EPC tasks may increase the likelihood of experiencing AVH or even act as a trigger for them. Therefore, it is essential to determine the direction of causation in any such relation. On the one hand, emotionally negative AVH might lead to a negative mood state, which is reflected in a bias toward negative affects in EPC tasks. This has been shown in the visual modality, in that depressed patients show a bias to perceive neutral emotional facial expressions as negative (Hale, Jansen, Bouhuys, & van den Hoofdakker, 1998). Thus, it is plausible that patients may live in an emotional negative state because of the intrusive hallucinations they experience, and this negative emotional state might bias

judgements regarding the emotional expressions of others, such as in the evaluation of emotional prosody. On this interpretation, the bias in EPC may be based in the distress that results from the experience of emotionally negative AVH. In this causal model, AVH cause distress, and distress may lead to EPC deficits.

That interpretation notwithstanding, we intend to discuss how AVH might be associated with underlying EPC disturbances, with the direction of causation leading from emotion processing deficits to the psychotic symptom. We will consider how EPC deficits may contribute to the formation of AVH and to what extent aberrant auditory processing might be underlying both phenomena. In order to demonstrate this link we will consider behavioral and brain functional and structural findings connecting AVH and EPC, and we will integrate this evidence to suggest a new neuropsychological model of AVH with the aim of explaining the phenomenology of the abnormal experience.

2. The link between AVH and EPC at the behavioral level

Until recently, the idea of an affective prosody impairment as a modular deficit in schizophrenia was controversial. It was assumed that impaired prosodic processing in schizophrenia patients merely reflects basic sensory deficits such as misperception of pitch and amplitude (Leitman et al., 2005). For example, Leitman et al. (Leitman et al., 2005) suggested that prosody processing deficits may be due, in part, to low-level pitch discrimination defects (Leitman et al., 2005). However, there is evidence to suggest that prosody processing deficits in schizophrenia cannot be solely explained by pitch perception defects. In fact, pitch is a feature of emotional as well as linguistic prosody. If pitch perception impairment were the only cause of prosody processing deficits, linguistic as well as emotional prosody should be equally affected in schizophrenia, but contrary to this assumption, stress prosody comprehension seems to be preserved in patients with schizophrenia (Murphy & Cutting, 1990). In line with this observation, it is plausible that the dysfunction of low-level auditory processing, even if partially contributing to EPC impairment, is not sufficient to explain the complexity of the emotional prosody deficits in schizophrenia and that these may rather relate to a specific emotion processing deficit.

In addition to the specificity of EPC impairment as an emotion processing deficit, EPC difficulties are particularly prominent in the subgroup of patients with schizophrenia who have a tendency towards AVH. In a study comparing patients with and without AVH in an EPC discrimination task (Rossell & Boundy, 2005), it was observed that when EPC performance was tested with non-lexical speech sounds spoken in different tones of voice, only the AVH patient group were impaired relative to controls. When EPC stimuli contained prosodic as well as semantic elements, both patient groups showed significantly worse performance than controls. The authors interpret these findings as suggesting a dissociation between EPC for auditory stimuli with prosodic and semantic content, which further supports the idea of a specific connection between AVH and EPC. The additional processing of semantic content may mask the relation between EPC and AVH. Deficits in semantic processing are found in schizophrenia in general, without being linked to any symptomatic manifestations of the disorder in particular (Rossell & David, 2006) while emotional prosodic deficits seem to be specific to the AVH subgroup (Rossell & Boundy, 2005).

Interestingly, some research did not find an association between EPC deficits and hallucinations. On the contrary, an association between negative symptoms and EPC deficits was found (Leitman et al., 2005). However, it should be noted that the cited study collapsed patients with different diagnoses in the same group (i.e. schizophrenia and schizoaffective disorder) and it applied a tool for the measurement of positive symptoms which did not distinguished between different modalities of hallucinations. Thus, it might be the case that prosody deficits might

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