A Cognitive Neuroscience View of Voice-Processing Abnormalities in Schizophrenia: A Window into Auditory Verbal Hallucinations?

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Abstract: Auditory verbal hallucinations (AVH) are a core symptom of schizophrenia. Like "real" voices, AVH carry a rich amount of linguistic and paralinguistic cues that convey not only speech, but also affect and identity, information. Disturbed processing of voice identity, affective, and speech information has been reported in patients with schizophrenia. More recent evidence has suggested a link between voice-processing abnormalities and specific clinical symptoms of schizophrenia, especially AVH. It is still not well understood, however, to what extent these dimensions are impaired and how abnormalities in these processes might contribute to AVH. In this review, we consider behavioral, neuroimaging, and electrophysiological data to investigate the speech, identity, and affective dimensions of voice processing in schizophrenia, and we discuss how abnormalities in these processes might help to elucidate the mechanisms underlying specific phenomenological features of AVH. Schizophrenia patients exhibit behavioral and neural disturbances in the three dimensions of voice processing. Evidence suggesting a role of dysfunctional voice processing in AVH seems to be stronger for the identity and speech dimensions than for the affective domain.

Keywords: auditory verbal hallucinations, cognitive neuroscience, EEG, event-related potential, neuroimaging, voice affect, voice identity, voice speech

Auditory hallucinations are a heterogeneous phenomenon typically involving vivid auditory perceptions in the absence of external stimulation, though less distinct and faded percepts may occur.[1,2] Auditory hallucinations are experienced by up to 70% of schizophrenia patients.[3] Most commonly perceived as voices (auditory verbal hallucinations—AVH), auditory hallucinations are not an exclusive symptom of schizophrenia: they can be observed in other psychiatric disorders (e.g., major depression, bipolar disorder) and also in a minority of individuals with no psychiatric or neurologic diagnosis.[4–6] Nonetheless, auditory hallucinations in schizophrenia represent a more severe and qualitatively distinct phenomenon, with higher levels of associated emotional distress.[7]

Here, we will focus on AVH, the most common type of auditory hallucinations in schizophrenia.[8] AVH are commonly experienced as having a specific identity.[9–11] They are typically heard at a normal conversational volume, in the form of commands or specific comments about what the patient is doing.[11] The linguistic complexity of AVH varies from hearing single words or random sentences to full conversations, with the former being more often reported.[11] They are often perceived as being spoken in the second or third person,[10] most often by an adult male.[9,11,12] The role of emotion in AVH generation and content is highlighted by their typically having a negative emotional tone[13,14] and being experienced as comments, criticisms, or commands, often with self-referential content.[12,15]

Different models have been formulated to explain AVH, including memory-based,[13,16] reality-monitoring,[3,17,18] auditory vivid imagery,[19] and verbal self-monitoring accounts[20–25] (see Text Box 1). Even though distinct mechanisms were proposed to underlie the experience of AVH, these models might not be mutually exclusive.[26]

To date, however, no theory has been capable of fully explaining why patients hear voices in the absence of external stimulation, or why AVH are often perceived as verbal

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Text Box 1
Main Assumptions of Conceptual Models Aiming to Explain AVH

Model and proponents	Arguments
Memory-based account (Morrison & Baker [2000]; Waters et al. [2006])[13,16]	AVH result from disturbances of intentional inhibition of thoughts and context memory: impaired intentional inhibition of traumatic memories produces intrusive thoughts that, due to impaired context memory, are not experienced as self-generated
Reality-monitoring account (Bentall [1990]; Brébion et al. [2000]; Brébion et al. [1996])[3,17,18]	AVH result from abnormal memory and reasoning processes underlying source's identification of internal events: these abnormalities compromise self-recognition and the capacity to remember whether the information has an external or internal source
Auditory vivid-imagery account (Aleman et al. [2002])[19]	AVH result from vivid auditory imagery: perception is highly influenced by auditory imagery, leading to impaired ability to identify internal events as self-generated
Verbal self-monitoring account (Allen et al. [2007]; Frith & Done [1989]; Frith [1987]; Johns et al. [2001]; Johns et al. [2006]; Jones [2010])[20–25]	AVH result from a failure in monitoring inner speech, leading to impaired distinction between self-generated and others' voices

messages uttered with a negative and derogatory content, and as being produced by an external agent. AVH, such as those perceived as real voices, convey a wide range of linguistic and paralinguistic information that allows not only the identification of speech's meaning but also the detection of its emotional salience and identity.[27] Previous studies have shown that the processing of identity,[28–33] affective,[34–42] and speech[43–46] information carried by the human voice is significantly disturbed in schizophrenia and, importantly, that impairments in these dimensions may be related to specific clinical symptoms, particularly AVH.[28–34,37,46] Therefore, it seems plausible that abnormalities in the processing of voice stimuli might play a role in the way AVH are experienced.

ABNORMAL VOICE PROCESSING IN SCHIZOPHRENIA

The human voice is probably the most important class of sounds that occurs in our environment and that we hear most often in our daily lives, carrying socially relevant information.[47] Therefore, the ability to discriminate and recognize voices is central to social communication. More recent studies suggest that vocal input is processed by specialized regions of the human auditory cortex,[48,49] and that different types of vocal information—speech, affective, and identity information—are processed in partially dissociated functional pathways that interact with each other during normal voice processing.[27,47–53] Examples of dissociation have been documented in patients with neurological disorders, such as patients with phonagnosia,[54–56] in which normal speech perception seems to coexist with impaired speaker recognition.

Disrupted bottom-up auditory processing is a central feature of schizophrenia.[57–61] The abnormalities extend to the processing of voice stimuli.[29–32,34–38,41,62,63] We still do not know, however, whether these abnormalities are observed for all dimensions of voice processing or whether some dimensions are more impaired than others. In addition, it is unclear whether these abnormalities relate to specific symptoms, such as AVH.

In the next sections we review behavioral, neuroimaging, and electrophysiological evidence shedding light on how schizophrenia patients process identity, speech, and affective dimensions of voice signals, taking into account the multidimensional model of voice processing from Belin and colleagues.[27] We aim to provide a comprehensive picture of the existing evidence by bringing together behavioral and brain data. Moreover, we discuss in what extent specific abnormalities in each dimension of voice processing (identity, speech, and affective information) may be associated with specific AVH phenomenological features. For each dimension of voice processing, we first review studies that tested schizophrenia patients regardless of symptom profile, and we then focus on studies specifically comparing patients with versus without AVH. Of note, during this review the terms *AVH* and *auditory hallucinations* will not be used interchangeably. Auditory hallucinations encompass a variety of phenomenological experiences (e.g., environmental sounds, noises, and musical hallucinations), whereas AVH—verbal in character—represent their most common type. Since different subcategories of auditory hallucinations probably reflect differences in the underlying mechanisms, throughout this review we differentiated studies that specifically tested patients with AVH from those referring to auditory hallucinations in general, without further specification; that is, the authors did not specify the type or content of auditory hallucinations. However, as most of the auditory hallucinations are experienced as voices, we considered that the inclusion of the studies generally approaching the phenomenon of "auditory hallucinations" would be relevant for the purposes of the current review.

The identification of studies for the present review was based on keyword or title searches in PubMed using the following terms: auditory verbal hallucinations, voice/vocal processing, voice/vocal identity, speaker identification, vocal affect, emotional prosody, vocal speech, auditory semantic,

external speech. In addition, we searched the reference sections of the articles so identified for further potential studies for inclusion.

ARE AVH ASSOCIATED WITH ABNORMAL PROCESSING OF VOICE-IDENTITY INFORMATION?

Recognizing the identity of a vocal stimulus is a complex process based on the analysis of paralinguistic features, which include fundamental frequency, formant frequency, intensity, and duration, leading to the recognition of the speaker.[27,47] The combination of these acoustic features results in a distinctive and exclusive voice signature for each individual.

AVH are experienced as real voices, more frequently with a specific identity,[10] and might be perceived as having a specific regional accent distinct from the patient's accent.[12] Most commonly, both male and female patients "hear" more than one single voice, often a middle-aged male voice, though younger patients might also "hear" younger voices.[10,12] The phenomenological features of AVH suggest that altered processing of voice identity, particularly of a self-generated voice, may underlie the experience of AVH.

Behavioral Evidence

Disturbed processing of voice identity in schizophrenia has been increasingly reported by behavioral studies.[23,24,29,30,32,64,65] Some of the verbal self-monitoring experiments provide evidence for impaired discrimination between self-generated versus non-self-generated voices in schizophrenia patients.[23,65] In these experiments, participants are typically required to talk or read aloud, and they receive immediate auditory feedback that is either intact or distorted in terms of the voice's source (self vs. non-self) or quality (undistorted vs. pitch-shifted).[23,65–68] They are instructed to judge if the voice was self-generated or produced by another person. Verbal self-monitoring was proposed to act by means of a corollary discharge mechanism from brain motor to sensory regions, in order to attenuate the sensory responsiveness to self-generated actions.[20,69] During speech production or inner-speech generation, frontal speech-production regions are thought to "alert" auditory sensory regions when the incoming speech is self-generated, dampening its responsiveness, and thus contributing to the distinction between self-generated and non-self-generated stimulation.[20,69] One study reported impaired recognition (i.e., reduced accuracy) of a self-generated voice when schizophrenia patients were judging distorted feedback of their own voices,[23] regardless of symptom profile (i.e., patients with and without AVH). Another study, however, observed a more generalized deficit in discriminating the identity of both self-generated and non-self-generated voices in schizophrenia patients, relative to healthy controls, especially when the feedback was pitch-shifted.[65]

Furthermore, irrespective of the presence or not of AVH, schizophrenia patients seem to be less accurate than healthy controls in recognizing the identity of famous voices[29] and in using specific acoustic cues to judge the similarities between different voices.[31] In the study by Chhabra and colleagues,[31] schizophrenia patients both with and without AVH showed impaired use of formant frequency dispersion to discriminate voices, relative to healthy controls, whereas the use of pitch cues was relatively intact. Formant frequency dispersion refers to the average distance between the first five formant frequencies resulting from the supra-laryngeal filtering, which, in turn, amplifies specific harmonics in the voice signal.[27,47] The supra-laryngeal filtering gives the acoustic quality to consonants and vowels. These findings suggest altered bottom-up processing of specific auditory cues, compromising speaker recognition.[31]

Even though the study by Chhabra and colleagues[31] failed to find differences between patients with and without AVH, the impaired use of formant dispersion may have important implications for understanding AVH phenomenology, such as why voices are often perceived as pertaining to a male and powerful identity. Indeed, this acoustic parameter is related to masculinity and dominance perceptions.[31] Although these abnormalities were observed in schizophrenia patients irrespective of symptom profile,[31] some studies[29,30,32,64] directly compared patients with, versus without, AVH in behavioral tasks involving the differentiation between familiar and unfamiliar voices. These studies reported impaired recognition in AVH patients, when compared to healthy controls (patients without AVH did not significantly differ from AVH patients and healthy controls).

Of special relevance for understanding AVH, impairments in the ability to discriminate between self-generated and unfamiliar voices have been specifically demonstrated in schizophrenia patients with AVH.[23,24,28,33,67–70] Verbal self-monitoring experiments provide evidence for an external misattribution bias that seems to be more pronounced in patients with AVH compared to patients without AVH and healthy controls.[23,24,67–70] Patients experiencing AVH, compared to the other two groups, tend to erroneously classify their own voices as being somebody else's.[22–24,33,67,68,70] This finding is especially noticeable when the auditory feedback of their own voice is pitch-distorted.[23,24,67–70] As an indication of a more generalized deficit, however, one study reported that AVH patients were less accurate than both patients without AVH and healthy controls in recognizing the identity of both self-generated and non-self-generated voices.[69] Put another way, patients with AVH tended to misidentify self-generated voices as alien voices, and vice versa.[69] It is plausible that the disturbed sensory processing of self-generated voices in AVH patients, evidenced by abnormalities in the use of acoustic cues (pitch-shifted and formant-dispersion cues) for voice differentiation,[23,24,31,33,68,69] might contribute to impairments in later stages of voice perception—an hypothesis that can be further explored with methodologies such as event-related potentials (ERPs).

Despite some evidence suggesting that impaired discrimination between self-generated and non-self-generated voices may be related to disrupted bottom-up processing,[23,24,31,33,69] other

evidence suggests the role of top-down impairments.[67] In the study by Ilankovic and colleagues,[67] paranoid patients with hallucinations and delusions tended to make more errors than healthy controls when identifying the identity of self-generated and unfamiliar voices during conditions in which expectations about the source of voices were incongruent with the voices that they heard. Participants' expectations about the source were manipulated by presenting participants with visual cues (the self or an unfamiliar face) before they heard either their own or unfamiliar voices. Although this study did not include a control group of patients without AVH, the findings demonstrated a failure of patients with AVH and delusions to inhibit top-down attentional processes during the recognition of their own versus unfamiliar voices—which may result in the misattribution of self-generated speech.[67]

Neuroimaging Evidence

Neuroimaging studies have provided evidence for the relationship between abnormalities in temporal and frontal brain regions and impaired discrimination between self-generated and non-self-generated voices in schizophrenia patients. Concretely, some functional magnetic resonance imaging (fMRI) experiments testing the corollary discharge mechanism have demonstrated differences in brain activation between schizophrenia and healthy controls. In the case of speech, this mechanism "alerts" auditory sensory regions when the incoming stimulus is self-generated, thus dampening their responsiveness.[69–71] In healthy controls, speaking and receiving auditory feedback that matches their expectations (i.e., unaltered voice feedback) result in attenuated responsivity of the auditory cortex, as compared to prediction-error situations in which the distorted feedback of their own voices or the undistorted feedback of non-self-generated voices (i.e., altered voice feedback) are perceived.[66,70,71] Moreover, when the perceived feedback does not match the expected one (i.e., altered voice feedback), increased temporal cortical activation is observed relative to expectation-matched feedback.[66,71]

Studies testing the corollary discharge mechanism found abnormalities in schizophrenia patients in terms of the auditory cortical responsivity to self-generated voice feedback.[65,72] The brain-response pattern associated with discriminating self-generated and non-self-generated voices seems to be altered in schizophrenia patients, regardless of symptom profile. One study found increased activation of the superior temporal gyrus (STG) bilaterally and reduced thalamic activation in a large group of schizophrenia patients (n = 63), compared to healthy controls, while they were reading aloud and receiving immediate vocal feedback of their voices (unaltered or pitch-shifted).[65] This finding suggests disturbances in the corollary discharge mechanism, reflected by abnormally reduced attenuation of auditory sensory regions in response to self-generated speech. Simons and colleagues[72] also reported—in patients with a history of prominent AVH, relative to healthy controls—both a similar failure in auditory cortical suppression during an inner-speech task and the hypoactivation of the cingulate gyrus. Since a comparison group of patients without AVH was not included in this latter study, we can ask if these abnormalities are specific to AVH patients or whether they may be found in schizophrenia patients irrespective of symptom profile.

In a study suggesting that corollary discharge abnormalities are more pronounced in AVH patients, patients with current auditory hallucinations, remitted patients, and healthy controls were given distorted feedback of their own voices during reading.[70] Increased activation of the bilateral STG was found in patients with current auditory hallucinations and delusions when they *wrongly* attributed the distorted feedback of their own voices to an external source, but found in healthy controls and remitted patients when they *accurately* identified the distorted feedback of their own voices. Similarly, Kumari and colleagues[65] reported a positive correlation between the experience of hallucinations and the hyperactivation of the right superior and middle temporal gyrus when patients were listening to self-generated and non-self-generated unaltered feedback during talking. Moreover, a different study suggested a failure of self-generated voice to result in modulation of the left STG in patients with AVH relative to patients without AVH and healthy controls.[28] During self-generated voice processing, reduced left STG activation was observed in healthy controls and patients without AVH, whereas this pattern was not found in patients with AVH.[28] Patients with AVH also failed to recruit the cingulate gyrus when hearing more ambiguous voice stimuli (i.e., distorted voices), whereas the cingulate gyrus was activated in patients without AVH and healthy controls.[28] These studies show that patients with prominent AVH tend to exhibit a distinct brain-activation pattern when compared to healthy controls; that is, they fail to show auditory cortical suppression to their self-generated voices. In addition, the results reviewed in this section suggest that abnormalities in self-generated-voice recognition in patients with more severe AVH may be related to functional disturbances in the STG.

The aberrant engagement of auditory cortical structures during tasks requiring the processing of self-generated voices in patients with AVH may reflect a failure of the corollary discharge mechanism to alert speech-perception regions about the incoming input, thereby impairing the accurate recognition of self-generated voices as self-generated. Given that the cingulate cortex is thought to be involved in attentional processes, conflict monitoring, and source monitoring of inner speech,[28] the reduced engagement of this brain region during tasks requiring higher levels of self-monitoring may contribute to the impaired ability to judge the identity of self-generated voices.

Of note, functional brain abnormalities are not restricted to the processing of self-generated versus non-self-generated voices in patients with AVH. For example, Zhang and colleagues[30] investigated familiar versus unfamiliar voice

processing in schizophrenia patients with AVH compared to patients without AVH and healthy controls. They reported altered processing of familiar voices—evidenced by increased activation of the left STG and decreased activation of the right STG—only in patients with AVH relative to healthy controls.[30]

Symptom-capture studies demonstrate that similar brain regions—including Broca's area,[73–75] STG,[73–79] and cingulate gyrus[73,75,80]—are activated during the experience of AVH (vs. remitted periods in the same patients) and during the processing of self-generated (vs. non-self-generated) speech. These findings highlight the role of dysfunctional self-generated voice processing in AVH.

Studies investigating brain structural and functional connectivity can provide important insights into both the basic (e.g., acoustic analysis of vocal signals) and more complex, higher-order cognitive processes (e.g., explicit assessment of emotional information conveyed by the voice) involved in voice processing. Recent investigations have shed some light on the relationship between AVH and disturbed neural communication among frontotemporal brain regions, which are critically involved in the perception and integration of self-generated voice signals. For instance, disturbances in the connectivity between frontal speech-production areas and temporal speech-perception areas may underlie the abnormalities in the corollary discharge mechanism by impairing the ability to predict sensory effects of self-generated voices.[81–83] Two diffusion tensor imaging studies measured fractional anisotropy, which is an index of the integrity of white matter fibers, and reported aberrant structural connectivity between frontal speech-production areas and temporal speech-reception areas. The abnormalities were reflected in increased fractional anisotropy levels in the arcuate fasciculus in patients with AVH (compared to patients without AVH and healthy controls)[81] or with auditory hallucinations in general (compared to healthy controls).[82] By contrast, Whitford and colleagues[83] found reduced fractional anisotropy levels of the arcuate fasciculus in schizophrenia patients, relative to healthy controls, pointing to a reduced integrity of white matter fibers linking frontal speech-production areas to temporal speech-reception areas. A possible explanation of these divergent results is that the latter study investigated a patient group without a specific symptom profile and did not include a group of patients with AVH.

A different study used fMRI and dynamic causal modeling to investigate self-generated versus non-self-generated voice processing in patients with AVH compared to two matched groups: patients without AVH and healthy controls.[68] Abnormally increased functional connectivity was observed between brain regions involved in source monitoring of speech (i.e., left superior temporal and anterior cingulate cortices) when patients with AVH were processing self-generated voices, whereas healthy controls and patients without AVH exhibited increased connectivity between those regions when the voice was uttered by an unfamiliar individual.[68]

The altered structural and functional connectivity between frontal, temporal, and cingulate regions may contribute to impairments in the capacity to track voice identity. In line with this hypothesis, a recent study reported reduced functional connectivity between right superior temporal and frontal regions during a voice-recognition task in patients with AVH compared to patients without AVH and healthy controls.[64] Also, disturbed frontotemporal connectivity was associated with reduced accuracy of voice-identity recognition.[64]

Studies using noninvasive neurostimulation techniques, such as repetitive transcranial magnetic stimulation (rTMS) provided further support for the involvement of disturbed brain functional connectivity in AVH.[84,85] By applying repeated pulses to evoke changes in the electrical current of particular brain areas, these studies have assessed the efficacy of rTMS as a treatment tool for AVH. Although these studies may, at first glance, appear to be beyond the scope of this subsection, the high spatial resolution of the TMS methodology (i.e., resolution of just a few millimeters) may provide important insights concerning the specific brain areas that are involved in both AVH experiences and dysfunctional voice-identity processing. Studies using this technique may also provide valuable information about the regions that are anatomically connected with particular stimulation sites and that may receive or send afferent/efferent information—which may be related to AVH maintenance and remission.[84,85] These studies have found that, after treatment with low-frequency rTMS (i.e., inhibitory stimulation) at the left parietotemporal cortex, tonic activity in the left STG and coupled regions (e.g., cerebellum, hippocampus) was reduced, whereas activity in the right STG and frontal cortex was enhanced.[84,85] Given these results, the extent to which abnormalities in frontotemporal connectivity contribute to the pathogenesis of AVH requires further investigation.

Electrophysiological Evidence

The existing studies using event-related potentials suggest altered sensory processing of self-generated voices in schizophrenia patients.[83,86–88] Several studies have measured the N1 ERP component to probe auditory cortical responsiveness during the generation of overt and inner speech, in contrast to the passive listening of self-generated speech. This ERP component is generated in the auditory cortex, indexing the sensory registration of auditory stimuli.[83] Studies in the last decade suggest that the N1 component can provide insights into the functioning of the corollary discharge mechanism.[69,83,86–94]

Studies with healthy subjects found an N1 attenuation effect (i.e., amplitude reduction) during speech production in comparison to the passive listening of a prerecorded self-generated voice.[69,83,86–92] This effect corroborates fMRI findings indicating reduced responsiveness of the auditory cortex to self-generated, relative to externally generated, stimuli.[65,66,70,71] Conversely, the N1 attenuation effect seems to

be significantly smaller in patients,[83,86,87] refuting the hypothesis that corollary discharge abnormalities are specific to patients experiencing AVH. The lack of N1 attenuation during speech production indicates that schizophrenia patients, in general, do not generate predictions about incoming self-generated voice stimuli—which may predispose patients to process self-generated and others' voices indiscriminately.

The altered auditory cortical responsiveness to self-generated voices observed in schizophrenia patients may be due to a significant delay in motor commands from frontal speech-production to temporal speech-perception regions, such as suggested by Whitford and colleagues.[83] In their study, schizophrenia patients heard their own voices immediately after pressing a button. Results revealed a reduced attenuation of N1 amplitude (i.e., higher amplitude) in patients relative to healthy controls, whereas N1 suppression comparable to healthy controls was observed when patients heard their voices with a 50 millisecond delay.[83] This study did not, however, test for correlations with clinical symptoms, such as AVH. The N1 attenuation effect observed when participants were hearing self-generated voices with no delay between production and feedback was related to increased fractional anisotropy levels in the arcuate fasciculus.

Although these studies failed to find an association between N1 anomalies and AVH, a trait study observed that patients with AVH exhibited an abnormally decreased N1 attenuation effect while they were talking aloud and receiving immediate feedback of their own voice.[69] The disturbed N1 attenuation effect occurred in AVH patients, compared to healthy controls, irrespective of whether the vocal feedback was undistorted or distorted (e.g., pitch-shifted), whereas patients without AVH did not significantly differ from the other two groups; that is, their N1 effect was in between patients with AVH and healthy subjects.[69] Thus, in line with previous suggestions,[87,89,90] it might be that alterations in the corollary discharge mechanism are associated with the vulnerability to experience hallucinations (trait) instead of the current experience of AVH (state). Studies investigating more homogeneous subgroups of patients with, versus without, AVH history may help to determine whether the N1 attenuation effect is specifically related to AVH or to a schizophrenia diagnosis.

Although just one of the above ERP studies suggests a direct relationship between the abnormal N1 attenuation effect and AVH,[69] support for disturbed frontotemporal connectivity in patients with more severe AVH symptoms is provided by electrophysiological studies looking at spatial coherence in specific EEG frequencies.[86,95] Spatial coherence is a frequency-dependent measure of simultaneous oscillatory activity between distinct neural structures, and it allows one to quantify the relationship between two brain regions.[86,95,96] Coherence in theta and delta frequencies between lateral frontal and posterior temporal regions during speech production was found to be greater in patients without AVH and healthy controls compared to patients with AVH, who exhibited reduced coherence between these brain regions, which are crucially involved in speech production and perception.[86,95] A different study investigated a different measure of neural synchrony—namely, temporal coherence preceding self-generated speech—and explored how it was associated with subsequent auditory cortical attenuation.[96] Reduced neural synchrony preceding talking (vs. listening) was observed in schizophrenia patients, especially those with AVH, when compared to healthy controls.[96] Since greater pre-speech neural synchrony was associated with subsequent attenuation of auditory cortical responsiveness in healthy subjects but not in patients,[96] these findings provide further evidence that reduced auditory cortical attenuation in response to self-generated speech plays a role in AVH generation.

The studies reviewed in the above subsections suggest alterations in the processing of voice identity in schizophrenia, with some studies indicating that these abnormalities are more pronounced in patients with AVH than in patients without AVH (see Supplemental Table 1, http://links.lww.com/HRP/A38).[23,24,28–31,33,64,65,67,70,72,81–83,87,89,90,92,95,96] This impairment does not seem to be restricted to distinguishing between self-generated and non-self-generated voices, but also extends to familiar and unfamiliar voices.[29,30,32,64]

We should note that discriminating between different voices and recognizing particular speakers represent distinct processes that recruit regions in the right parietal lobe and bilateral superior temporal sulcus, respectively.[47,54] Thus, it is important to understand whether schizophrenia abnormalities lie in voice-identity discrimination or in recognition impairments. The existing evidence suggests a more generalized deficit involving both the discrimination and recognition of voices. However, more studies are needed to support this assertion.

ARE AVH RELATED TO ABNORMAL PROCESSING OF SPEECH INFORMATION?

Abnormalities in speech-information processing are a core feature of schizophrenia,[44,104] and they are also observed in unaffected first-degree relatives of schizophrenia patients.[105]

Phenomenological reports of AVH suggest that voices might variously employ distinct levels of linguistic complexity (i.e., single words, sentences, full conversations)[10] and that they are more commonly experienced as verbal messages with negative and self-referential content.[12,15] Therefore, in view of the disturbances in processing speech information in schizophrenia and the prominent negative quality of AVH semantic content, it is plausible that abnormalities in speech-information processing are enhanced in patients experiencing AVH relative to patients without AVH.

Behavioral Evidence

Behavioral studies have demonstrated that schizophrenia patients perform poorly on a variety of semantic tasks (e.g., tasks of semantic fluency and semantic priming), which suggests that impairments in semantic processing are a general feature of the disorder.[44,104,105]

Nonetheless, a relationship between specific disturbances in speech processing and hallucinations was demonstrated by studies showing a significant association between the severity of hallucinations and both increased lexical activation[106] and more pronounced semantic dysfunction.[107] Furthermore, increased top-down semantic effects on auditory perception were observed in patients with more severe auditory hallucinations.[108,109] For instance, compared to patients without auditory hallucinations and healthy controls, patients with auditory hallucinations expecting changes in an unchanging word reported the highest number of word "transformations" (i.e., illusory perception of changes in a repeatedly presented word).[109] This finding suggests that semantic expectations may play a role in generating auditory misperceptions in AVH. Similar effects were observed in nonpsychiatric individuals with higher proneness to auditory hallucinations, as demonstrated by a positive correlation between the severity of hallucination proneness and the number of top-down errors.[4,110] That is, subjects with greater hallucination proneness tended to report hearing a word that was expected within the preceding sentence context, although the word presented was actually an unpredictable word or only noise.[4,110] The proneness to hallucinate was not associated with phonological expectations, indicating that top-down modulation of auditory percepts in individuals more prone to hallucinate might occur more prominently at the semantic level.[110] However, Daalman and colleagues[4] failed to find these effects in schizophrenia patients.

Finally, some evidence suggests that emotion plays a modulatory role in the semantic-processing abnormalities observed in patients with AVH. Difficulties in recognizing self-generated voice stimuli in patients with AVH relative to patients without AVH and healthy controls were found to be enhanced for speech stimuli with negative semantic content (vs. stimuli with neutral and positive content).[23,24,111] A similar pattern was observed in nonpsychiatric individuals with higher hallucination proneness when compared to individuals without hallucination proneness.[112] Therefore, it seems that negative emotional semantic content is more likely to interact with identity processing of self-generated voices in individuals with higher predisposition to hallucinate.

Neuroimaging Evidence

In healthy individuals, semantic information carried by the voice is processed in a segregated neural pathway, involving regions in the anterior and posterior superior temporal sulcus, predominantly in the left hemisphere.[27,47]

Earlier studies have shown differences in brain-activation patterns when comparing healthy individuals and schizophrenia patients in tasks of semantic information processing.[113–115] Functional abnormalities were found in brain networks underlying lexical-semantic processes, including abnormal engagement of left frontal and temporal regions, specifically when patients were processing highly and weakly related word pairs, as well as unrelated word pairs.[113]

Even though various studies (beyond the scope of this review) suggest an overall disturbance of semantic processing in schizophrenia, two neuroimaging studies found a relationship between auditory hallucinations and hyperactivation of brain semantic networks.[113,115] These results corroborate behavioral evidence showing an association between abnormally increased lexical-semantic activation and the severity of hallucination.[106,107] Patients with more prominent auditory hallucinations exhibited increased activation of left temporal and frontal regions to word pairs with high and low connectivity.[113] Also, during a perceptual-encoding task requiring judgments as to whether visually presented words were in upper- or lowercase letters, a correlation between the severity of auditory hallucinations and increased activation of the left superior temporal gyrus was observed in schizophrenia patients.[115] While the latter study is not directly related to the aims of the current discussion (as words were presented visually), the former study suggests a relationship between increased auditory hallucination severity and more disturbed lexical-semantic activation within left temporal regions.

Other studies have suggested that, in patients currently experiencing auditory hallucinations, external voices compete with AVH for the same neural resources. The reason is that, relative to healthy controls, patients with more severe hallucinations exhibit reduced activation in the posterior part of the left superior temporal region when processing external speech,[46] and relative to their own periods of remission, the same patients exhibit reduced activation of the right middle temporal gyrus.[116]

Of note, two fMRI studies support the role of emotion in semantic-processing abnormalities in AVH: patients with chronic AVH processed emotional information in an anomalous way, overactivating brain regions involved in semantic processing and in detecting personal and emotional salience.[117,118] In these patients, emotional semantic words elicited, compared to healthy controls, increased activation in temporal regions (left middle temporal gyrus, right STG), orbitofrontal cortex, insula, cingulate cortex, and right amygdala,[118] and elicited, compared to patients without AVH and healthy controls, increased activation in bilateral amygdala and parahippocampal gyrus.[117] Temporal cortical areas and emotion-related structures (amygdala, insula, and orbitofrontal cortex) have been previously implicated in the experience of auditory hallucinations.[119,120] The activation pattern of such areas in these studies[117,118] suggests that experiencing AVH engages the same neural resources underlying emotional semantic processing. We should note, however, that an important limitation of the study by Sanjuán and colleagues[118] is the absence of a comparison group of patients without AVH—which necessitates a careful interpretation of these data.

Indirect evidence for the involvement of semantic abnormalities in AVH comes from fMRI studies investigating verbal self-monitoring, since most of them used words and

sentences as stimuli. Considering the role of the left STG in semantic comprehension (e.g., Wernicke's area), the finding of enhanced activation of the left STG in response to self-generated speech may point to AVH patients' aberrant processing of speech information contained in self-generated voices (as observed in earlier studies: Allen et al. [2007],[22] Kumari et al. [2010],[65] and Simons et al. [2010][72]). If so, the potential implication is that emotional and self-referential contents are more deeply processed by semantic and emotional brain regions (in line with previous findings: Escartí et al. [2010][117] and Sanjuan et al. [2007][118]). The enhanced processing of emotional versus neutral semantic information in self-generated voices may help to explain the higher salience of negative and personal information underlying AVH. In addition, as some behavioral studies have demonstrated, increased vulnerability of auditory perception to top-down expectations and semantic effects in patients with more severe AVH[107,108] may contribute to the aberrant processing of semantic information with negative valence.

Electrophysiological Evidence

Electrophysiological studies investigating semantic processing in schizophrenia have measured the N400 ERP component, a negative-going component elicited at around 400 milliseconds after stimulus presentation.[94,121,122] This ERP component reflects general semantic processing of meaningful stimuli (e.g., speech stimuli, pictures, faces) and can be used to probe semantic memory.[121–123] N400 amplitude changes inversely to the degree of predictability of a stimulus in its context.[121–123] Most of the schizophrenia studies have used visual stimuli and reported semantic-processing disturbances. A reduced difference in N400 amplitude between congruent and incongruent conditions suggests that schizophrenia patients process related and unrelated concepts similarly.[45,93,94,105,121,122] Studies of semantic processing in the auditory modality—ones that will take advantage of the high temporal resolution of ERPs—are sorely needed.

Although not directly related to vocal semantic processing abnormalities in AVH, it is worth noting that some of the visual N400 studies found an association between abnormal semantic processing in schizophrenia (reflected in larger N400 amplitude to targets that related to their priming stimuli) and positive symptoms such as hallucinations and delusions.[122,124] This association points to the role that abnormalities in semantic network activation might play in the experience of hallucinations—which is consistent with neuroimaging findings.[113,115] To our knowledge, only one study assessed auditory semantic processing in schizophrenia, and it reported abnormalities that were similar to those reported in the visual modality, even though no association was found between AVH and semantic-processing deficits.[45] Although the studies discussed in this section have made important contributions (see Supplemental Table 2, http://links.lww.com/HRP/A39),[4,45,46,97–103,106–111,113,117,118,125] studies testing the relationship between speech processing and AVH are lacking. In addition, many of the existing studies did not assess the contribution of hallucination severity to semantic abnormalities.[107,126] It is therefore possible that AVH might mediate that relationship.[107]

ARE AVH RELATED TO ABNORMAL PROCESSING OF VOCAL AFFECTIVE INFORMATION?

The effective processing of vocal affective stimuli implies the ability to extract relevant acoustic information from speech, such as intensity, pitch, and timbre.[27,47,127] The nonverbal vocal expression of emotion in a speech signal is designated as emotional prosody,[127,128] and its proper identification is one of the cornerstones of adequate functioning in social environments.[127]

The perception of emotional information in speech is a complex process involving sensory-, cognitive-, and emotional-processing systems and multiple interacting stages.[127] These stages include the sensory processing of the vocal stimulus, the detection of its emotional salience, and the cognitive evaluation of emotionally significant information.[127] Considering that AVH are often experienced as having negative emotional intonation, such as a taunting or menacing tone, it would make sense to predict more pronounced abnormalities in vocal affective processing in patients with AVH, compared to patients without AVH.

Behavioral Evidence

Behavioral studies have consistently demonstrated impairments in the ability of schizophrenia patients to recognize vocal affective information, as reflected in increased error rates in recognition tasks.[35,36,40,42,62,129–136] Some found specific abnormalities in the recognition of negative emotions, such as anger and sadness.[42,43] The contribution of basic auditory-processing deficits (e.g., pitch discrimination) to impaired vocal affective processing[35,36,132,136] has been highlighted.

It remains controversial, however, whether affective prosody abnormalities are present in schizophrenia patients regardless of the subtype[35,36,62,130–133,137] or whether more pronounced abnormalities are associated with, or limited to, specific clinical symptoms, as observed in more homogeneous samples of patients with, versus without,[34,37,63] AVH. For example, a recent study found an association between the severity of positive symptoms (hallucinations and delusions) and impaired recognition of vocal and facial affect.[135] In a different study, Rossel and Boundy[37] observed that, although all schizophrenia patients exhibited deficits in perceiving auditory affective stimuli with semantic content, only patients with a history of AVH, compared to patients without AVH or healthy controls, showed deficits in recognizing meaningless affective sounds. This finding suggests that semantic processing disturbances may obscure differences between patients with and without AVH.

Along the same lines, Shea and colleagues[63] observed that patients with AVH, relative to patients without AVH and healthy controls, exhibit increased difficulties in identifying the affective prosody of semantically neutral sentences uttered

with happy, sad, and neutral tones. Furthermore, the authors proposed that the inability of patients with AVH to effectively process affective prosodic cues in the vocal signal might contribute to the impaired monitoring of voice identity, which, in turn, may prime the misrecognition of internal events (i.e., thoughts) as being produced by an external source.[63]

Importantly, different variables might have contributed to the apparent contradiction between studies reporting abnormalities in vocal affect recognition in schizophrenia patients and those reporting results in more homogeneous samples of patients with AVH. The former studies did not specify the schizophrenia subtype[35,36,132,136,137] and included patients with schizophrenia and schizoaffective disorder in the same group;[35,36,62,131] this heterogeneity in the samples may have obscured the relationship between AVH and disturbed vocal-emotion recognition. The link between AVH and abnormalities in vocal affective processing may also be obscured by the instruments (e.g., Positive and Negative Syndrome Scale)[99] used in those studies,[35,36,62,131,132] since the instruments do not clearly discriminate auditory from visual hallucinations. While vocal affect recognition might be relatively altered in schizophrenia patients, partial evidence suggests that more pronounced abnormalities are limited to the subgroup of patients with AVH.[37,63,135] However, disentangling which aspects of vocal-affect recognition are impaired in schizophrenia patients or are specific to AVH patients still needs further clarification. The correlation between positive symptoms (hallucinations and delusions) and abnormal recognition of happy prosody may be indicative of specific alterations in processing happy tones in patients experiencing hallucinations, especially because the researchers failed to find that association when overall mean recognition rates were analyzed.[135] Therefore, the experience of AVH might be more strongly associated with impairments in specific categories of vocal affect, although differences in the complexity of the stimuli used (nonverbal vocalizations, as in Tseng et al. [2013][135] vs. sentences uttered with distinct emotional tones, as in Shea et al. [2007][63]) preclude any robust conclusion.

Providing support for the specific relationship between abnormal vocal affective processing and the experience of AVH, a recent dichotic listening study revealed that automatic attention in AVH patients is aberrantly modulated by the emotional prosody of vocal stimuli.[138] In that study, each trial in the emotional conditions comprised both an affective and a neutral vocalization, with each vocalization being concomitantly presented to the other ear. In the neutral baseline condition, each trial contained two neutral vocalizations. Participants were required to judge the gender of the voice stimulus presented to the attended ear (right or left) while ignoring the stimulus presented to the other. As expected, healthy controls were slower in deciding the gender of voice stimuli when the vocalizations presented to the attended right ear had an emotional tone. The same finding was observed in patients without AVH, indicating increased interference (i.e., slower response time) on the voice-gender task when emotionally salient cues were presented to the attended right, versus left, ear. This finding suggests a right hemisphere advantage (i.e., attended left ear) for the processing of both vocal affect and voice gender. It also suggests that, for the nonspecialized left hemisphere (i.e., attended right ear), emotional salience of vocal stimuli that are in the focus of attention is more difficult to ignore and leads to behavioral distraction regarding the gender-discrimination task, resulting in increased reaction time. Nonetheless, the performance of patients with AVH was worse (i.e., slower reaction times) irrespective of whether vocal affective stimuli were presented to unattended or attended ears, or to the left or right sides. These findings suggest that patients with AVH do not show the right hemisphere advantage for vocal affective processing that is observed in patients without AVH and healthy controls. In addition, the findings suggest that patients with AVH are more sensitive to the affective salience of stimuli, with attention being automatically attracted by vocal affective information even in conditions in which emotion is presented to the "'supposed" unattended channel or to the brain's right hemisphere, which is known to be specialized in both vocal affect and gender processing.[138] Considering that patients with AVH were faster during the neutral baseline condition relative to the emotional conditions and that accuracy levels did not significantly differ across the three groups, these findings seem to reflect not a general impairment in the voice-gender decision task but, instead, a reduced capacity to ignore affectively salient voice cues. Since AVH are more often described as having negative content and prosody, it is plausible that attentional disturbances contribute to the enhanced processing of negative vocal affect of inner voices in AVH patients and to the disrupted capacity of directing attention to other prosodic features.

Neuroimaging Evidence

In healthy individuals, the decoding of the speaker's affective state recruits the temporo-medial regions, anterior insula, amygdala, and inferior prefrontal cortex more predominantly in the right hemisphere.[27,47]

The neuroimaging evidence on vocal affective processing in schizophrenia is still scarce, yet some studies suggest that, irrespective of symptom profile, patients exhibit abnormally reduced activation of the right temporal cortex and increased recruitment of left insula,[139] and aberrant activity in right temporal and parietal cortices.[140]

Evidence linking AVH to alterations in the neural substrates of vocal affective processing in schizophrenia comes from studies showing functional and structural abnormalities in brain structures recruited during both vocal affective comprehension[117,118] and the experience of AVH.[73,74] For example, the neural mechanisms underpinning the processing of vocal affective information seem to be altered in patients with chronic AVH, as demonstrated by studies showing increased activation in the left middle temporal gyrus, right STG, orbitofrontal cortex, insula, cingulate cortex, and right

amygdala relative to healthy controls,[118] and by increased activation of the amygdala and parahippocampal gyrus in these individuals relative to patients without AVH and healthy controls.[117] Of note, however, a recent study reported underactivation of the amygdala when patients with AVH, versus patients without AVH or healthy controls, were listening to crying vocalizations, indicating that the persistence of AVH is associated not only with impaired emotional speech prosody perception but also with abnormal processing of nonverbal vocalizations.[141] The divergent results concerning amygdala activation may be due to differences in the stimuli used, since the latter study[141] used nonverbal emotional vocalizations, and the former studies selected words based on reports of AVH experiences. Indeed, words have higher complexity (carrying both semantic and emotional prosody information) and ecological validity (the imperative tone and the emotional content may closely correspond to the voices heard during AVH) than vocalizations. In addition, the decreased activation of the amygdala in patients with chronic AVH while listening to crying sounds was hypothesized as associated with the severity and long duration of the patients' auditory hallucinations.[141] The reduced activation of the amygdala to emotional stimuli may also reflect increased tonic activation of that region.[34,141,142]

When compared to remitted periods, the experience of AVH is associated with the increased activation of temporal lobe regions (in the left STG,[74,75,77] right STG,[78,79] or both[76]), inferior frontal regions,[73,74] and emotion-processing regions, such as the amygdala,[73,74] insula,[73,74] orbitofrontal cortex,[80] and parahippocampal gyri.[80] At the structural level, gray matter reductions in the STG,[142,143] insula, and left amygdala[143] were observed in patients with prominent AVH, relative to healthy controls. Moreover, the connectivity between frontotemporal and limbic regions was found to be increased in patients with AVH, as larger fractional anisotropy levels in the left cingulate bundle were reported in AVH patients relative to patients without AVH.[81] The enhanced structural connectivity between frontotemporal and limbic regions in patients with AVH may lead to abnormal activation in voice- and emotion-processing regions, possibly contributing to impaired integration of salient vocal affective information. When applied to internally generated voices, the above structural-connectivity abnormalities might contribute to the commonly reported negative affective tone of AVH.

Electrophysiological Evidence

Few studies have examined the ERP correlates of emotional prosody processing in schizophrenia.[38,41] Using both sentences[41] and single words[38] as stimuli, these studies reported N1 and P2 abnormalities in schizophrenia, indicating alterations in two stages of emotional prosody processing: the sensory processing of a prosodic signal (N1) and the detection of the emotional salience of a vocal stimulus (P2). ERP abnormalities were enhanced for speech stimuli with intelligible versus unintelligible semantic content. Of note, no association was found with AVH severity. It is also worth noting that abnormally large P2 amplitude for happy prosody in sentences with intelligible semantic content was associated with delusions, suggesting a relationship between abnormal detection of voice salience and a specific positive symptom.[41] As the authors of the study observed, further research with a larger, more homogeneous sample would help to clarify the relationship between ERP indices and positive symptoms. Abnormalities in the integration of emotionally salient vocal cues (which would lead to the proper identification of the prosodic content of vocal stimuli), along with structural and functional abnormalities in the posterior part of the STG, may contribute to the experience of AVH.[34]

Although few studies have investigated the relationship between AVH and the processing of vocal affective information, some behavioral and neuroimaging studies suggest that an inability to "read" emotions from a tone of voice may potentially play a role in the development of AVH (see Supplemental Table 3, http://links.lww.com/HRP/A40).[35,37–41,59,62,63,81,97–103,117,118,130–138,141–143] Further research, at both the behavioral and brain levels, is much needed. Moreover, as abnormalities in the processing of vocal affective information may be associated with the severity and long durability of auditory hallucinations,[141] it is important to investigate vocal affective processing in patients with distinct levels of AVH severity and duration.

DISCUSSION

Conclusions and Recommendations

The studies reviewed here provide evidence for significant alterations in the three distinct domains of voice processing in schizophrenia—speech, identity, and affective information.[27,47] They suggest generalized voice-processing abnormalities that are not restricted to specific dimensions, contrary to other clinical conditions in which dissociations between these distinct dimensions are observed (e.g., phonagnosia). Impaired capacity to process voice identity in patients with AVH, especially of self-generated voice, involves alterations in the function of the STG and cingulate gyrus, as well as structural and functional connectivity abnormalities between frontal, temporal, and cingulate cortices. The relationship between the experience of AVH and alterations in the corollary discharge mechanism was suggested by a few EEG/ERP studies. Patients with more severe AVH seem more likely to exhibit more pronounced brain alterations during the processing of speech information conveyed by the voice (e.g., aberrant lexical-semantic processing, greater sensitivity to top-down semantic effects during auditory perception, and anomalous modulation of self-generated voice processing by negative emotional information). These alterations have been linked to functional abnormalities in the brain's semantic network, particularly involving left STG regions, as evidenced by N400 anomalies. Disrupted recognition of vocal affect in patients with AVH, compared to patients without AVH, has

been associated with increased functional abnormalities in the temporal cortex and limbic regions (especially the amygdala), when compared to patients without AVH. Some evidence from ERP studies suggests that disturbed vocal affective processing might be specifically related to abnormal detection of salience from vocal cues. Brain regions underlying the experience of AVH appear to overlap with brain regions normally engaged during voice-information processing, further supporting the relationship between both phenomena. Based on the existing evidence, the association between AVH and specific dimensions of voice processing seems to be stronger for the dimensions of identity[23,24,26,28–30,32,33,64,68,69] and speech,[46,106–108,111,116–118] but further studies are needed (see Figure 1).

Correlational findings do not establish causality; third variables may be moderating such associations. Nevertheless, the findings presented here do suggest an important relationship between AVH and disturbed dimensions of voice processing. Some of the trait studies also point to such a relationship. While the nature of this relationship needs further clarification, some phenomenological aspects of AVH may reflect specific impairments in voice-information processing. The experience of voices as spoken by an external agent in AVH may reflect an anomalous processing of the *identity* of self-generated voices (including inner voices/inner thoughts). Dysfunctional processing of emotional *speech* information may contribute to the highly negative and derogatory content of AVH. Emotional alterations (high levels of anxiety, depression, and anger), along with associated negative thoughts about the self that precede the onset of AVH,[14] may direct attention to negative emotional contents. Finally, disturbances in the processing of vocal *affective* information might lead to the prominently negative tone of AVH. Increased automatic attention to negative prosodic cues may explain why hallucinated voices are often perceived as uttered with a negative emotional tone.

Considering that the verbal content of voices may vary substantially from patient to patient, it is critical to further specify the phenomenological aspects of AVH in studies probing this symptom (e.g., whether voices are uttered by a specific familiar or unfamiliar entity; whether voices are talking with each other; whether voices are commenting on what the patient is doing). Furthermore, since AVH are commonly experienced as a form of communication from another "speaker" (e.g., as a command or a comment about the subject) and therefore represent a type of "social experience,"[144]

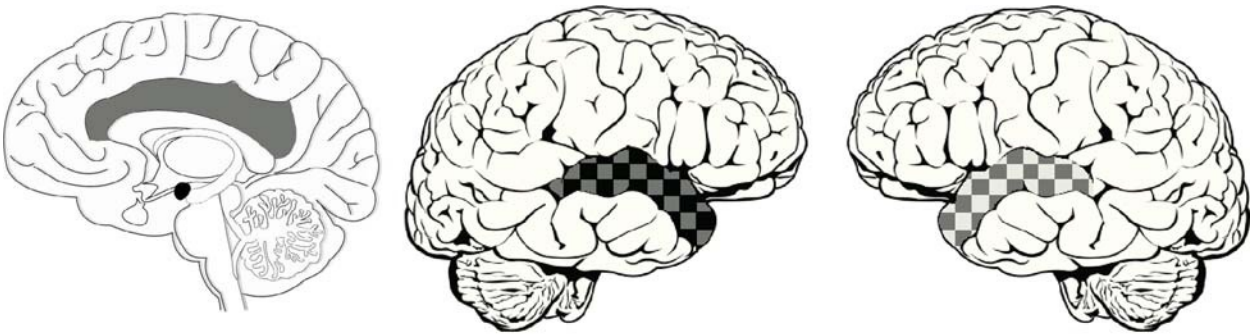

Figure 1. Brain regions altered in schizophrenia patients with AVH and involved in different dimensions of voice processing. STG, superior temporal gyrus.

the social features of AVH should be a major focus of attention for researchers investigating hallucinations (e.g., how do patients perceive the "social power" of AVH, and how do they interact with their voices?).

Because the existing evidence is insufficient for drawing specific conclusions about the association between the nature and degree of voice-processing abnormalities and the phenomenological features of AVH, further research is seriously needed. Future studies should test whether patients reporting AVH as pertaining to specific and differentiated "personae" also display greater impairments in the perception of voice identity, especially of self-generated voice identity, or whether patients experiencing AVH as uttered by a nonspecific external agent (e.g., voices resembling the patient's voice) exhibit more preserved processing of self-generated voices. Along the same lines, the relationship between the (in)ability to perform personality judgments (e.g., dominance, masculinity, friendliness) and the tendency to experience AVH as originating from a male and powerful identity should also be explored. At the level of vocal speech information, it is relevant to test whether the degree of derogatory and self-referential content in AVH is associated with more pronounced abnormalities in the processing of verbal stimuli with emotional semantic content. Finally, studies looking at the relationship between AVH and disturbed vocal affective processing might examine whether patients experiencing hallucinated voices uttered in a more negative emotional tone also exhibit increased abnormalities in vocal affective processing.

Nonetheless, it is also possible that altered processing of voice information may occur as a nonspecific consequence of AVH pathophysiology. If so, impairments in voice-information processing should be targeted by future treatment programs for AVH, given the relevance of voice-processing abilities in daily social communication and overall functioning. This topic may be addressed in longitudinal studies tracking changes in both AVH and the ability to process voice information, from the prodromal to chronic stages, all in comparison to individuals in the same stages but with no AVH. Another aspect that remains poorly understood is the interaction between distinct dimensions of voice processing and how each might specifically contribute to the experience of AVH. It is plausible that impairments in one dimension contribute to abnormalities in another. For example, emotional semantic information seems to contribute to the misrecognition of voice identity,[23,24,111] and semantic processes may modulate disturbances in vocal affective processing.[37,38,41]

Limitations

Despite support for the role of disturbed voice processing in AVH, the current picture on the mechanisms underlying the experience of AVH is still puzzling. Therefore, the findings presented here should be interpreted with caution. An important limitation of the studies reviewed has been the use of non-homogeneous inclusion criteria, such as the classification of patients as hallucinating versus non-hallucinating.[24,68,69,109,117] In particular, the apparently conflicting results when testing the relationship between AVH and voice-processing abnormalities (e.g., Allen et al. [2007],[22] Allen et al. [2004],[33] Chhabra et al. [2012],[31] Johns et al. [2006],[24] Vercammen & Aleman [2010],[110] Whitford et al. [2011][83]) may be due to differences in sample composition. For example, in the 2007 study by Allen and colleagues,[22] non-hallucinating patients had to have no current or previous history of AVH, whereas in the study by Alba-Ferrara and colleagues,[29] non-hallucinating patients had no current AVH and a score ≤ 1 in the hallucination global score in the Scale for the Assessment of Positive Symptoms (SAPS),[97] but previous AVH history was not mentioned. In the study by Chaabra and colleagues,[31] the criteria for classifying patients as hallucinating versus non-hallucinating were not clearly stated; in Zhang and colleagues' study,[30] hallucinating patients had to score ≥ 4 in the SAPS auditory hallucination item, and AVH should had been experienced in the two weeks before the assessment; and in Allen and colleagues' 2007 study,[22] hallucinating patients had to score ≥ 3 in the SAPS auditory hallucination item, and AVH should had been experienced in the last week. Because of these differences, particular groups of patients could be classified as hallucinators in one study and as non-hallucinators in another. Moreover, most of the studies used small samples, which might increase the likelihood of a type II error: the reduced number of participants might compromise the power to detect differences, thus leading to the acceptance of the null hypothesis. Finally, a significant number of the studies did not specify the schizophrenia subtype (e.g., Hubl et al. [2004],[81] Kumari et al. [2010][65]), which may have resulted in high sample heterogeneity.

Importantly, some of the instruments (e.g., SAPS),[97] used in the reviewed studies do not clearly separate different types of auditory hallucinations and their phenomenological features, or do not even separate auditory from non-auditory hallucinations (e.g., PANSS,[99] Brief Psychiatric Rating Scale[100]). Hence, the resulting heterogeneity may bias any conclusions regarding the extent to which abnormalities in voice-information processing are related to specific symptoms. Also, whereas most of the reviewed studies have specifically focused on the experience of AVH, others did not detail the type of auditory hallucinations experienced by schizophrenia patients.

The absence of a healthy control group in some of the studies[107] might also obscure the interpretation of the findings. Since some studies also did not include a control group of patients without a history of AVH (e.g., Sanjuan et al. [2007],[118] Simons et al. [2010][72]), one may ask whether such findings were due to another feature of the disorder. As most patients were taking typical or atypical antipsychotics, we cannot rule out the effects of medication on the data reviewed here. Further studies with antipsychotic-naive patients may contribute

to the clarification of these effects. In addition, a relevant aspect to be examined is the integrity of voice processing in different stages of the disease (i.e., high-risk individuals, and first-episode and chronic patients) and its relationship to AVH symptoms. This approach would allow us to better understand and track changes in voice processing during the course of the disease.

Aware of these limitations, we hope that the current and future insights from cognitive neuroscience research will open a wider window that sheds light on the complex processes underlying the experience of AVH. The evidence linking AVH with abnormalities in voice-information processing might inspire novel inroads toward a unified model of AVH.

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