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Auditory verbal hallucinations of epileptic origin

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ABSTRACT

Complex auditory hallucinations are often characterized by hearing voices and are then called auditory verbal hallucinations (AVHs). While AVHs have been extensively investigated in psychiatric patients suffering from schizophrenia, reports from neurological patients are rare and, in most cases, incomplete. Here, we characterize AVHs in 9 patients suffering from pharmacoresistant epilepsy by analyzing the phenomenology of AVHs and patients' neuropsychological and lesion profiles. From a cohort of 352 consecutively examined patients with epilepsy, 9 patients suffering AVHs were identified and studied by means of a semistructured interview, neuropsychological tests, and multimodal imaging, relying on a combination of functional and structural neuroimaging data and surface and intracranial EEG. We found that AVHs in patients with epilepsy were associated with prevalent language deficits and damage to posterior language areas and basal language areas in the left temporal cortex. Auditory verbal hallucinations, most of the times, consisted in hearing a single voice of the same gender and language as the patient and had specific spatial features, being, most of the times, perceived in the external space, contralateral to the lesion. We argue that the consistent location of AVHs in the contralesional external space, the prominence of associated language deficits, and the prevalence of lesions to the posterior temporal language areas characterize AVHs of neurological origin, distinguishing them from those of psychiatric origin.

1. Introduction

Auditory hallucinations cover a variety of elementary experiences, such as hearing noises or sounds, and complex experiences, such as hearing music or voices. Complex auditory hallucinations are most often characterized by the hearing of a voice or voices that are generally called auditory verbal hallucinations (AVH). About 70% of patients with schizophrenia and a variety of psychiatric and neurological patients suffer from AVHs [1]. While literature on AVHs in psychiatric patients is extensive [2–4], only a few reports are available on AVHs in neurological patients [5–8].

Several functional and neural mechanisms have been proposed to account for AVHs. Auditory verbal hallucinations have been attributed to 1) aberrant perceptions of altered auditory processing [9]; 2) language-related deficits such as a failure in self-attributing inner speech (self-monitoring deficit) [10]; and 3) a memory deficit, i.e., abnormal remembering of memories of speech [11], among others [12]. Neuroimaging studies in psychiatric patients have provided evidence in favor of all these hypotheses. During AVHs, some studies revealed altered activations in auditory cortices [13–15], other studies described activations of Broca's and Wernicke's areas [16,17], whereas others reported activation in the hippocampus/parahippocampus memory regions [18] (see [19] for a review).

Auditory verbal hallucinations in neurological patients may depend on focal or distributed brain damage and may be associated with selective neuropsychological deficits (i.e., auditory, language, or memory deficits). Auditory verbal hallucinations in neurological patients have been reported most often in spontaneous seizures and been localized to the temporal cortex [7]. Auditory verbal hallucinations may also be evoked directly by electrical cortical stimulation (ECS) in patients with medically intractable temporal lobe epilepsy. Using this technique, Penfield localized AVHs to the superior and middle temporal gyri of either hemisphere with a left-sided predominance [6,9]. Halgren et al. confirmed these findings but also induced AVHs by ECS of inferior temporal and mesial temporal structures [8]. However, neuropsychological results have not been reported in any of the above studies, and a complete analysis of the phenomenology of AVHs in neurological patients is lacking. Here, we tested for the presence of AVHs in a sample of 352 consecutive patients hospitalized for presurgical epilepsy evaluation. We found 9 patients with AVHs and analyzed AVH phenomenology and the associated neurological, neuropsychological, and neuroanatomical profiles.

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2. Methods

2.1. Subjects

From a sample of 352 patients with pharmacoresistant epilepsy evaluated in the Geneva-Lausanne Presurgical Epilepsy Evaluation Center, we selected patients (N = 9; ≈3%) with paroxysmal AVHs. For each patient, a short summary of clinical findings is given in Table 1. A case-by-case detailed description of patients' clinical history, AVHs, and neuropsychological profile is available as Supplemental material online (S1). The study was approved by the ethical committee of the University Hospital of Geneva.

2.2. Etiology

Four (44%) AVH patients had tumoral lesions (glioneuronal tumor, DNET). Two (22%) AVH patients had hippocampal sclerosis that was associated with lateral temporal cortical lesions. One AVH patient had severe cortical dysplasia, one had glioneuronal heterotopias, and one had nodular periventricular heterotopia.

2.3. Phenomenology

Each case was analyzed by means of a semistructured interview, which recorded specific information about the auditory experiences, as detailed previously [20,21] plus a series of questions from recent questionnaire studies about the AVHs of psychiatric patients [22]. Questions are listed in Supplemental material online (S2). Briefly, we focused on the following several dimensions of AVHs: 1) number of voices, gender, and language of the voices; 2) content and emotional valence; 3) spatial features; 4) frequency and time of AVHs; and 5) associated nonauditory experiences.

2.4. Clinical and neuropsychological examination

In each patient, a complete neurological examination and an extensive neuropsychological (language, attention, executive functions, and memory; see Table 3) and psychiatric examination were carried out. Language was tested by means of Boston Naming Test and Semantic Fluency Task; Attention with Trail Making Test; Short-term Memory with the Digit Span for the verbal component and the Corsi Span for the spatial component; Long-term memory with the Rey 15-word Verbal Memory Test for the verbal component and the Rey 15-item Visual Memory Test for the spatial component; and Executive Functions with Phonetic Fluency and the Stroop Test. This battery was administered for interictal evaluation. Immediately after seizures (postictal), a short version of the battery was also administered. References for French versions of the applied tasks are reported in Supplemental material online (S3).

2.5. Surface and intracranial EEG, electrical cortical stimulation

Continuous long-term video-EEG recordings with 29 scalp and 2 sphenoidal electrodes were carried out in all patients. Two patients were further investigated using subdural grid recordings (Ad-Tech, USA) because of inconclusive noninvasive investigations. Subdural electrodes and electrical stimulation were used as described before [20,21].

2.6. Neuroimaging and lesion analysis

Magnetic resonance imaging (MRI) was available in 9 (100%) patients, positron emission tomography (PET) in 8 (88%) patients, and ictal and/or interictal single photon emission computed tomography (SPECT) in 7 (77%) patients.

The epileptogenic zone was identified using a multimodal imaging approach as described previously [23,24], relying on a combination of functional and structural neuroimaging data, surface and intracranial EEG and ECS data, and MRI, PET, and SPECT scans. Brain scans were normalized to the smoothed T1 template. Intracranial EEG was coregistered to the normalized MRI scans, and the lesion site was defined as the location of those implanted electrodes (on the standard T1 template) where AVHs could be induced (plus an additional radius of 10 mm around the corresponding electrode). Lesions were subsequently traced manually using MRIcron (http://www.sph.sc.edu/comd/rorden/mricron) [25] (see Supplemental material online for details; S4).

3. Results

3.1. Auditory phenomenology

3.1.1. Content, language, and emotional features

Phenomenology results are reported in Table 2. Seven AVH patients only heard voices during auditory hallucination, 2 (22%) patients also heard sounds, and 1 patient heard music. Two (22%) patients heard multiple voices, whereas the other 5 (55%) patients heard a single voice. In the latter cases, the voice was a male voice in 5 (55%) patients and a female voice in 2 patients. In 6 (86%) out of the 7 patients who could indicate the gender of the voice, the hallucinated voice had the same gender as that of the patient.

Three (33%) AVH patients heard a negative voice, 2 heard an “authoritative” or “serious” voice, whereas the other AVH patients did not report this information or were not questioned specifically on this. The voice was familiar in 5 (55%) AVH patients and unfamiliar in 2 (22%) patients, while 2 patients were not able to indicate or were not questioned. Speech was comprehensible for 4 (44%) patients and incomprehensible for the other 4 (1 patient could not specify). When speech was comprehensible, the language used was the same as the patient's mother tongue. Patient 9 reported to hear her mother speaking in her local dialect during AVHs (provoked by ECS).

Table 1
Demographic and clinical data of AVH patients. Patients' mean age was 32.7 years (range: 22–44 years; 4 females). The mean age of onset of epilepsy was 14.4 years (range: 5–31 years), and the average duration of epilepsy was 18.2 years (range: 8–32 years). PD = personality disorder.

Patient	Age at evaluation (years)	Age of onset (years)	Gender	Handedness	Duration of epilepsy	Psychiatric symptoms
A-1	30	5	F	R	25	No
A-2	33	9	F	R	24	Narcissistic PD
A-3	27	12	F	Ambidextre	15	Schizotypic PD
A-4	22	7	F	R	15	No
A-5	32	7	M	R	25	No
A-6	39	31	M	R	8	Schizophrenia
A-7	30	18	M	R	12	Narcissistic PD
A-8	37	29	M	R	8	Depression
A-9	44	12	M	R	32	Emotional lability