

Transient musical hallucinosis of central origin: a review and clinical study

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Abstract

A 52 year old, right handed, hearing impaired woman was admitted with headache and neck stiffness. The only neuropsychological symptom was transient auditory perceptions in the left ear, which were musical, seemed familiar and were not influenced by verbal communication. CT and MRI showed a right subarachnoid haemorrhage, while brainstem auditory evoked potentials failed to reveal a brainstem lesion. In patients with organic cerebral disease, unilateral auditory hallucinations (AHs) may indicate a lesion in the contralateral hemisphere. However, according to this review the type of AHs (verbal versus musical) is not consistently associated with a cerebral lesion on either side.

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Bryant⁵⁵ stated that unilateral AHs are unquestionably due to unilateral ear disease. On the other hand, Bergman⁵⁶ and Tanabe *et al*¹⁵ assumed that, in patients with organic brain disease, unilateral AHs could be considered a reliable indication of contralateral cerebral pathology. Berrios⁵⁷ emphasised the important role played by the non-dominant hemisphere in the development of AHs of the musical type.

We studied a hearing impaired patient who developed a transient unilateral auditory hallucinosis in her ear, after a contralateral right subarachnoid haemorrhage.

Case report

A 52 year old, right handed woman of normal intelligence, presented with a previous medical history of multiple streptococcal sore throats complicated by acute rheumatic arthritis in childhood, a bilateral asymmetrical hearing loss, common migraine attacks for several years, a thrombo-embolic small intestinal infarct at the age of 49, gastrointestinal blood loss three months before the current event, a cardiac murmur based on mitral and aortic insufficiency, and hypertension. She admitted misuse of ergotalkaloids in the past. There was no history of overconsumption of alcohol.

In July 1988, she developed sudden right temporal headaches, progressively increasing and accompanied by an aching stiffness of the cervical spine. She looked ill, her right eye was slightly proptotic and she could not move her neck without great pain. She complained of paresthesiae in both upper limbs. Her blood pressure was 200/70 mm Hg. The rest of the neurological examination was normal. Paracetamol made her feel more comfortable.

Two days later, she started complaining of hearing music in her left ear. The music began suddenly at 11 pm, and was of such clarity that the patient thought that the radio was playing in the hospital corridor. After asking the nurses about the noise and then lying down on her better ear, she realised that the music was coming from inside her head. From that moment, she was always aware of the hallucinatory character of the musical perceptions. She described the music as popular classical, comprising orchestral arrangements with occasional vocals. She always recognised the tunes, which never bored her, although she sometimes wished for respite. They varied, and did not repeat themselves constantly. On rare occasions the contents of the hallucinations were distorted. Occasionally she heard bells

Auditory hallucinations (AHs) are auditory perceptions that occur in the absence of a corresponding external acoustic stimulus.¹ The subject may or may not be fully aware of their imaginary nature.²⁻⁴ AHs may be either elementary, for example, tinnitus, whistles, buzzing; or complex, such as music, voices or spoken words.⁵⁻⁶ AHs may occur in isolated form, but some are associated with other hallucinations, for instance, visual or tactile.⁷⁻¹² AHs may be perceived in either, or both ears.¹³⁻¹⁵ AHs have been described in different conditions: several psychiatric disorders,^{1-14 16-30} prolonged alcoholism,¹ intoxication with drugs and other chemical agents,^{1 36-41} hypertension,⁴² several neurological disorders including migraine^{43 44} and epilepsy,^{1 42 45-49} and ear diseases.^{13 16 50-53} The exact origin of AHs remains unclear. From a physiological and biochemical viewpoint, AHs may result from an abnormal brain excitability or a central disinhibitory factor, and from defective neurotransmission mechanisms.^{1,6} Some investigators^{13 50-52} claim that neural or end organ disease alone is sufficient to produce AHs, thus postulating sensory deprivation to be the primary neurophysiological mechanism. Others^{16 53} maintain that a combination of peripheral and central dysfunction is required.

Hécaen and Ropert⁴² argued that lateralisation of AHs has no localising value in hallucinosis—that is, organic hallucinations of central origin, but would consistently implicate the worst ear when of otopathic origin.⁵⁴

chiming. She heard music when she awoke during the night, and when talking. The hallucinations occurred consistently, whether or not she used the hearing aid in her left ear. Only twice did she experience verbal hallucinations, comprising unknown voices speaking whole sentences which she could not understand. She did not experience hallucinations in other modalities. A few days later, the perceptions lessened in intensity, and were sometimes superseded by the ticking of a clock. The hallucinations disappeared gradually over the following days and three weeks after admission, the patient was free of them. On re-examination three months later, she had no complaints and her neurological status was normal.

Clinical investigation at the time of the hallucinations found no evidence of major psychiatric disorders. Laboratory tests did not disclose any intoxication.

A CT examination performed two days after admission demonstrated the hyperdense aspect of the right sylvian fissure and the right parietal sulci, presumably reflecting localised subarachnoid haemorrhage (SAH). A cerebral angiography showed vasospasm of the operculofrontal branches of the right middle cerebral artery. An aneurysm could not be identified. CT scans obtained 10 and 20 days after admission showed progressive disappearance of the hyperdense aspect of the right sylvian fissure. CT also disclosed the presence of old, bilateral capsuloputaminal lacunar infarcts. MRI revealed an area of increased signal intensity on a sagittal T1-weighted image through the right sylvian fissure, indicating subacute localised SAH (fig.).

The day before the onset of the musical hallucinations, the EEG showed a symmetrical recording which was within normal limits. There was no evidence of epileptic activity nor of intoxication. Brainstem auditory evoked potentials (BAEP) at the time of the musical perceptions were normal.

Ear, nose and throat examination at the time of the auditory phenomena revealed a perceptive hearing loss on the left with a Fletcher

index of 45 dB, and a mixed perceptive-conductive hearing loss on the right, Fletcher index, 90 dB. Otoscopy revealed a normal left drum and a posterior retracted drum on the right with a suspected incudostapedial dislocation. However, her otological status had remained unchanged since 1986.

Neurolinguistic and neuropsychological examinations at the time of the hallucinations failed to reveal any signs of aphasia, apraxia, agnosia, visuospatial disorders or hemispatial neglect. The Sound Lotto Test⁵⁸ excluded auditory agnosia. The patient had an IQ of 117 on Raven's⁵⁹ standard progressive matrices.

Discussion

The patient's peri-acute neuropsychological symptomatology consisted of auditory hallucinatory perceptions in the absence of other major disorders of the higher mental functions. A comparison of her clinical picture, as well as personal and medical histories, with the different conditions that may cause AHs, allowed us to reject all but two.

Elementary and complex AHs have been reported in patients with a long history of progressive, sensorineuronal deafness in one or both ears, together with tinnitus. They are often of acute onset at the time of a further loss of hearing, and are experienced predominantly in the deaf or deafer ear.^{51 54 60 61} They are not associated with a psychosis.^{13 57} AHs in deafness seem to be permanent.^{52 53 62 63} It appears that musical hallucinations may disappear after some time, and be replaced by short but repetitive musical phrases, like the noise of a scratched record.⁵⁷ Transitory, unilateral AHs have been mentioned in cases of acute ear disease. However, they parallel the evolution of the lesion, and gradually disappear on recovery.⁵⁴

Our patient had suffered progressive bilateral hearing loss for many years, greater on the right, not associated with tinnitus. ENT investigation during admission failed to disclose an acute lesion, and neither otoscopic changes nor further hearing loss were found when compared with the previous examination. In other words, our patient's perceptions did not occur at the time of a further decrement in hearing. The auditory phenomena progressively disappeared over a period of two weeks, which is not compatible with the persistent nature of otogenic AHs. As our patient did not present with an acute ear disease, the gradual disappearance of her musical perceptions does not fit transitory AHs of otopathic origin. Moreover, she experienced the musical perceptions in her better ear, which is quite unusual in AHs of otopathic origin.^{54 55 64} Finally, otogenic AHs seem to respond favourably to treatment with a hearing aid,^{60 61} whereas our patient's auditory perceptions were not altered or abolished by her prosthesis. Consequently, these findings indicate that the origin of our patient's auditory perceptions cannot be reduced to a mere otopathy.

Transient AHs may also be encountered in several cerebral conditions.^{7 11 15 42 45 49 56 65-72}

Figure Sagittal T1-weighted spin echo (short TR/short TE) MRI through the right sylvian fissure reveals a mild increase in signal intensity, presumably representing subacute haemorrhage (arrows). The high signal intensity represents methemoglobin formation.

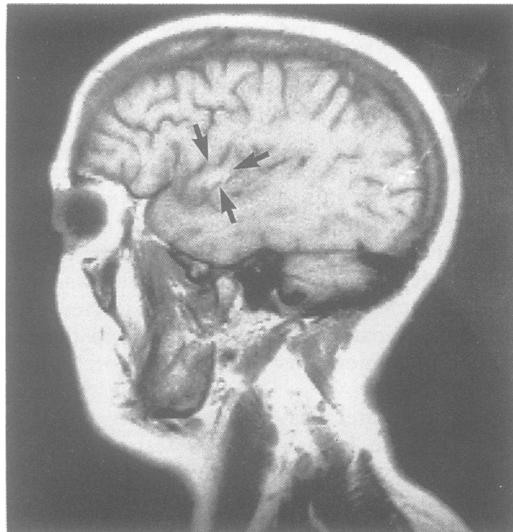


Table 1 Reviewed cases of spontaneous musical or verbal AHs, or both, of central origin, whether or not associated with peripheral pathology, with exclusion of psychiatric and alcoholic aetiologies*

Author(s)	Number of relevant cases (n = 118)	Recorded cases
Aizenberg <i>et al</i> ¹⁶	1	case 1
Bergman ⁵⁶	12	
Berrios ⁵⁷	1	case 1
Berthier and Starkstein ⁶⁷	1	
Boza and Liggett ⁷⁴	1	
Cambier <i>et al</i> ³	4	
Courville ⁷	11	cases 1, 2, 3, 4 cases 4663, 15863, 19244, 20125, 22359, 43, 582, 22383, 18273, 15440, 22539
Cummings <i>et al</i> ⁶⁸	1	
David <i>et al</i> ⁷⁵	2	
De Ajuriaguerra <i>et al</i> ⁸	2	cases 11, 12
De Morsier ⁶⁵	7	cases 25, 26, 28, 29, 30, 31, 32
De Reuck <i>et al</i> ⁴⁵	1	case 1
Dyck ⁷²	1	
Fenton and McRae ⁶⁰	1	
Hameke <i>et al</i> ⁵³	2	
Hécaen and Ropert ⁴²	19	cases 1, 2, 3, 4, 7, 8, 9, 10, 14, 15, 16, 20, 21, 23, 24, 27, 28, 30, 32
Keschner <i>et al</i> ⁹	4	
Keshavan <i>et al</i> ⁷⁶	1	
Lennox ⁵²	1	
Mikati <i>et al</i> ⁴⁷	1	
Miller and Crosby ⁵¹	1	
Moniz and Loff ⁶⁶	1	
Mulder and Daly ⁷⁷	1	
Neppe ⁴⁸	1	
Neumann <i>et al</i> ⁶⁹	1	
Paillas and Subirana ⁷⁸	5	
Patterson ⁴¹	1	
Penfield and Perot ⁴⁹	21	cases 6, 7, 10, 12, 16, 24, 29, 41, 42, 43, 44, 45, 46, 47, 58, 59, 60, 62, 63, 64, 65 case 9
Robertson ⁷⁹	1	
Riese ¹⁰	1	
Saijo <i>et al</i> ⁷⁰	1	
Scott ¹¹	1	
Shapiro <i>et al</i> ⁶³	1	
Tanabe <i>et al</i> ¹⁵	1	
Tarachow ¹²	6	

* In addition, Penfield and Perot⁴⁹ reported 26 cases in which they observed complex auditory phenomena following cortical stimulation.

Elementary and complex AHs may also occur with lesions of the tegmentum of the pons and lower midbrain.^{5, 6} MRI, CT and BAEP, however, failed to disclose a brainstem lesion in our patient, indicating that it was not brainstem auditory hallucinosis. Tanabe *et al*¹⁵ described a patient who developed a transient verbal hallucinosis in the right ear, which lasted for 17 days, as well as fluent aphasia following a left temporal haemorrhagic infarction. CT scans obtained during admission disclosed a low density area with small, high density spots in the left superior temporal gyrus, which was partially enhanced by contrast medium. However, when the hallucinosis had disappeared, the area with abnormal attenuation values was

no longer found. A linear, low density area remained at the lateral edge of the left superior temporal gyrus, and enlargement of the left sylvian fissure was noticed.

The concomitant disappearance of both the hyperdensities on the CT scans and the auditory phenomena might suggest that the second of these was caused by the abnormal excitatory effect of the haemorrhagic component of the lesion. According to Berrios,⁵⁷ musical perceptions tend to disappear together with the underlying pathology when associated with strokes. Such an abnormal brain excitability may well have brought forth the unilateral musical perceptions in our patient as well, as there seems to be a parallelism between the course of the phenomena experienced in the left ear and the progressive disappearance on CT of the hyperdense aspect of the right sylvian fissure. Moreover, the lesion location in our patient, as shown by CT and MRI, is consistent with the one described in those patients with unilateral complex AHs that Tanabe *et al*¹⁵ recorded in the literature on unilateral AHs of central origin: the lesions were located around the sylvian fissure and centred on the first temporal convolutions. In the cases reviewed by Tanabe *et al*¹⁵ as in our patient, the auditory phenomena were lateralised in the opposite ear to the involved hemisphere. Further evidence of this lateralisation phenomenon in AHs of central origin can be found in Penfield and Perot.⁴⁹ For instance, they induced a voice lateralised in the left ear by stimulating the right temporal cortex of their patient (case 29), and reported a patient (case 45) with left temporal lobe pathology who experienced verbal and musical phenomena in the right hemisphere.

The crucial question is whether verbal hallucinosis is associated with left hemisphere, and musical hallucinosis with right hemisphere lesions in right handed individuals. Berrios⁵⁷ expressed the view that the non-dominant hemisphere plays an important role in the generation of musical phenomena in patients with brain lesions, and that this finding fits in well with what is known about music processing in the brain. He also claims that there might be a dissociation between musical perceptions and the putative mechanisms related to hallucinatory voices in schizophrenic subjects, which seem to be associated with the dominant hemisphere.⁷³ To evaluate this view, we reviewed 118 patients with spontaneous musical or verbal AHs or both (table 1).

Only those cases with sufficient data on the contents of the hallucinations and the site of lesion were chosen as subjects of investigation. We excluded cases with concomitant convulsive speech acts, concomitant hallucinations in modalities other than the auditory one, concomitant mental disorders, past history of alcohol overconsumption and of mental illness, deafness and ear disease, evidence of bilateral cerebral involvement, insufficient information on handedness, cerebral dominance, or clinical picture. Table 2 gives an overview of the remaining 28 selected cases, according to the type of auditory perceptions, lateralisation

Table 2 Selected cases of complex AHs of central origin (Letters refer to authors' initials, followed by their patient's case number)

Type of perceptions	Spontaneous lesion		Experimental stimulus	
	Right	Left	Right	Left
Musical, songs		PP46 HR15 D1	PP5, PP9 PP17 PP30	PP11 PP31
Verbal, voices	PP29	PP43 PP44 DM25 HR10 HR16 ML	PP3, PP7 PP8, PP15 PP16, PP28	PP12
Verbal and musical	PP7	PP45 T		PP13 PP14

D = David *et al*⁷⁵; DM = De Morsier⁶⁵; HR = Hécaen and Ropert⁴²; ML = Moniz and Loff⁶⁶; PP = Penfield and Perot⁴⁹; T = Tanabe *et al*¹⁵.

(right versus left hemisphere) and nature (spontaneous versus experimental condition) of the lesion. No significant differences were found either between left versus right lateralisation of lesion/stimulus and type of auditory phenomena ($G = 1.37 < \chi^2 = 5.99$, $p = 0.05$) or between the nature of lesion/stimulus and type of auditory phenomena ($G = 1.81 < \chi^2 = 5.99$, $p = 0.05$). Our analysis indicates that there is no close association between the kind of phenomena experienced (verbal versus musical) and laterality of the lesion, and consequently does not enable us to confirm Berrios' ⁵⁷ conclusions. It supports Hécaen and Albert's ⁸⁰ findings that both complex musical and verbal hallucinosis with a clear, external sensory quality can be produced by lesions confined to the same hemisphere.

In conclusion, we reviewed the different clinical conditions that may cause AHs, and compared them with the clinical picture of the patient we described. We retained two major hypotheses to explain the occurrence of her auditory phenomena. Although the clinical evidence favours a neurological aetiology, the occurrence of a concomitant ear disease does not enable us to ignore the possible contributory and precipitating role of the otological factor. Whether the combination of the central and peripheral dysfunction was the necessary condition to cause the patient's musical perceptions remains uncertain. Nevertheless, both the description of our patient and the review of reported cases support the view that, in patients with organic cerebral disease, unilateral AHs can be considered a significant clinical sign indicating a lesion in the contralateral hemisphere. But we were not able to confirm the hypothesis that, in cases of hemispheric lesion, musical perceptions are related to pathology of the right hemisphere only.

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